Internalising symptoms and executive function difficulties in adolescents with and without Developmental Coordination Disorder

Serif Omer

Submitted for the Degree of

Doctor of Psychology
(Clinical Psychology)

School of Psychology
Faculty of Health and Medical Sciences
University of Surrey
Guildford, Surrey
United Kingdom

September 2018
Abstract

Background: There is growing evidence that individuals with Developmental Coordination Disorder (DCD) experience elevated internalising symptoms and executive function (EF) difficulties compared to their typically developing (TD) peers. Research also suggests that EFs are important for psychological wellbeing.

Aims: This study aimed to explore whether adolescents with DCD experience greater levels of internalising symptoms and everyday EF difficulties than their TD peers. It also explored whether EF difficulties mediate the relationship between DCD status and internalising symptoms. Methods and procedures: Fourteen adolescents with a diagnosis of DCD and 29 TD adolescents (ages 12-15) participated. A cross-sectional survey was conducted to collect parent-reported EF difficulties and self-reported internalising symptoms. Outcomes and results: Self-reported internalising symptoms and parent-reported EF difficulties were significantly higher in the DCD group compared to the TD group. A bias-corrected, bootstrapped mediation analysis identified that the effect of DCD on internalising symptoms was mediated by parent-reported EF difficulties. Exploratory analyses identified that this indirect effect was greatest for symptoms of depression through behavioural regulation difficulties.

Conclusions and implications: These findings support previous research indicating that adolescents with DCD experience greater levels of internalising symptoms and EF difficulties than their TD peers. This highlights the need for increased awareness, routine screening, and intervention for mental health and EF difficulties in people with DCD. The findings also highlight the potential benefits of targeting EF deficits in people with DCD to improve emotional wellbeing. However, larger scale, longitudinal research is needed.
Keywords: Developmental Coordination Disorder, DCD, internalising symptoms, depression, anxiety, executive functions
Acknowledgements

I would like to express thanks to my research supervisor, Dr Hayley Leonard, for her support and expertise throughout all aspects of this research project. I would also like to acknowledge Ana M. Jijon for her assistance with the data collection/extraction for the literature review and Dr Harriet Tenenbaum who provided critical feedback on the meta-analysis.

I am very grateful to the Dyspraxia Foundation for their help distributing recruitment advertisements for the empirical research study. I would also like to thank the schools and youth clubs that supported with data collection. I would like to thank all the young people and their parents who kindly volunteered their time to participate in my research.

Finally, I would like to thank my university tutors and placement supervisors who have supported me throughout the clinical training programme.
Publications and presentations

Parts of this thesis have been published in:


# Table of Contents

Abstract .......................... 2  
Acknowledgements .................. 4  
Publications and presentations ..... 5  
Part 1: Major Research Project Empirical Paper - *Internalising symptoms and executive function difficulties in adolescents with and without Developmental Coordination Disorder*  
  Abstract .................................. 8  
  Introduction ................................ 10  
  Method ..................................... 19  
  Results .................................... 27  
  Discussion .................................. 37  
  References .................................. 50  
  List of appendices .......................... 70  
  Appendices ................................ 71  
Part 2: Major Research Project Literature review - *Internalising symptoms in Developmental Coordination Disorder: A systematic review and meta analysis*  
  Abstract .................................. 123  
  Introduction ................................ 125  
  Method ..................................... 129  
  Results .................................... 135  
  Discussion .................................. 154  
  References .................................. 160  
  Appendix .................................. 173  
Part 3: Summary of Clinical Experience .......................... 174  
Part 4: Table of Assessments Completed During Training .......................... 177
Part 1: Major Research Project Empirical Paper

Internalising symptoms and executive function difficulties in adolescents with and without Developmental Coordination Disorder

Word count: 9420

Statement of Journal Choice
Research in Developmental Disabilities
“Research in Developmental Disabilities” primarily publishes empirical studies of an interdisciplinary nature that contribute to the understanding of problems associated with developmental disabilities. It has published numerous cross-sectional studies investigating the impact of Developmental Coordination Disorder. See Appendix A for more details.
Abstract

**Background:** There is growing evidence that individuals with Developmental Coordination Disorder (DCD) experience elevated internalising symptoms and executive function (EF) difficulties compared to their typically developing (TD) peers. Research also suggests that EFs are important for psychological wellbeing. **Aims:** This study aimed to explore whether adolescents with DCD experience greater levels of internalising symptoms and everyday EF difficulties than their TD peers. It also explored whether EF difficulties mediate the relationship between DCD status and internalising symptoms. **Methods and procedures:** Fourteen adolescents with a diagnosis of DCD and 29 TD adolescents (ages 12-15) participated. A cross-sectional survey was conducted to collect parent-reported EF difficulties and self-reported internalising symptoms. **Outcomes and results:** Self-reported internalising symptoms and parent-reported EF difficulties were significantly higher in the DCD group compared to the TD group. A bias-corrected, bootstrapped mediation analysis identified that the effect of DCD on internalising symptoms was mediated by parent-reported EF difficulties. Exploratory analyses identified that this indirect effect was greatest for symptoms of depression through behavioural regulation difficulties. **Conclusions and implications:** These findings support previous research indicating that adolescents with DCD experience greater levels of internalising symptoms and EF difficulties than their TD peers. This highlights the need for increased awareness, routine screening, and intervention for mental health and EF difficulties in people with DCD. The findings also highlight the potential benefits of targeting EF deficits in people with DCD to improve emotional wellbeing. However, larger scale, longitudinal research is needed.
**Keywords:** Developmental Coordination Disorder, DCD, internalising symptoms, depression, anxiety, executive functions
**Introduction**

Developmental Coordination Disorder (DCD) is a neurodevelopmental disorder affecting between 5-6% of children (American Psychiatric Association, 2013). It is characterised by significant impairment in motor coordination which interferes with everyday life, is evident in the early developmental period, and cannot be explained by another medical condition or intellectual disability. Individuals with DCD might have difficulties with self-care (e.g. tying shoe laces, brushing teeth), leisure activities (e.g. catching a ball, balancing) and academic tasks (e.g. handwriting). However, the impact of DCD also extends beyond these difficulties with motor tasks. Research has identified that DCD can impact on a wide range of physical, social, and psychological domains (Zwicker, Harris, & Klassen, 2013) and that difficulties can continue through childhood into adulthood (Cousins & Smyth, 2003; Hill, Brown, & Sorgardt, 2011). Given the relatively high prevalence of DCD, and evidence that it is often poorly understood by healthcare and education professionals (Gaines, Missiuna, Egan, & McLean, 2008; B. N. Wilson, Neil, Kamps, & Babcock, 2013), research exploring the wider impact of the condition is important to develop more effective, holistic interventions for this population.

**DCD and internalising symptoms**

One area that has recently received increased attention is the impact of DCD on an individual’s mental health, specifically internalising symptoms (Mancini, Rigoli, Cairney, Roberts, & Piek, 2016). ‘Internalising’ is a broader construct referring to symptoms of both depression and anxiety, which have been found to commonly overlap
in childhood and adolescence (Eaton et al., 2013; Kovacs & Devlin, 1998). A systematic review and meta-analysis of the literature identified that children and adolescents with DCD experience greater levels of internalising symptoms than their TD peers (see Part 2). Although the reviewed studies tended to adopt a cross-sectional design, two longitudinal studies were identified that found that a diagnosis of DCD in childhood predicted internalising symptoms later on in adolescence (Harrowell, Hollén, Lingam, & Emond, 2017; Wagner, Jekauc, Worth, & Woll, 2016). Research investigating this relationship in community samples of children (i.e. not specifically focusing on individuals diagnosed with DCD) has also identified that poor motor skills in childhood, measured as a continuous construct, predict internalising symptoms in adolescence (Piek, Barrett, Smith, Rigoli, & Gasson, 2010; Sigurdsson et al., 2002) and adulthood (Poole et al., 2015). Additionally, there is evidence that interventions focused on improving motor skills can have a positive effect on mental health (Piek et al., 2015). Together, these findings would suggest a causal relationship between DCD and mental health difficulties.

The Environmental-Stress Hypothesis was introduced as a framework to understand this relationship (Cairney, Rigoli, & Piek, 2013). It proposes that the motor impairments in DCD can expose an individual to a range of secondary psychosocial stressors which, over time, can result in greater levels of internalising symptoms. Recent research has explored a number of these secondary stressors, providing support for the Environmental-Stress Hypothesis. For example, there is evidence that increased peer victimisation (Campbell, Missiuna, & Vaillancourt, 2012), reduced leisure activities (Raz-Silbiger et al., 2015), poorer self-esteem (Rigoli, Piek, & Kane, 2012), physical
inactivity (Li et al., 2018), reduced social support (Rigoli et al., 2017), and lower perceived academic performance (Lingam et al., 2012) may all mediate the relationship between motor difficulties and internalising symptoms. The findings would suggest that targeting these psychosocial factors, in addition to the motor difficulties, could have beneficial outcomes for the emotional wellbeing of people with DCD. As such, further research exploring the mediating factors through which DCD might impact on internalising symptoms will have important implications for treatment in this population.

**DCD and executive function**

Recent research has also investigated the role of executive functions (EF) in DCD. EFs refer to a set of higher-order cognitive processes that regulate, monitor and control cognition, emotions and behaviour in order to achieve a particular goal (Diamond, 2013). Three core EFs have been identified consisting of inhibition (i.e. the ability to control natural responses or ignore irrelevant stimuli), working memory (i.e. the ability to temporarily hold information in mind whilst simultaneously manipulating information) and cognitive flexibility (i.e. the ability to switch between thinking about different concepts or tasks), which together underlie more complex EFs including planning, problem solving and reasoning (Miyake et al., 2000).

It has been highlighted that there is an important overlap in the development of motor skills and EFs, with both sharing underlying neural pathways involving the prefrontal cortex and cerebellum (Diamond, 2000; Koziol, Budding, & Chidekel, 2012). Compromise to these neural pathways has been implicated in the motor difficulties experienced by people with DCD (Biotteau et al., 2016). As such, research has also
found that individuals with DCD perform worse on a wide range of EF tasks compared to their TD peers (Leonard & Hill, 2015; P. H. Wilson, Ruddock, Smits-Engelsman, Polatajko, & Blank, 2013). This includes performance-based tasks of inhibition (Mandich, Buckolz, & Polatajko, 2002), working memory (Alloway, 2007), cognitive flexibility (Wuang, Su, & Su, 2011) and planning (Piek et al., 2004). Individuals with DCD also self-report greater difficulties with EF in their everyday life compared to TD controls (Tal Saban, Ornoy, & Parush, 2014). Additionally, findings from longitudinal studies have found motor difficulties in childhood predict EF difficulties at a later age (Bernardi, Leonard, Hill, Botting, & Henry, 2018; Michel, Roethlisberger, Neuenschwander, & Roebers, 2011). This would suggest that EF impairments are a significant feature of DCD and may be inextricably linked to the motor difficulties that characterise the disorder.

Executive function and internalising symptoms

Despite growing evidence that individuals with DCD experience greater levels of both internalising symptoms and EF difficulties compared to their TD peers, there has been no attempt to explore the relationship between the two within this population. The Environmental-Stress Hypothesis has been an important contribution to the field in understanding how motor difficulties can contribute to elevated internalising symptoms, however it only considers the role of physical and psychosocial factors. Given the impact of DCD on EFs, it is important to consider how these higher-order cognitive processes might also impact on the mental health of individuals with DCD.
There is a growing body of literature highlighting the importance of EFs for mental health. Meta-analytic reviews have identified EF deficits in both children (Wagner, Müller, Helmreich, Huss, & Tadić, 2015) and adults (Snyder, 2013; Snyder, Kaiser, Warren, & Heller, 2014) with depression and anxiety disorders. Neuroimaging studies have also found the brain networks involved in EF to be atypical in individuals with mood disorders (Koenigs & Grafman, 2009; Price & Drevets, 2010). Additionally, there is evidence to suggest that EF difficulties precede internalising symptoms in children and adolescents. For example, working memory (Evans, Kouros, Samanez-Larkin, & Garber, 2016; Letkiewicz et al., 2014), inhibitory control (Lengua, 2006; Martel et al., 2007; Nigg, Quamma, Greenberg, & Kusche, 1999; Riggs, Blair, & Greenberg, 2003), cognitive flexibility (Evans et al., 2016) and global EF difficulties (Han et al., 2016) measured during childhood have been found to prospectively predict internalising symptoms. In support of this relationship, improvements in EF have been found to mediate the positive effects of neurocognitive intervention programmes on reducing internalising symptoms in children (Riggs, Greenberg, Kusché, & Pentz, 2006).

Together, these findings highlight the role of EF in mental health and the potential importance it has for people with DCD.

The relationship between EFs and internalising symptoms can be understood through a variety of possible pathways. One explanation is the role of EFs in emotion regulation. Emotion regulation refers to the process of actively changing the intensity or duration of an emotional response (Koole, 2009). High levels of maladaptive emotion regulation strategies (e.g. avoidance, rumination, worry) and low levels of adaptive emotion regulation strategies (e.g. problem solving, cognitive reappraisal) have been
associated with internalising symptoms (Garber, Braafladt, & Weiss, 1995; Mezulis, Priess, & Hyde, 2011; Silk, Steinberg, & Morris, 2003). It has been suggested that EFs play a key role in the effective use of these strategies (Joormann & D’Avanzato, 2010; Schmeichel & Tang, 2015; Wante, Mezulis, Beveren, & Braet, 2017). For example, cognitive flexibility impairments have been linked to increased internalising symptoms through perseveration of negative thinking, such as worry and rumination (Brosschot, Gerin, & Thayer, 2006; Demeyer, De Lissnyder, Koster, & De Raedt, 2012; Johnson, 2009). Difficulties with inhibition have been associated with depression through increased rumination and reduced reappraisal strategies (Joormann & Gotlib, 2010). Difficulties in updating working memory have also been linked with depression and anxiety through increased rumination and worry (Meiran, Diamond, Toder, & Nemets, 2011; Owens, Stevenson, Hadwin, & Norgate, 2012). Generally, this research indicates that individuals with EF deficits find it harder to shift their attention away from negative emotional information and use fewer adaptive strategies, which contributes to higher levels of depression and anxiety. In addition to emotion regulation, EFs are also important for academic functioning (Best, Miller, & Naglieri, 2011) and social skills (Dawson, Shear, & Strakowski, 2012). Difficulties with each of these areas can also have implications for emotional wellbeing (Ende, Verhulst, & Tiemeier, 2016; Nilsen, Karevold, Røysamb, Gustavson, & Mathiesen, 2013; Zhang, Zhang, Chen, Ji, & Deater-Deckard, 2018).
The present study

Given the emerging evidence that individuals with DCD experience deficits in EF abilities and the role that these higher-order cognitive processes have in psychological wellbeing, it is likely that these deficits have important implications for the mental health of people with DCD. To date, no research has investigated this potential relationship between EF difficulties and internalising symptoms in DCD. However, parallels can be drawn from research with other neurodevelopmental disorders that impact on EF. For example, Gardiner and Iarocci (2018) found an association between parent-rated EF difficulties and depression in children with an autism spectrum disorder (ASD), suggesting that improving EFs could be an important target for reducing internalising symptoms. More specifically, Lawson et al. (2015) found that a diagnosis of ASD, compared to a TD group, predicted difficulties with cognitive flexibility which in turn predicted higher levels of anxiety and depression. This suggests that individuals with ASD experience greater levels of internalising symptoms than their TD peers and that this relationship may be mediated through EF difficulties. EFs have been found to have a similar explanatory role for the high levels of internalising symptoms experienced in other childhood disorders, including spina bifida (Kelly et al., 2012).

The purpose of the present study, therefore, was to investigate EF difficulties and internalising symptoms in adolescents with and without DCD to better understand these difficulties in the disorder. First, the study set out to replicate previous research findings that adolescents with DCD have higher levels of both EF difficulties and internalising symptoms compared to TD adolescents. Second, it explored whether the impact of DCD on internalising symptoms is mediated through the difficulties in EF that are often
reported in this population (see Figure 1). Adolescents were chosen as the focus for this study given that much of the research into internalising symptoms in DCD has focused on pre-adolescent children (see Part 2). Furthermore, there is some suggestion that the impact of DCD on mental health may be greater for adolescents compared to younger children (Missiuna, Moll, King, King, & Law, 2007; Skinner & Piek, 2001). Given that adolescence is also generally considered a time of high risk for mental health difficulties and an important period for the ongoing development of EFs (Blakemore & Choudhury, 2006; Ernst, Pine, & Hardin, 2006; Twenge & Nolen-Hoeksema, 2002), focusing the present study on this age group was deemed highly relevant.

Figure 1. Mediation model depicting the indirect effect of DCD status on internalising symptoms through EF difficulties
Previous research into EFs in DCD has focused predominantly on performance-based measures of EF (Leonard & Hill, 2015; P. H. Wilson et al., 2013). Although these tasks are useful for assessing the specific impact of DCD on individual components of EF in controlled conditions, there is an argument that they do not always represent functioning in the ‘real-world’ environment (Burgess et al., 2006; Toplak, West, & Stanovich, 2013). Therefore, studies adopting more ecological measures of EF difficulties, such as behaviour rating scales, are also necessary to complement the findings from standardised, laboratory-based tasks. Indeed, Tal Saban et al. (2014) found greater EF deficits in adults with DCD compared to TD adults based on self-report of everyday EF difficulties. However, no prior research has explored everyday EF difficulties in adolescents with DCD using such measures. Therefore, the present study adopted a behaviour rating scale as its measure of EF (Gioia, Isquith, Guy, & Kenworthy, 2000) to better understand how EF difficulties in day-to-day life are impacted by DCD in adolescents.

A self-report measure of internalising symptoms (Ebesutani et al., 2012) was adopted for the present study, given that parents may under-report the emotional difficulties of adolescents (Cantwell, Lewinsohn, Rohde, & Seeley, 1997; Hope et al., 1999; Smith, 2007; Sourander, Helstelä, & Helenius, 1999). A parent-reported measure of everyday EF difficulties was used to minimise the impact of common method bias that could otherwise be problematic for mediation analyses if self-report measures are used for both the mediator and dependent variable (Podsakoff, MacKenzie, Lee, & Podsakoff, 2003; Podsakoff, MacKenzie, & Podsakoff, 2012).
The present study, therefore, had three main hypotheses: (1) adolescents with DCD would have greater self-reported internalising symptoms than TD adolescents; (2) adolescents with DCD would have greater parent-reported EF difficulties than TD adolescents; and (3) parent-reported EF difficulties would mediate the relationship between DCD status (i.e. DCD vs. TD) and self-reported internalising symptoms (see Figure 1).

Method

Design

The study adopted a quantitative cross-sectional design to measure EF difficulties and internalising symptoms in a group of adolescents with DCD and a group of TD adolescents at one point in time.

Participants

DCD group. Participants were eligible for the DCD group if they met the following criteria: (i) a diagnosis of DCD which was confirmed by the researchers through inspection of clinical reports, (ii) aged 12-years-0-months to 15-years-11-months, (iii) scored ≤57 on the Developmental Coordination Disorder Questionnaire – 07 (DCDQ-07) to confirm the current presence of motor difficulties (see measures), and (iv) had a parent/guardian willing to complete questionnaire measures. Adolescents were excluded if they had a comorbid diagnosis of Attention Deficit Hyperactivity Disorder (ADHD) or ASD, as reported by their parents, so that findings could be more easily attributed to DCD. They were also excluded if they lived outside the United Kingdom.
(UK), due to international variations in diagnosis and intervention, or if any of the questionnaire measures were missing. Participants with DCD were recruited through advertisements distributed via a national charity, registers of participants from previous studies, a secondary school in England specialising in specific learning difficulties, and word of mouth. Thirty-nine adolescents and their parents responded to the advertisements, of which 30 went on to complete the initial screening questionnaire. Fifteen were subsequently excluded due to the adolescent being older than 16 (n = 4), reporting a diagnosis of ADHD (n = 2), reporting a diagnosis of ASD (n = 7), living outside the UK (n = 1), or not providing clinical reports to confirm their diagnosis (n = 1). Of the remaining 15 participants that went on to complete the questionnaire measures, the Revised Child Anxiety and Depression Scale – Short Version (RCADS-25) was missing for one participant. As such, the DCD sample consisted of 14 adolescents and their parents.

**TD group.** Participants were eligible for the TD group if they met the following criteria: (i) aged 12-years-0-months to 15-years-11-months, (ii) scored ≥58 on the DCDQ-07, and (iii) had a parent/guardian willing to complete questionnaire measures. Adolescents were excluded if they had a diagnosis of DCD, ADHD or ASD, as reported by their parents. They were also excluded if they lived outside the UK or if any of the questionnaire measures were missing. TD participants were recruited through advertisements distributed via secondary schools and youth clubs in south-east England, registers of participants from previous studies, and word of mouth. Forty adolescents and their parents responded to the advertisements, of which 37 went on to complete the initial screening questionnaire. Four were subsequently excluded due to the adolescent
being older than 16 (n = 2), reporting a diagnosis of ADHD (n = 1), and reporting a
diagnosis of ASD (n = 1). Of the remaining 33 participants that went on to complete the
questionnaire measures, three had scores below the cut-off on the DCDQ-07 and one did
not complete the RCADS-25. As such, the TD sample consisted of 29 adolescents and
their parents.

Measures

Motor coordination difficulties. The Developmental Coordination Disorder
Questionnaire (DCDQ-07; Wilson et al., 2009) is a 15-item parent-report questionnaire
that measures motor coordination in children and adolescents (see Appendix B). It
consists of three subscales that measure control during movement, fine motor skills, and
general coordination. It provides a total score ranging from 15 to 75 with higher scores
indicating better motor coordination. The DCDQ-07 is commonly used as a screening
measure for DCD. In adolescents, a cut-off of 57 or below indicates the possible
presence of DCD. The DCDQ-07 has been found to have high internal consistency
(Cronbach’s alpha, \( \alpha = .94 \)), concurrent validity with performance-based measures of
motor ability (\( r = .55 \)), and a good ability to detect DCD (sensitivity: 88.5%; specificity:
75.6%) (Wilson et al., 2009). In the current study, all parents were asked to complete the
DCDQ-07 to confirm group assignment and to obtain a measure of the severity of motor
difficulties in both study groups. Cronbach’s alpha for the DCDQ-07 Total score in the
current study was high (\( \alpha = .98 \)).

Internalising symptoms. The Revised Child Anxiety and Depression Scale –
Short Version (RCADS-25; Ebesutani et al., 2012) is a 25-item self-report questionnaire
that assesses depression (10 items) and anxiety (15 items) in children and adolescents (see Appendix C). Adolescents are asked to rate how often they have been experiencing difficulties on a four-point Likert-type scale from 0 (Never) to 3 (Always). The RCADS-25 provides a total internalising symptoms score, in addition to separate subscale scores for depression and anxiety. Raw scores are converted to age- and gender-standardised T-scores based on normative reference groups ($M = 50$, $SD = 10$). T-scores of 65 or more can be considered borderline clinically significant. The RCADS-25 has been found to have acceptable internal reliability in both community (Anxiety, $\alpha = .94$; Depression, $\alpha = .79$) and clinic-referred samples (Anxiety, $\alpha = .96$; Depression, $\alpha = .80$), and can discriminate children with anxiety disorders and depression (Ebesutani et al., 2012). All adolescents were asked to complete the RCADS-25 as a measure of internalising symptoms. Cronbach’s alpha for the total score and the subscale scores in the current study were high (Total, $\alpha = .92$; Depression, $\alpha = .86$; Anxiety, $\alpha = .90$).

**EF difficulties.** The Behaviour Rating Inventory of Executive Function (BRIEF; Gioia, Isquith, Guy, & Kenworthy, 2000) is an 86–item questionnaire that assesses EF deficits in everyday life (see Appendix D). Parents are asked to rate their child’s behaviour on a three-point Likert-type scale (‘Never a problem’, ‘Sometimes a problem’, ‘Often a problem’). The BRIEF consists of eight clinical subscales that form two composite indexes, the Behavioural Regulation Index (BRIEF BRI; i.e. inhibition, shifting, emotional control) and the Metacognitive Index (BRIEF MCI; i.e. initiation, working memory, planning, organisation of materials, monitoring). It also provides an overall General Executive Composite (BRIEF GEC). Raw scores are converted to age- and gender-standardised T-scores based on normative reference groups ($M = 50$, $SD = $
10). T-scores of 65 or more are suggested as the threshold for clinical significance (Gioia et al., 2000). The BRIEF has been found to have high internal consistency ($\alpha = .82-.98$), test-retest reliability ($r = .72-.84$), and demonstrates predictive, convergent, and divergent validity (Gioia et al., 2000). The Cronbach’s alpha for the BRIEF GEC and the two subscale indexes in the present study were high (GEC, $\alpha = .98$; BRI, $\alpha = .96$; MCI, $\alpha = .98$).

**Procedure**

Advertisements for the study were distributed widely, as described in the participants section (see Appendix E). This included via email, newsletters, social media, and word of mouth. Interested participants were asked to read an information sheet containing the study details (see Appendix F). Parents were asked to sign informed consent whilst adolescents were asked to sign informed assent (see Appendices G and H). Parents then completed an initial eligibility questionnaire (see Appendix I). Those meeting criteria for the study were asked to complete the main questionnaires as part of a survey. This included a section for the parent-report questionnaires and a section for the adolescent self-report questionnaire. A survey containing the questionnaires was available in paper form and online using Qualtrics survey software, depending on participant preferences. At the end of the survey, participants were provided an opportunity to enter a prize draw for a £100 Amazon voucher. The study received a favourable ethical opinion from the University of Surrey Faculty of Health and Medical Sciences Ethics Committee (see Appendix J).
Data analysis

**Preliminary analyses.** Data were analysed using IBM SPSS Statistics version 24. Scale and subscale scores were calculated for each of the questionnaire measures. Missing data on the questionnaires were imputed using the mean of the available scores for that scale. The data were screened for univariate outliers and parametric assumptions within the DCD and TD groups separately. Homogeneity of variance was explored for independent samples *t*-tests. Additionally, multivariate outliers, independence of errors, linearity, homoscedasticity, normally distributed errors, homogeneity of regression, and multicollinearity were explored for regression analyses. Descriptive statistics were calculated to characterise the demographics for each group, with differences explored using independent samples *t*-tests and chi-square tests. For each group, one-sample *t*-tests were conducted to compare the RCADS-25 and BRIEF scores to the normative samples for each measure.

**Group comparisons.** To test Hypotheses 1 and 2, internalising symptoms (RCADS-25 Total) and EF difficulties (BRIEF GEC) were compared between the DCD and TD groups using separate independent samples *t*-tests with alpha values set at .05. Where parametric assumptions were not met, 95% bias-corrected and accelerated (BCa) bootstrapped confidence intervals were calculated for the mean difference, based on 10,000 samples. If confidence intervals did not cross zero, then the difference can be interpreted as significant. Based on guidelines, it was not deemed appropriate to adjust for multiple comparisons since only a small number of pre-planned, specific hypotheses were explored (Streiner & Norman, 2011). Instead, the exact *p*-values and confidence intervals are reported to enable appropriate interpretation. Cohen’s *d* was also calculated.
for each comparison as a measure of the effect size, whereby 0.3, 0.5 and 0.8 equate to small, medium and large effect sizes respectively (Cohen, 1992).

Mediation analysis. A simple mediation analysis was conducted to test for an indirect effect of DCD status on internalising symptoms through EF difficulties (Hypothesis 3). DCD Status was dummy coded for these analyses (TD = 0, DCD = 1).

First, Pearson’s correlation coefficients were calculated to explore zero-order correlations between all study variables and to determine whether demographics should be included as covariates. 95% BCa bootstrap confidence intervals were calculated for the correlation coefficients due to deviations from normality in some study variables. The mediation analysis was then conducted using the PROCESS macro for SPSS (Hayes, 2013). A bootstrapping approach was used based on 10,000 samples with replacement, to calculate a 95% bias-corrected confidence interval for the indirect effect (path ab; see Figure 1). If the confidence interval for the indirect effect does not cross zero, then it can be considered significant. This approach makes no assumptions about the sampling distribution of the indirect effect, increases power, minimises the risk of Type II error, and is recommended for small samples (Fritz & MacKinnon, 2007; Preacher & Hayes, 2004, 2008). The partially standardised effect size was calculated which represents the number of standard deviations on the dependent variable (RCADS-25 Total) by which the DCD and TD groups differ because of the indirect path. Given its ease of interpretation, this measure is recommended for use in mediation analyses with a dichotomous independent variable (Hayes, 2013; Lachowicz, Preacher, & Kelley, 2018).

Post-hoc analyses. Additional post-hoc analyses were conducted to better understand the nature of any significant findings. This includes further independent
samples $t$-tests and mediation analyses using the subscales of the RCADS-25 and BRIEF. These further analyses were exploratory to develop additional hypotheses and therefore, based on guidelines, no adjustment for multiple testing was made (Streiner & Norman, 2011). Again, $p$-values and confidence intervals are reported to enable appropriate interpretation.

**Power calculation**

Based on previous research, a medium-large effect size ($d = 0.6$) was expected for the group differences in internalising symptoms (Missiuna et al., 2014; see also Part 2) and a large effect ($d = 0.8$) for EF difficulties (Tal-Saban, Ornoy, & Parush, 2014; P. H. Wilson et al., 2013). Power calculations, conducted using G Power v.3.1 (Faul, Erdfelder, Lang, & Buchner, 2007) identified that a sample size of 90 would be required to detect an effect size of 0.6 with 80% power and a 5% significance level. As such, the study sample size of 43 was underpowered to detect a medium-large effect for Hypotheses 1 and 2.

Power calculations for mediation analyses can be calculated based on the effect size of path $a$ and path $b$ (Fritz & MacKinnon, 2007). As described above, a large effect was expected for path $a$. A medium-large effect was also expected for path $b$, based on previous research into the relationship of the BRIEF with internalising symptoms (Ghassabian et al., 2014; White, Jarrett, & Ollendick, 2012). Fritz & MacKinnon (2007) estimated that, for bias-corrected bootstrapping analyses, a minimum sample of 54 is required to detect an indirect effect, with 80% power, when the effect of path $a$ is large
and path $b$ is medium (a conservative estimate). As such, the study sample size of 43 was slightly underpowered to detect the expected indirect effect.

**Results**

**Preliminary analysis and descriptive statistics**

The final sample consisted of 14 adolescents in the DCD group and 29 adolescents in the TD group. The BRIEF was missing a single item for three participants which were imputed using the mean values of the available scores. The data were screened for parametric assumptions (see Appendices K and L). No univariate or multivariate outliers were identified. Both the RCADS-25 and BRIEF scores were positively skewed in the TD group and negatively skewed in the DCD group.

Table 1. Summary of the demographics in the DCD and TD groups

<table>
<thead>
<tr>
<th>Variable</th>
<th>DCD ($n = 14$)</th>
<th>TD ($n = 29$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, $M$ (SD) years</td>
<td>13.84 (1.13)</td>
<td>13.72 (1.08)</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male, $n$ (%)</td>
<td>9 (64.3%)</td>
<td>16 (55.2%)</td>
</tr>
<tr>
<td>Female, $n$ (%)</td>
<td>5 (35.7%)</td>
<td>13 (44.8%)</td>
</tr>
<tr>
<td>DCDQ-07 Total, $M$ (SD)</td>
<td>28.64 (8.54)</td>
<td>69.90 (4.86)</td>
</tr>
<tr>
<td>DCDQ-07 Control, $M$ (SD)</td>
<td>12.50 (4.62)</td>
<td>28.10 (2.55)</td>
</tr>
<tr>
<td>DCDQ-07 Fine, $M$ (SD)</td>
<td>7.71 (2.46)</td>
<td>18.17 (1.83)</td>
</tr>
<tr>
<td>DCDQ-07 General, $M$ (SD)</td>
<td>8.43 (2.71)</td>
<td>22.93 (1.96)</td>
</tr>
</tbody>
</table>

DCD: Developmental Coordination Disorder; DCDQ-07: Developmental Coordination Disorder Questionnaire – 07; TD: Typically developing
Table 1 summarises the descriptive statistics of the sample. Overall, there were more males than females in the sample. There was a higher percentage of males in the DCD group, however this difference was not significant, $\chi^2 (1) = 3.22, p = 0.57$. There was also no significant difference in ages between the two groups, $t (41) = -3.50; p = 0.74$. Age and gender were, therefore, not included as covariates in group comparisons. As expected, the DCD group had significantly lower total scores on the DCDQ-07, $t (41) = -20.23, p < .001$, than the TD group.

The scores for the study measures are summarised in Table 2. Overall, the mean total score on the RCADS-25 was lower in the TD group compared to the normative sample and higher in the DCD group. One sample $t$-tests within each group found this difference to be significant in the TD group, $t (28) = -4.12, p < .001$, but not significantly different in the DCD group, $t (13) = 0.69, p = .50$. Within the TD group, one participant (3.4%) scored above the clinical cut-off ($T \geq 65$) for anxiety, but no participants scored above cut-off for depression or overall internalising symptoms. Within the DCD group, one participant (7.1%) scored above the clinical cut-off for depression, two for anxiety (14.3%), and four (28.6%) for overall internalising symptoms.

The mean scores on the BRIEF GEC in the TD group were broadly comparable with the normative samples, though the scores in the DCD group were higher. This difference was significant in the DCD group, $t (13) = 15.26, p = .001$, but not in the TD group, $t (27) = -2.01, p = .055$. Only one participant (3.4%) scored above the clinical cut-off ($T \geq 65$) on the BRIEF GEC in the TD group. However, thirteen (93%) of the DCD participants scored above the cut-off, indicating that the clear majority experienced significant EF difficulties according to parent report.
Group differences

Independent samples $t$-tests were conducted to compare the two groups on internalising symptoms (RCADS Total, Hypothesis 1) and EF difficulties (BRIEF GEC, Hypothesis 2). Given that the distribution of the variables deviated from normality (see Appendix K), bootstrapping with 10,000 samples was used to generate BCa 95% confidence intervals for the mean difference.

Table 2. Summary of the scores for study variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>DCD ($n = 14$)</th>
<th>TD ($n = 29$)</th>
<th>Mean difference</th>
<th>BCa 95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>RCADS-25 Total, $M$ (SD)</td>
<td>52.23 (12.09)</td>
<td>41.98 (10.29)</td>
<td>10.24</td>
<td>2.96, 17.24*</td>
</tr>
<tr>
<td>RCADS-25 Depression, $M$ (SD)</td>
<td>54.12 (9.64)</td>
<td>41.19 (9.33)</td>
<td>12.93</td>
<td>6.78, 18.78*</td>
</tr>
<tr>
<td>RCADS-25 Anxiety, $M$ (SD)</td>
<td>50.49 (12.83)</td>
<td>44.19 (10.83)</td>
<td>6.30</td>
<td>-0.91, 13.64</td>
</tr>
<tr>
<td>BRIEF GEC, $M$ (SD)</td>
<td>77.07 (6.64)</td>
<td>46.69 (8.80)</td>
<td>30.38</td>
<td>25.65, 34.77*</td>
</tr>
<tr>
<td>BRIEF BRI, $M$ (SD)</td>
<td>75.36 (8.88)</td>
<td>48.28 (9.37)</td>
<td>27.08</td>
<td>21.42, 32.75*</td>
</tr>
<tr>
<td>BRIEF MCI, $M$ (SD)</td>
<td>74.50 (7.21)</td>
<td>46.97 (9.13)</td>
<td>27.53</td>
<td>22.31, 32.44*</td>
</tr>
</tbody>
</table>

* 95% BCa bootstrapped confidence interval is significant

BCa: Bias-corrected and accelerated; BRI: Behavioural Regulation Index; BRIEF: Behaviour Rating Inventory of Executive Function; CI: Confidence interval; DCD: Developmental Coordination Disorder; GEC: Global Executive Composite; MCI: Metacognitive Index; RCADS-25: Revised Child Anxiety and Depression Scale – Short version; TD: Typically developing

Internalising symptoms. Adolescents in the DCD group reported greater levels of internalising symptoms ($M = 52.23; SE = 3.23$) compared to adolescents in the TD group ($M = 41.98; SE = 1.91$). This difference, 10.24, BCa 95% CI [2.96, 17.24], was significant, $t (41) = 2.89, p = 0.006$ and represents a large effect size, $d = 0.90$.

EF difficulties. Adolescents in the DCD group reported greater levels of EF difficulties ($M = 77.07; SE = 1.77$) compared to adolescents in the TD group ($M = 46.69; SE = 1.77$).
SE = 1.63). This difference, 30.38, BCa 95% CI [25.65, 34.77]; p < .001) was significant, t (41) = 11.42, p < .001 and represents a large effect size, d = 3.57.

**Intercorrelations:**

Intercorrelations between the study variables were computed using Pearson’s correlation coefficients. Bootstrapped BCa 95% confidence intervals were calculated, given the deviation from normality for some variables. There were significant correlations between the independent, mediator and dependent variables as expected (see Table 3). However, no significant correlation was identified between DCD status and the RCADS-25 Anxiety subscale. Neither age nor gender were significantly correlated with any of the study variables and were, therefore, not included as covariates in the mediation analyses.

**Mediation analysis**

A simple mediation analysis was conducted to test each of the paths in the mediation model. As shown in Figure 2 and Table 4, adolescents with a diagnosis of DCD had higher levels of self-reported internalising symptoms, \( c = 10.24, p = .006 \), and higher levels of parent-reported EF difficulties, \( a = 30.38, p < .001 \), compared to TD adolescents. Adolescents with higher levels of parent-reported EF difficulties also self-reported higher levels of internalising symptoms after controlling for DCD status, \( b = 0.55, p = .007 \). A bias-corrected (BC) bootstrap confidence interval for the indirect effect, based on 10,000 bootstrap samples, did not cross zero, \( ab = 16.62, BC 95\% CI \)
Table 3. Pearson correlation coefficients of variables with 95% bias-corrected and accelerated bootstrapped confidence intervals

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. DCD status</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. BRIEF GEC</td>
<td>.87*</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(.78, .94)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. BRIEF BRI</td>
<td>.82*</td>
<td>.93*</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(.70, .90)</td>
<td>(.88, .97)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. BRIEF MCI</td>
<td>.84*</td>
<td>.96*</td>
<td>.83*</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(.73, .92)</td>
<td>(.92, .99)</td>
<td>(.73, .90)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. RCADS-25 Total</td>
<td>.41*</td>
<td>.54*</td>
<td>.54*</td>
<td>.49*</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(.10, .66)</td>
<td>(.33, .73)</td>
<td>(.32, .73)</td>
<td>(.26, .69)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. RCADS-25 Depression</td>
<td>.55*</td>
<td>.62*</td>
<td>.63*</td>
<td>.57*</td>
<td>.86*</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(.28, .77)</td>
<td>(.40, .79)</td>
<td>(.42, .80)</td>
<td>(.34, .75)</td>
<td>(.74, .94)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. RCADS-25 Anxiety</td>
<td>.25</td>
<td>.40*</td>
<td>.36*</td>
<td>.94*</td>
<td>.64*</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(-.08, .55)</td>
<td>(.16, .63)</td>
<td>(.14, .64)</td>
<td>(.10, .60)</td>
<td>(.90, .97)</td>
<td>(.40, .82)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Age</td>
<td>.06</td>
<td>.15</td>
<td>.07</td>
<td>.16</td>
<td>.02</td>
<td>.07</td>
<td>-</td>
<td>.02</td>
</tr>
<tr>
<td></td>
<td>(-.26, .38)</td>
<td>(-.41, .17)</td>
<td>(-.23, .36)</td>
<td>(-.16, .47)</td>
<td>(-.28, .33)</td>
<td>(-.24, .39)</td>
<td>(-.29, .26)</td>
<td></td>
</tr>
<tr>
<td>9. Gender</td>
<td>-.09</td>
<td>.76</td>
<td>-.02</td>
<td>-.06</td>
<td>.13</td>
<td>.15</td>
<td>.10</td>
<td>-.11</td>
</tr>
<tr>
<td></td>
<td>(-.36, .20)</td>
<td>(-.33, .25)</td>
<td>(-.30, .28)</td>
<td>(-.35, .23)</td>
<td>(-.16, .41)</td>
<td>(-.15, .43)</td>
<td>(-.20, .39)</td>
<td>(-.41, .17)</td>
</tr>
</tbody>
</table>

* 95% BCa bootstrapped confidence interval is significant

BCa: Bias-corrected and accelerated; BRI: Behavioural Regulation Index; BRIEF: Behaviour Rating Inventory of Executive Function; CI: Confidence interval; DCD: Developmental Coordination Disorder; GEC: Global Executive Composite; MCI: Metacognitive Index; RCADS-25: Revised Child Anxiety and Depression Scale – Short version.
The direct effect of DCD status on internalising symptoms was also no longer significant after accounting for EF difficulties, \(c' = -6.38, p = .346\). The partially standardised indirect effect was 1.41, BC 95% CI [0.30, 2.37]. These results indicate that adolescents with DCD reported internalising symptoms that, on average, were 1.41 standard deviations (approximately 17 points on the RCADS-25) higher compared to TD adolescents because of the indirect effect through parent-reported EF difficulties.

Table 4. Results of the mediation analysis using the RCADS-25 Total scores and BRIEF GEC

<table>
<thead>
<tr>
<th>Effect of IV on M (Path a)</th>
<th>(B) coefficient</th>
<th>(SE)</th>
<th>(t)</th>
<th>(p)-value</th>
<th>Bias-corrected bootstrap 95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Effect of M on DV (Path b)</td>
<td>0.55</td>
<td>0.19</td>
<td>2.85</td>
<td>.007</td>
<td></td>
</tr>
<tr>
<td>Total Effect (Path c)</td>
<td>10.24</td>
<td>3.54</td>
<td>2.89</td>
<td>.006</td>
<td></td>
</tr>
<tr>
<td>Direct effect (Path c')</td>
<td>-6.38</td>
<td>6.69</td>
<td>-0.95</td>
<td>.346</td>
<td></td>
</tr>
<tr>
<td>Indirect Effect (a x b)</td>
<td>16.62</td>
<td></td>
<td></td>
<td></td>
<td>[4.16, 27.04]*</td>
</tr>
</tbody>
</table>

* 95% bias-corrected bootstrapped confidence interval significant

BRIEF GEC: Behaviour Rating Inventory of Executive Function – Global Executive Composite; DV: Dependent variable (RCADS-25); IV: Independent variable (group status); M: Mediator (BRIEF GEC); RCADS-25: Revised Child Anxiety and Depression Scale – Short version.

Post-hoc analyses

Exploratory analyses were conducted using the subscales of the RCADS-25 and BRIEF. Independent samples \(t\)-tests were conducted to compare the two groups. Given that the distribution of the variables deviated from normality (see Appendix M), bootstrapping with 10,000 samples was used to generate bias-corrected and accelerated 95% confidence intervals for the mean differences.
Figure 2. Results of the mediation analysis using the RCADS-25 Total scores and BRIEF GEC

**RCADS-25 subscales.** On the RCADS-25, adolescents in the DCD group reported greater levels of depression ($M = 54.12; SE = 2.58$) compared to adolescents in the TD group ($M = 41.19; SE = 1.73$). This difference, 12.93, BCa 95% CI [6.78, 18.78], was significant, $t(41) = 4.21, p < .001$, and represents a large effect size, $d = 1.32$.

Adolescents in the DCD group also reported greater levels of anxiety ($M = 50.49; SE = 3.43$) compared to adolescents in the TD group ($M = 44.19; SE = 2.01$). This difference, 6.30, BCa 95% CI [-0.91, 13.64], was not significant $t(41) = 1.68, p = .100$. However, it did represent a medium effect size ($d = 0.52$).

**BRIEF subscales.** On the BRIEF, adolescents in the DCD group had greater levels of parent-reported behavioural regulation difficulties ($M = 75.36; SE = 2.37$) compared to adolescents in the TD group ($M = 48.28; SE = 1.74$). This difference, 27.08, BCa 95% CI [21.42, 32.75], was significant, $t(41) = 9.03, p < .001$, and represents a
large effect size, $d = 2.82$. Adolescents in the DCD group also had greater levels of parent-reported metacognitive difficulties ($M = 74.50; SE = 1.93$) compared to adolescents in the TD group ($M = 46.99; SE = 1.70$). This difference, 27.53, BCa 95% CI [22.31, 32.44], was significant $t(41) = 9.87, p < .001$, and represents a large effect size ($d = 3.08$).

**Subscale mediator models.** Two parallel multiple mediation models were conducted to explore the relationship between DCD status and each subscale of the RCADS-25 (Depression and Anxiety) separately. For both models, the separate subscales on the BRIEF (BRIEF BRI and BRIEF MCI) were included as parallel mediators (path $ab_1$ and $ab_2$, respectively). This allowed further exploration of the potential paths through which EF difficulties mediate the relationship between DCD and internalising symptoms. The relevant assumptions were confirmed for each model prior to analysis (see Appendix N). The results are summarised in Table 5.

**Depression symptoms:** A parallel multiple mediation model was conducted to explore the effect of DCD status on depression through both behavioural regulation difficulties and metacognitive difficulties. Bias-corrected bootstrap confidence intervals for the indirect effects, based on 10,000 bootstrap samples, found a significant indirect effect of behavioural regulation difficulties, $a_1 = 9.77$, 95% CI [1.81, 17.74], but not metacognitive difficulties, $a_2 = 2.05$, 95% CI [-8.59, 12.87]. The partially standardised indirect effect through behavioural regulation difficulties was 0.88, BC 95% CI [0.12, 1.56]. The findings did not change when investigating the mediators independently in two simple mediation models.
**Anxiety symptoms:** Although no significant difference between the two groups in levels of anxiety symptoms was identified, evidence of an association between the independent and dependent variables is not a requirement for mediation (Zhao, Lynch, & Chen, 2010). Therefore, a parallel multiple mediation model was also conducted to explore the effect of DCD status on anxiety through both behavioural regulation difficulties and metacognitive difficulties. Bias-corrected bootstrap confidence intervals for the indirect effects, based on 10,000 bootstrap samples, crossed zero for both behavioural regulation difficulties, $a_1 = 8.84$, 95% CI [-3.09, 20.48] and metacognitive difficulties, $a_2 = 6.07$, 95% CI [-8.82, 21.84], suggesting no significant indirect effect.

Table 5. Results of the parallel multiple mediation models using the subscales of the BRIEF and RCADS-25

<table>
<thead>
<tr>
<th>DV: RCADS-25 Depression</th>
<th>B coefficient</th>
<th>SE</th>
<th>t</th>
<th>p-value</th>
<th>Bias-corrected bootstrap 95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Path $a_1$: IV-M$_1$</td>
<td>27.08</td>
<td>3.00</td>
<td>9.03</td>
<td>&lt;.001</td>
<td></td>
</tr>
<tr>
<td>Path $b_1$: M$_1$-DV</td>
<td>0.36</td>
<td>0.17</td>
<td>2.13</td>
<td>.040</td>
<td></td>
</tr>
<tr>
<td>Path $a_2$: IV-M$_2$</td>
<td>27.53</td>
<td>2.79</td>
<td>9.87</td>
<td>&lt;.001</td>
<td></td>
</tr>
<tr>
<td>Path $b_2$: M$_2$-DV</td>
<td>0.07</td>
<td>0.18</td>
<td>0.41</td>
<td>.686</td>
<td></td>
</tr>
<tr>
<td>Path $c$: Total Effect</td>
<td>12.93</td>
<td>3.07</td>
<td>4.21</td>
<td>&lt;.001</td>
<td></td>
</tr>
<tr>
<td>Path $c'$: Direct effect</td>
<td>1.11</td>
<td>5.81</td>
<td>0.19</td>
<td>.849</td>
<td></td>
</tr>
<tr>
<td>$a_1b_1$: Indirect effect</td>
<td>9.77</td>
<td></td>
<td></td>
<td></td>
<td>[1.81, 17.74]*</td>
</tr>
<tr>
<td>$a_2b_2$: Indirect effect</td>
<td>2.05</td>
<td></td>
<td></td>
<td></td>
<td>[-8.59, 12.87]</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>DV: RCADS-25 Anxiety</th>
<th>B coefficient</th>
<th>SE</th>
<th>t</th>
<th>p-value</th>
<th>Bias-corrected bootstrap 95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Path $a_1$: IV-M$_1$</td>
<td>27.08</td>
<td>3.00</td>
<td>9.03</td>
<td>&lt;.001</td>
<td></td>
</tr>
<tr>
<td>Path $b_1$: M$_1$-DV</td>
<td>0.33</td>
<td>0.21</td>
<td>1.56</td>
<td>.126</td>
<td></td>
</tr>
<tr>
<td>Path $a_2$: IV-M$_2$</td>
<td>27.53</td>
<td>2.79</td>
<td>9.87</td>
<td>&lt;.001</td>
<td></td>
</tr>
<tr>
<td>Path $b_2$: M$_2$-DV</td>
<td>0.22</td>
<td>0.22</td>
<td>0.98</td>
<td>.333</td>
<td></td>
</tr>
<tr>
<td>Path $c$: Total Effect</td>
<td>6.30</td>
<td>3.74</td>
<td>1.68</td>
<td>.100</td>
<td></td>
</tr>
<tr>
<td>Path $c'$: Direct effect</td>
<td>-8.61</td>
<td>7.15</td>
<td>-1.20</td>
<td>.236</td>
<td></td>
</tr>
<tr>
<td>$a_1b_1$: Indirect effect</td>
<td>8.84</td>
<td></td>
<td></td>
<td></td>
<td>[-3.09, 20.48]</td>
</tr>
<tr>
<td>$a_2b_2$: Indirect effect</td>
<td>6.07</td>
<td></td>
<td></td>
<td></td>
<td>[-8.82, 21.84]</td>
</tr>
</tbody>
</table>

* 95% bias-corrected bootstrapped confidence interval significant
TD sample. Due to the small sample in the DCD group and the potential heterogeneity between the two groups due to differing sampling strategies, the mediation analysis was repeated in the TD group only (see Table 6). For this model, the total score on the DCDQ-07 was used as a continuous independent variable. The three TD participants excluded from the main analysis due to scoring below the cut-off on the DCDQ-07 were included in this analysis \((n = 32);\) see Appendix O for exploration of assumptions. The results found that poorer motor coordination in TD adolescents was associated with higher levels of self-reported internalising symptoms, \(c = -0.53, p = .028,\) and higher levels of parent-reported EF difficulties, \(a = -0.66, p < .001.\) TD adolescents with higher levels of parent-reported EF difficulties also self-reported higher levels of internalising symptoms after controlling for motor coordination, \(b = 0.71, p = .001.\) A bias-corrected bootstrap confidence interval for the indirect effect, based on 10,000 bootstrap samples, did not cross zero, \(ab = -0.47, BC 95\%\; CI [-0.84, -0.16].\) The direct effect of motor coordination on internalising symptoms was also no longer significant after accounting for EF difficulties, \(c' = -0.06, p = .787.\) The completely standardised indirect effect was \(-0.34, 95\%\; BC\; CI [-0.57, -0.13].\) These results indicate that, in TD adolescents, a decrease of one standard deviation in motor coordination skills is associated with, on average, an increase of 0.34 standard deviations in self-report internalising symptoms because of the indirect effect through parent-reported EF difficulties.
Table 6. Results of the mediation analysis based on the TD group only

<table>
<thead>
<tr>
<th></th>
<th>B coefficient</th>
<th>SE</th>
<th>t</th>
<th>p-value</th>
<th>Bias-corrected bootstrap 95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Effect of IV on M (Path a)</td>
<td>-0.66</td>
<td>0.18</td>
<td>-3.60</td>
<td>.001</td>
<td></td>
</tr>
<tr>
<td>Effect of M on DV (Path b)</td>
<td>0.71</td>
<td>0.19</td>
<td>3.70</td>
<td>.001</td>
<td></td>
</tr>
<tr>
<td>Total Effect (Path c)</td>
<td>-0.53</td>
<td>0.23</td>
<td>-2.31</td>
<td>.028</td>
<td></td>
</tr>
<tr>
<td>Direct effect (Path c’)</td>
<td>-0.06</td>
<td>0.23</td>
<td>-0.27</td>
<td>.787</td>
<td></td>
</tr>
<tr>
<td>Indirect Effect (a x b)</td>
<td>-0.47</td>
<td></td>
<td></td>
<td></td>
<td>[-0.84, -0.16]*</td>
</tr>
</tbody>
</table>

* 95% bias-corrected bootstrapped confidence interval significant

BRIEF: Behaviour Rating Inventory of Executive Function; DV: Dependent variable (Revised Child Anxiety and Depression Scale – Short version); IV: Independent variable (Developmental Coordination Disorder Questionnaire – 07 Total Score); M: Mediator (Behaviour Rating Inventory of Executive Function).

Discussion

The aim of this study was to investigate internalising symptoms and EF difficulties in adolescents with and without DCD. As hypothesised, adolescents with DCD had greater levels of self-reported internalising symptoms (Hypothesis 1) and greater levels of parent-reported EF difficulties (Hypothesis 2) compared to TD adolescents. The results also identified that parent-reported EF difficulties mediated the relationship between DCD status and internalising symptoms (Hypothesis 3). Further exploratory analyses identified that the difference in internalising symptoms between the two groups was greatest for symptoms of depression and that this effect was mediated by behavioural regulation difficulties, but not metacognitive difficulties.

Internalising symptoms in DCD

The findings replicate previous research identifying that individuals with DCD experience greater levels of internalising symptoms than their TD peers (Mancini et al., 2016; Missiuna & Campbell, 2014, see Part 2 for a review). More specifically, the
findings are consistent with the few existing studies that have investigated internalising symptoms in adolescents with the disorder (Harrowell et al., 2017; Li et al., 2018; Skinner & Piek, 2001; Wagner et al., 2016). The magnitude of this effect was also large, with adolescents in the DCD group scoring almost one standard deviation higher on self-reported internalising symptoms compared to the TD adolescents. This difference can be considered clinically significant (Norman, Sloan, & Wyrwich, 2003) and would equate to the adolescents with DCD reporting one point higher on at least ten items of the RCADS-25.

However, the magnitude of the difference in the present study was greater than might be expected based on past research, since meta-analytic findings suggest the difference to be around half a standard deviation (see Part 2). It is likely that the effect in the present study was inflated due to the use of convenience sampling, which has been found to result in greater effect sizes (Crane, Sumner, & Hill, 2017; Hill & Brown, 2013; Pratt & Hill, 2011; Wagner, Bos, Jascenoka, Jekauc, & Petermann, 2012; Watson & Knott, 2006) compared to studies using a population-based screening strategy (Campbell et al., 2012; Francis & Piek, 2003; Harrowell et al., 2017; Missiuna et al., 2014; Skinner & Piek, 2001; Wagner et al., 2016). Indeed, the TD group in the present study reported significantly lower internalising symptoms than the normative population (Chorpita, Moffitt, & Gray, 2005) with no participants scoring above the clinical threshold, suggesting it was an unrepresentative sample. Despite the potentially inflated effect size, the direction of the effect is consistent with higher quality studies suggesting individuals with DCD experience greater levels of internalising symptoms than their TD peers (Harrowell et al., 2017; Wagner et al., 2016).
These findings are in line with the growing consensus that the impact of DCD extends beyond the core motor difficulties that characterise the disorder and includes significant implications for the mental health of those affected. The environmental-stress hypothesis (Cairney et al., 2013) would suggest that the motor difficulties experienced by the DCD group impacts on their mental health indirectly through a variety of secondary psychosocial stressors. Although these stressors were not measured in the present study, they might include increased peer victimisation (Campbell et al., 2012), reduced leisure activities (Raz-Silbiger et al., 2015), poorer self-esteem (Rigoli, Piek, & Kane, 2012), physical inactivity (Li et al., 2018), reduced social support (Rigoli et al., 2017), and lower perceived academic performance (Lingam et al., 2012).

Interestingly, the findings revealed that the two groups differed significantly on symptoms of depression but not anxiety. This contradicts previous research that has found DCD to impact on specific measures of both depression (Campbell et al., 2012; Francis & Piek, 2003; Harrowell et al., 2017; Hill & Brown, 2013; Missiuna et al., 2014; Piek et al., 2007; Watson & Knott, 2006) and anxiety (Hill & Brown, 2013; Missiuna et al., 2014; Pratt & Hill, 2011; Schoemaker & Kalverboer, 1994). The failure to detect a difference in anxiety symptoms likely reflects insufficient power in the present study, since a medium effect size was still found. However, these preliminary findings do suggest that the impact of DCD on internalising symptoms in adolescents may be greater for depressive symptoms than anxiety symptoms. Indeed, Missiuna et al. (2014) reported a greater effect size for self-reported depressive symptoms than for self-reported anxiety symptoms in adolescents with DCD compared to their TD peers. This suggestion would fit with findings that some of the secondary psychosocial stressors proposed in the
environmental-stress hypothesis are more often linked with depression than they are with anxiety, including reduced self-esteem (Sowišlo & Orth, 2013) and peer victimisation (Hawker & Boulton, 2000).

**EF difficulties in DCD**

The present findings are also consistent with previous research highlighting EF deficits in DCD (Alloway, 2007; Bernardi et al., 2018; Leonard & Hill, 2015; Mandich et al., 2002; P. H. Wilson et al., 2013; Wuang et al., 2011). The DCD group was found to have significantly higher levels of EF difficulties for both behavioural regulation and metacognitive functions. This is in line with past research highlighting difficulties for people with DCD across a wide range of EF task, including inhibition, cognitive flexibility, working memory and planning (Leonard & Hill, 2015). These difficulties can be understood through the shared underlying neurological mechanisms for both motor skills and EFs, which include the prefrontal cortex and cerebellum (Diamond, 2000). As such, the underdevelopment of motor skills that characterise DCD are closely interrelated with the EF deficits (Koziol et al., 2012; Rigoli, Piek, Kane, & Oosterlaan, 2012).

This study is also the first to identify EF difficulties in adolescents with DCD using a behaviour rating scale of EF. This suggests that the EF deficits observed on performance-based measures in adolescents with DCD also generalises to EF difficulties in everyday life, supporting similar findings in an adult sample (Tal Saban et al., 2014). The effect size in the present study was also very large, with parent-reported EF difficulties in the DCD group being over 3 standard deviations higher than in the TD
group. Although a large effect was expected based on past research (Tal Saban et al., 2014; P. H. Wilson et al., 2013), the magnitude was even greater in the present study. It is again possible that the effect was inflated due to design limitations, especially the use of a convenience sampling strategy. However, these preliminary findings might suggest that the EF difficulties experienced by adolescents with DCD may be even more marked in day-to-day life than has been previously identified on highly structured, laboratory-based tasks.

**Internalising symptoms in DCD are mediated by EF difficulties**

A notable finding in the present study is that the elevated internalising symptoms in adolescents with DCD, compared to their TD peers, was mediated by EF difficulties. This is consistent with expectations, given evidence that EF deficits are associated with mental health difficulties (Evans et al., 2016, 2016; Han et al., 2016; Lengua, 2006; Letkiewicz et al., 2014; Martel et al., 2007; Nigg et al., 1999; Riggs et al., 2003). This mediating effect also sits alongside the findings of research with other childhood disorders that impact on both EFs and internalising symptoms (Kelly et al., 2012; Lawson et al., 2015). Research has identified that EFs are important for the effective use of appropriate emotion regulation strategies (Joormann & D’Avanzato, 2010; Schmeichel & Tang, 2015). It is possible that the EF impairments in the adolescents with DCD contributed to an increased use of maladaptive emotion regulation strategies such as rumination (Demeyer et al., 2012; Johnson, 2009; Meiran et al., 2011), worry (Brosschot et al., 2006) and withdrawal (Wante et al., 2017), and a reduced use of adaptive strategies such as reappraisal (Joormann & Gotlib, 2010) and distraction.
(Wante et al., 2017). This, in turn, may have contributed to increased levels of internalising symptoms. EF deficits may also have impacted on other areas of functioning, including academic performance (Best et al., 2011) and social functioning (Dawson et al., 2012), which may have contributed to elevated internalising symptoms (Ende et al., 2016; Nilsen et al., 2013; Zhang et al., 2018).

It is also of note that, after accounting for the indirect effect through EF difficulties, the direct effect of DCD on internalising symptoms was no longer significant. Additionally, the effect size of the mediation analysis indicated that the adolescents with DCD reported internalising symptoms that were over one standard deviation higher, compared to TD adolescents, due to the indirect effect through EF difficulties. This highlights that the role of EF deficits in explaining internalising symptoms in DCD may be a major one. Yet previous attempts to explain the increased risk of internalising symptoms in DCD (i.e. the environmental-stress hypothesis) have failed to include these higher-order cognitive abilities in their model (Cairney et al., 2013; Mancini et al., 2016). The environmental-stress hypothesis may benefit from the inclusion of EFs as another mediating factor between DCD and internalising symptoms. This might be through previously untested pathways, including its impact on emotion regulation, academic functioning or social skills. Additionally, it is possible that EFs may moderate and mediate the other identified pathways in the model. For example, EFs may influence self-perceptions (Hughes & Ensor, 2011), social support (Beauchamp & Anderson, 2010) and resilience against peer victimisation (Agoston & Rudolph, 2016).

Exploratory analyses identified that the indirect effect can be best explained as DCD impacting on depressive symptoms through behavioural regulation difficulties. No
indirect effect was found for depression through metacognitive difficulties. Additionally, no indirect effect was found for anxiety through either type of EF. The failure to find an indirect effect through these other paths could be due to insufficient power in the present study, since only a medium-large indirect effect could be detected with the sample size. However, the results would suggest that difficulties in behavioural regulation may be most pertinent to the mental health difficulties in DCD, especially depression. To some extent, these findings would fit with the proposed explanation that emotion regulation deficits underlie the link between EF difficulties and internalising symptoms. This is because the BRI on the BRIEF measures a person’s ability to shift and modulate behaviours and emotions via appropriate inhibitory control (Gioia et al., 2000). Emotional control is a key aspect of the BRI, as is inhibition and cognitive flexibility which have been implicated in emotion regulation strategies (Demeyer et al., 2012; Johnson, 2009; Joormann & Gotlib, 2010; Meiran et al., 2011). The MCI, on the other hand, measures a person’s cognitive ability to problem-solve, monitor performance and self-manage tasks. There is generally less research linking these metacognitive abilities to emotion regulation. Instead, metacognitive abilities may be important for more practical and social skills (Gilotty, Kenworthy, Sirian, Black, & Wagner, 2002; Pugliese et al., 2015). Consistent with this, Gardiner and Iarocci (2018) found that behavioural regulation difficulties predicted depressive symptoms in adolescents, whereas metacognitive difficulties were a better predictor of daily living skills, communication skills and socialisation abilities. It is, therefore, a plausible finding that deficits in EFs related to behavioural regulation are most important in explaining the link between DCD and internalising symptoms, particularly depression.
Finally, EFs were also found to mediate the relationship between motor
difficulties and internalising symptoms in the TD adolescents. This is consistent with the
emerging literature investigating motor difficulties as a dimensional construct in
community samples, including its relationship with internalising symptoms (Piek et al.,
2010; Poole et al., 2015; Sigurdsson E et al., 2002) and EF difficulties (Rigoli, Piek,
Kane, et al., 2012). This would suggest that subclinical symptoms of DCD, and motor
difficulties in general, can impact on internalising symptoms and that this may also be
mediated by EF difficulties. This finding also provides extra support for the main results
of this study. It suggests that the findings cannot be explained solely by the potential
bias arising through the unrepresentativeness of the TD sample.

**Strengths and Limitations**

There are several limitations in the present study and so the results should be
interpreted with caution. Given the cross-sectional design, it is not possible to establish
causality in the relationships identified. Although prior longitudinal research suggests
that DCD predicts both EF difficulties and internalising symptoms, and that EF
difficulties predict internalising symptoms, these directional relationships could not be
confirmed in the present study. For example, there is evidence that internalising
symptoms can exacerbate EF difficulties (Vilgis, Silk, & Vance, 2015). Alternatively,
the relationship between variables might be better explained by their shared associations
with another untested variable.

Despite attempts to recruit participants across a wide range of sources, the
response rate in both the DCD and TD groups was very low. This resulted in a small
sample size with insufficient power to detect medium effect sizes. As such, interpretations could only be made about the larger effects and are somewhat tentative. The low response rate, and the use of a convenience sampling strategy, also raises the question of how representative the sample was of the populations studied. It is possible that only a certain subgroup of parents and adolescents volunteered to participate in the study. In the DCD group, this may have included parents and adolescents who were closely involved with the organisations that aided in advertising the study. Given the supportive nature of these organisations, these volunteers may benefit from greater protective resources than those with no links to such support. Indeed, the internalising symptoms in the DCD group were not found to differ significantly from the normative population, suggesting the difficulties in mental health were less apparent than might be expected in the DCD population. On the contrary, internalising symptoms in the TD group were significantly lower than the normative population, suggesting the sample was unrepresentative. The use of a convenience sampling strategy likely provided less accurate estimates of the effects compared to a population screening strategy.

Although the two groups did not differ significantly in age and gender, and the results did not change when controlling for these confounders, other untested confounders may have influenced the results. For example, socioeconomic status (SES) was not measured, though it has been linked to both internalising symptoms (Letourneau, Duffett-Leger, Levac, Watson, & Young-Morris, 2013) and EFs (Hackman & Farah, 2009). However, the reported effects of SES are small and inconclusive (Gioia et al., 2000; Twenge & Nolen-Hoeksema, 2002). Other factors that could potentially be important are maternal depression (Harrowell et al., 2017), ethnicity (Twenge & Nolen-
Hoeksema, 2002) and IQ (Lingam et al., 2012), though the use of IQ as a covariate in studies investigating neurodevelopmental disorders is controversial (Dennis et al., 2009). These potential confounding factors often go unmeasured in research into DCD since recruitment is often challenging and the primary measures take priority. Additionally, although adolescents with comorbid disorders were excluded based on self-reported diagnoses, it is also unclear to what extent undiagnosed difficulties or subclinical symptoms of ADHD or ASD could have contributed to the findings.

Another limitation is that internalising symptoms were based only on self-report and EF difficulties on parent-report. Although clinical cut-offs could be used, only diagnostic interviews could establish the rates of psychiatric diagnoses in each group. Additionally, behaviour rating scales of EF only correlate modestly with performance-based measures of EF. It is, therefore, unclear if similar findings would be found using more structured, controlled measures of EF. Related to this, the present study relied on parent-report and a screening measure to rule out a diagnosis of DCD in the TD group. A more rigorous, independent assessment based on performance-based measures and a clear developmental history would be more accurate. This is especially important given that DCD is often undiagnosed (Gaines et al., 2008), placing greater needs on researchers to assess this independently.

However, this is the first study to investigate whether difficulties in EFs mediate the relationship between DCD and internalising symptoms. This is an important factor that has not been included in previous explanations for this relationship (Cairney et al., 2013; Mancini et al., 2016). It is also the first study to investigate everyday EF difficulties in adolescents with DCD compared to their TD peers. The use of different
respondents for the mediator and dependent variables minimises common method bias which is often a major limitation among studies investigating mediation models. Generally, the effect sizes were also of a large magnitude, suggesting that further exploration of these relationships in future research could have important theoretical and clinical implications.

**Clinical implications**

These findings echo previous research indicating that the impact of DCD extends beyond the motor difficulties that characterise the disorder. They highlight the need for healthcare and education professionals to become more aware of the impact the disorder has on both internalising symptoms and on EF. This is especially important given that the condition is poorly understood among professionals (Gaines et al., 2008; B. N. Wilson et al., 2013) and given the difficulties families report in obtaining the right support (Stephenson & Chesson, 2008). It suggests that individuals with DCD would benefit from further assessment of their EF abilities and appropriate support with this in both education and healthcare settings. Indeed, individuals diagnosed with DCD in adulthood have highlighted their EF difficulties as being amongst their main concerns and reasons for seeking support, as opposed to the motor difficulties itself (Purcell, Scott-Roberts, & Kirby, 2015). The findings also support the practice of routine screening of mental health difficulties in individuals with DCD to identify potentially undiagnosed comorbid difficulties, especially depression. Given the numerous pathways through which DCD might impact on internalising symptoms (Mancini et al., 2016), these suggestions would require close collaboration among teachers, educational
psychologists, occupational therapists, medical practitioners, social workers and mental health professionals to meet an individual’s needs holistically.

The findings also suggest that EF deficits may play a key role in the increased risk of internalising symptoms in people with DCD. Given that research has found interventions can improve EFs if targeted during childhood (Diamond & Lee, 2011), this provides an important area of treatment for individuals with DCD and comorbid mental health difficulties. Although the findings are only preliminary, it would suggest that this may be most important for depressive symptoms in adolescents with DCD. It also suggests that a focus on behavioural regulation strategies may be most effective. This might include learning to shift attention away from negative emotional stimuli (e.g. negative thoughts), inhibit negative behavioural and emotional responses, and develop more effective emotion regulation strategies. The findings would also suggest that, for mental health professionals presented with a client with DCD and comorbid depression, it would be helpful to be aware of these behavioural regulation difficulties and perhaps consider them as part of their intervention. For example, there is some suggestion that third-wave cognitive-behavioural models may improve behavioural regulation, including elements of mindfulness training (Flook et al., 2010), promoting psychological flexibility (Whiting, Deane, Simpson, McLeod, & Ciarrochi, 2017), and developing emotion regulation strategies (McMain, Korman, & Dimeff, 2001). On the contrary, attempts to improve metacognitive skills such as problem-solving, planning and organisation may be less helpful.
Future research

Given the limitations of the present study, further research is needed to support the current findings. Longitudinal studies, controlling for the stability of EF difficulties and internalising symptoms over time, are necessary to better understand the causal pathways linking DCD, EFs, and internalising symptoms. Future research could also explore the specific pathways through which EF difficulties lead to internalising symptoms in DCD. This might include indirect effects through emotion regulation strategies and through the existing pathways suggested in the environmental stress hypothesis (e.g. peer victimisation, social support, self-esteem). More complex analytical techniques, such as structural equation modelling, could provide further insight into the complexity of the relationships between these variables. These future studies should also adopt large-scale, population-based screening strategies controlling for a range of potential confounders to maximise the quality of their findings. The addition of performance-based measures of various EFs would also allow for more in-depth analyses into the specific EF abilities that are important in these relationships. It would also be important to replicate findings across the age groups, including younger children and adults. Additionally, there is a need for intervention studies to determine whether targeting EF deficits in DCD can improve the mental health of this population.

Conclusion

This study supports the findings of previous research indicating that adolescents with DCD experience greater levels of internalising symptoms and EF difficulties compared to their TD peers. It is also the first study to identify EF difficulties as a key
explanatory factor in the link between DCD and mental health problems. Although these findings are considered preliminary, it suggests that EF difficulties could be an important addition to previous attempts at conceptualising the elevated internalising symptoms in this population (Cairney et al., 2013; Mancini et al., 2016). It also highlights EFs as a potential intervention target for improving the emotional wellbeing of adolescents with DCD. However, further research is needed to support these findings.

References


switching, Stroop, working memory updating and post-conflict adaptation.

*Psychiatry Research, 185*(1–2), 149–156.


*Depression Research and Treatment, 2011*, 487873.


functioning and attention in school aged children. *Archives of Clinical Neuropsychology, 19*(8), 1063–1076.


implications for treatment in acceptance-based therapies: A conceptual review.

*Neuropsychological Rehabilitation, 27*(2), 263–299.


List of appendices

Appendix A: Guidelines for authors 71
Appendix B: Developmental Coordination Disorder Questionnaire – 07 (DCDQ-07) 72
Appendix C: Revised Child Anxiety and Depression Scale – Short version (RCADS-25) 73
Appendix D: Behaviour Rating Inventory of Executive Function (BRIEF) 74
Appendix E: Study advertisement 75
Appendix F: Information sheets 77
Appendix G: Parent consent forms 90
Appendix H: Adolescent assent forms 93
Appendix I: Eligibility screening questionnaire 95
Appendix J: Favourable ethical opinion 96
Appendix K: Assumptions for independent samples t-tests 97
Appendix L: Assumptions for simple mediation analysis 102
Appendix M: Assumptions for subscale analyses using independent samples t-tests 107
Appendix N: Assumptions for parallel multiple mediation analysis based on subscales 114
Appendix O: Assumptions for simple mediation analysis within the TD sample 117
Appendix A: Guidelines for authors

[Appendix removed for E-Thesis submission]
Appendix B: Developmental Coordination Disorder Questionnaire – 07 (DCDQ-07)

[Appendix removed for E-Thesis submission]
Appendix C: Revised Child Anxiety and Depression Scale – Short version

(RCADS-25)

[Appendix removed for E-Thesis submission]
Appendix D: Behaviour Rating Inventory of Executive Function (BRIEF)

[Appendix removed for E-Thesis submission]
Appendix E: Study advertisements

A copy of the advertisement distributed for participants with DCD is displayed below:

---

**University of Surrey research**

We are researchers at the University of Surrey looking to understand the mental health and thinking abilities in adolescents with and without Developmental Coordination Disorder (also known as Dyspraxia).

In this study, we will ask parents to complete some questionnaires about their child’s motor ability and thinking abilities (e.g. planning and memory). Your child will also be asked to complete a questionnaire about their mental health.

Your child can take part if he/she is aged between 12 and 15 and has a diagnosis of Developmental Coordination Disorder / Dyspraxia. All your responses will be anonymous and you will be given the chance to enter a raffle to win a £100 Amazon voucher!

If interested, please email Serif at s.omer@surrey.ac.uk for more information.

This study has received a favourable ethical opinion from the Faculty of Health and Medical Sciences Ethics Committee, at the University of Surrey

---
University of Surrey research

Hi! My name is Serif Omer. We are conducting a research project to find out more about the link between motor coordination, thinking skills, and mental health. As part of this project, we are looking for young people (age 12-15) and their parents/guardians to complete some brief online questionnaires. They take about 15 minutes for parents/guardians and 5 minutes for the young person. All your responses will be anonymous and you will be given the chance to enter a raffle to win a £100 Amazon voucher!

Please visit the following web link to access the survey:

https://surreyfahs.eu.qualtrics.com/jfe/form/SV_a2wl5KRUw0vNXX7

Paper copies can also be provided. Please feel free to contact me if you have any questions: s.omer@surrey.ac.uk.

Thank you very much!
Appendix F: Information sheets

Separate information sheets were produced for parents and adolescents in each study group. Copies of these information sheets are provided on the following pages.
Title of Project

Executive function and internalizing problems in adolescents with and without Developmental Coordination Disorder

Introduction

We would like to invite you and your child to take part in a research project. Before you and your child decide to take part, you need to understand why the research is being done and what it will involve. Please take the time to read the following information carefully. Talk to others about the study if you wish.

This study is being conducted by Serif Omer (Trainee Clinical Psychologist), under the supervision of Dr Hayley Leonard (Lecturer in Developmental Psychology and Neurodevelopmental Disorders). Please ask questions if there is anything you do not understand.

What is the purpose of the study?

We know that adolescents with Developmental Coordination Disorder (DCD; sometimes also called ‘Dyspraxia’) often have difficulties with high-level thinking abilities, such as planning and memory. They also often report increased symptoms of anxiety and depression compared to others their age without the disorder. The current project aims to investigate this further by understanding how these difficulties may be related to each other.

Why have I been invited to take part in the study?

As part of this study, we are seeking adolescents (between the age of 12 and 16) with a diagnosis of DCD / dyspraxia and one of their parents/guardians. You and your child have been invited to take part because you might meet these criteria.

Do I have to take part?

No, neither you nor your child has to participate. There will be no adverse consequences if you decide not to participate. You can also withdraw your participation at any time without giving a reason.
What will my involvement require?

As the child’s parent or guardian, you will be asked to complete a consent form. You will then be asked a set of questions. These can be completed online, by post, over the phone, or face-to-face if we visit you at home. This should take no more than 30 minutes in total and will include questions about your child’s movement abilities (such as sports skills, handwriting, etc.) and their high-level thinking skills (such as planning and memory).

You will also be asked to provide a copy of the clinical report confirming your child’s diagnosis of DCD. This will be checked by the research team to ensure your child is eligible and any relevant scores on diagnostic tests will be recorded.

You will not receive any payment for taking part. You will be given the option at the end of the survey to enter a prize draw for a £100 Amazon voucher. The winner will be contacted by email at the end of study in April 2018.

What will my child’s involvement require?

If you consent for your child to take part and they are suitable for the study, then they will be asked to independently complete a questionnaire concerning their moods and emotions. It should take approximately 10 minutes and can be completed online, over the phone, or on paper.

Your child will not receive any payment for taking part.

What will I have to do?

If you and your child would like to take part, please contact the research team on s.omer@surrey.ac.uk (Serif Omer) or using the contact details below. A researcher will then be in contact to obtain consent and administer the questionnaires.

Please feel free to contact the research team if you have any questions.

What are the possible disadvantages or risks of taking part?

There is a possibility that some of the questions may highlight sensitive issues for you or your child. If you want to talk about the questions or your answers, please contact the research team. If your child appears to experience any distress, the session will be terminated immediately and the research team will alert you.

What are the possible benefits of taking part?

It is unlikely that you or your child will benefit directly from taking part. However, it is hoped that the study will improve our understanding of mental health and
thinking abilities in people with motor coordination difficulties, which could have clinical implications for improving the lives of this group of people.

What happens when the research study stops?

You may receive a summary of the research findings at the end of the study (after February 2018). You can indicate if and how you would like to receive this on the initial screening questionnaire. This summary will only include the overall findings of the study. We will not provide individual feedback on your child’s or your own responses.

What if there is a problem?

Any complaint or concern about any aspect of the way you have been dealt with during the course of the study will be addressed; please contact Dr Hayley Leonard (Research Supervisor) on 01483 686888 or h.leonard@surrey.ac.uk. You may also contact the Head of School, Professor Derek Moore, on 01483 686933 or d.g.moore@surrey.ac.uk.

Will taking part in the study be kept confidential?

Yes. All of the information that you and your child give will be anonymised so that those reading reports from the research will not know who has contributed to it.

All project data will be held for at least 6 years and all research data for at least 10 years in accordance with University policy. Data will be stored securely in accordance with the Data Protection Act 1998.

However, should you or your child disclose information that suggests your child is at risk then the researcher may need to report this to an appropriate authority. This would usually be discussed with you first.

Contact details

Serif Omer
Trainee Clinical Psychologist
School of Psychology
AD Building
University of Surrey
Guildford
GU2 7XH
s.omer@surrey.ac.uk

Dr Hayley Leonard
Lecturer
School of Psychology
AD Building
University of Surrey
Guildford
GU2 7XH
h.leonard@surrey.ac.uk
01483 686888

Who is organising and funding the research?
This research is unfunded and is being completed by Serif Omer as part of a Doctorate in Clinical Psychology at the University of Surrey.

**Who has reviewed the project?**

The study has been reviewed and received a Favourable Ethical Opinion (FEO) from the Faculty of Health and Medical Sciences Ethics Committee, at the University of Surrey.

**Thank you for taking the time to read this Information Sheet.**
Adolescent Information Sheet (DCD group; 17.11.17; Version 2)

Title of Project

Executive function and internalizing problems in adolescents with and without Developmental Coordination Disorder

Introduction

Hi, my name is Serif Omer and I would like to invite you to take part in a research study. At the University of Surrey, we are trying to learn more about the effect of Developmental Coordination Disorder on young people. People with Developmental Coordination Disorder (DCD), which is sometimes called 'Dyspraxia', have difficulties with movement tasks (such as handwriting and sports). However, they may also have difficulties with their mood and some thinking skills (such as memory and planning ahead). We would like to know more about whether young people with DCD have differences in their mood and thinking skills compared to young people without DCD and why this might be.

Why is this study being done?

We hope that this research will help to better understand the effect of DCD/dyspraxia on young people which could help to improve their lives. This research could also help to better understand difficulties with mood and thinking skills in young people that do not have DCD.

Why have I been invited to take part?

As part of this study, we are looking for young people with a diagnosis of DCD/dyspraxia between the age of 12 and 16. You have been invited to take part because you might be suitable. Your parent/guardian has also agreed for you to take part.

Do I have to take part?

Remember, being in this study is up to you. Even if your parents give their permission for you to participate in this study, you still can decide for yourself if you want to take part. You don't have to be in this study if you don't want to! Even if you agree to take part in the study, you can still stop at any time without giving a reason. If you decide not to take part, this will not affect you negatively in any way.

What will happen if I take part?

If you agree to be in this study, we will ask you to sign a consent form. We will then ask you to complete a questionnaire which asks you about your mood and emotions. It should take approximately 10 minutes and can be completed online, over the phone, or on paper.
Unfortunately, you will not receive any money or reward for taking part. Your parent/guardian will be given the option at the end of the survey to enter a prize draw for a £100 Amazon voucher. The winner will be contacted by email at the end of study in April 2018.

**Will information about me be available to anyone?**
The information that we collect from you is confidential and anonymous. Only members of our team will be able to see this information. Your school and your parents/guardians will not have access to the information we collect. In our findings we will discuss the results of many young people - we do not single out or name any one participant.

The only other time that we will tell somebody else about something you have said is if we think you or someone else may be at risk of harm.

This research study has been reviewed and given a Favourable Ethical Opinion by Faculty of Health and Medical Sciences Ethics Committee, at the University of Surrey. This means that the study has been checked and it is ethical for it to be conducted.

**What if I have a question or there is a problem?**
You can ask any questions that you have about the study at any time. If you have a question you can contact me or other people in my team using the contact details below.

**Serif Omer**
Trainee Clinical Psychologist
School of Psychology
AD Building
University of Surrey
Guildford
GU2 7XH
s.oemer@surrey.ac.uk
01483 686888

**Dr Hayley Leonard**
Lecturer
School of Psychology
AD Building
University of Surrey
Guildford
GU2 7XH
h.leonard@surrey.ac.uk
01483 686888

Thank you for taking the time to read this Information Sheet.
Title of Project

Executive function and internalizing problems in adolescents with and without Developmental Coordination Disorder

Introduction

We would like to invite you and your child to take part in a research project. Before you and your child decide to take part, you need to understand why the research is being done and what it will involve. Please take the time to read the following information carefully. Talk to others about the study if you wish.

This study is being conducted by Serif Omer (Trainee Clinical Psychologist), under the supervision of Dr Hayley Leonard (Lecturer in Developmental Psychology and Neurodevelopmental Disorders). Please ask questions if there is anything you do not understand.

What is the purpose of the study?

We know that adolescents with Developmental Coordination Disorder (DCD; sometimes also called ‘Dyspraxia’) often have difficulties with high-level thinking abilities, such as planning and memory. They also often report increased symptoms of anxiety and depression compared to others their age without the disorder. The current project aims to investigate this further by understanding how these difficulties may be related to each other. To understand this, we need to also recruit adolescents without dyspraxia, which is why we are inviting your child to participate in the study.

Why have I been invited to take part in the study?

As part of this study, we are seeking to recruit adolescents without a diagnosis of DCD / dyspraxia between the age of 12 and 16 and one of their parents/guardians. You and your child have been invited to take part because you might meet these criteria.

Do I have to take part?

No, neither you nor your child has to participate. There will be no adverse consequences if you decide not to participate. You can also withdraw your participation at any time without giving a reason.
What will my involvement require?

As the child’s parent or guardian, you will be asked to complete a consent form and some initial information about your child (e.g. date of birth). You will then be asked a set of questions which can be completed on paper or, if preferred, online or over the phone. This should take no more than 30 minutes in total and will include questions about your child’s movement abilities (such as sports skills, handwriting, etc.) and their high-level thinking skills (such as planning and memory).

You will not receive any payment for taking part. You will be given the option at the end of the survey to enter a prize draw for a £100 Amazon voucher. The winner will be contacted by email at the end of study in April 2018.

What will my child’s involvement require?

If you consent for your child to take part and they are suitable for the study, they will be asked to complete a questionnaire concerning their moods and emotions. In total this should take no more than 10 minutes. They can complete this questionnaire online, over the phone, or on paper.

Your child will not receive any payment for taking part.

What will I have to do?

If you and your child would like to take part, please complete the enclosed consent form and questionnaires.

Please feel free to contact the research team if you have any questions.

What are the possible disadvantages or risks of taking part?

There is a possibility that some of the questions may highlight sensitive issues for you or your child. If you want to talk about the questions or your answers, please contact the research team. If your child appears to experience any distress, the session will be terminated immediately and the research team will alert you and/or their teachers.

What are the possible benefits of taking part?

It is unlikely that you or your child will benefit directly from taking part. However, it is hoped that the study will improve our understanding of people with DCD/dyspraxia, which could have clinical implications for improving the lives of this group of people. Understanding the moods and emotions of adolescents without DCD/dyspraxia, and how they affect skills that are central to learning such as planning and memory, will also be of great use to teachers and researchers.
**What happens when the research study stops?**

You may receive a summary of the research findings at the end of the study (after February 2018). You can indicate if and how you would like to receive this on the initial screening questionnaire. This summary will only include the overall findings of the study. We will not provide individual feedback on your child’s or your own responses.

**What if there is a problem?**

Any complaint or concern about any aspect of the way you have been dealt with during the course of the study will be addressed; please contact Dr Hayley Leonard (Research Supervisor) on 01483 686888 or h.leonard@surrey.ac.uk. You may also contact the Head of School, Professor Derek Moore, on 01483 686933 or d.g.moore@surrey.ac.uk.

**Will taking part in the study be kept confidential?**

Yes. All of the information that you and your child give will be anonymised so that those reading reports from the research will not know who has contributed to it.

All project data will be held for at least 6 years and all research data for at least 10 years in accordance with University policy. Data will be stored securely in accordance with the Data Protection Act 1998.

However, should you or your child disclose information that suggests your child is at risk then the researcher may need to report this to an appropriate authority. This would usually be discussed with you first.

**Contact details**

Serif Omer  
Trainee Clinical Psychologist  
s.omer@surrey.ac.uk

Dr Hayley Leonard  
Lecturer  
h.leonard@surrey.ac.uk

School of Psychology  
AD Building  
University of Surrey  
Guildford  
GU2 7XH

01483 686888
Who is organising and funding the research?

This research is unfunded and is being completed by Serif Omer as part of a Doctorate in Clinical Psychology at the University of Surrey.

Who has reviewed the project?

The study has been reviewed and received a Favourable Ethical Opinion (FEO) from the Faculty of Health and Medical Sciences Ethics Committee, at the University of Surrey.

Thank you for taking the time to read this Information Sheet.
Title of Project
Executive function and internalizing problems in adolescents with and without Developmental Coordination Disorder

Introduction
Hi, my name is Serif Omer and I would like to invite you to take part in a research study. At the University of Surrey, we are trying to learn more about the effect of movement skills on young people's mood and thinking. We are comparing young people with no movement difficulties, like you, with young people who do have movement difficulties.

Why is this study being done?
People with Developmental Coordination Disorder (DCD), which is sometimes called 'Dyspraxia', have difficulties with movement tasks (such as handwriting and sports). However, they may also have difficulties with their mood and some thinking skills (such as memory and planning ahead). We would like to know more about whether young people with DCD have differences in their mood and thinking skills compared to young people without DCD and why this might be. We hope that this research will help to better understand the effect of DCD on young people which could help to improve their lives. This research could also help to better understand difficulties with mood and thinking skills in young people, like you, that do not have DCD.

Why have I been invited to take part?
As part of this study, we are looking for young people between the age of 12 and 16 who do not have DCD/dyspraxia. You have been invited to take part because you might be suitable. Your parent/guardian has also agreed for you to take part.

Do I have to take part?
Remember, being in this study is up to you. Even if your parents give their permission for you to participate in this study, you still can decide for yourself if you want to take part. You don't have to be in this study if you don't want to! Even if you agree to take part in the study, you can still stop at any time without giving a reason. If you decide not to take part, this will not affect you negatively in any way.

What will happen if I take part?
If you agree to be in this study, I will ask you to sign a consent form. I will also ask you to complete a questionnaire which asks you about your mood and emotions. Unfortunately, you will not receive any money for taking part. Your parent/guardian will be given the option at the end of the survey to enter a prize draw for a £100
Amazon voucher. The winner will be contacted by email at the end of study in April 2018.

**Will information about me be available to anyone?**
The information that we collect from you is confidential and anonymous. Only members of our team will be able to see this information. Your school and your parents/guardians will not have access to the information we collect. In our findings we will discuss the results of many young people - we do not single out or name any one participant.

However, if your parent/guardian has a concern, we may share a report of your strengths and weaknesses with them which they can pass on to a professional if they so choose. The only other time that we will tell somebody else about something you have said is if we think you or someone else may be at risk of harm.

This research study has been reviewed and given a Favourable Ethical Opinion by Faculty of Health and Medical Sciences Ethics Committee, at the University of Surrey. This means that the study has been checked and it is ethical for it to be conducted.

**What if I have a question or there is a problem?**
You can ask any questions that you have about the study at any time. If you have a question you can contact me or other people in my team using the contact details below.

**Serif Omer**
Trainee Clinical Psychologist
s.omer@surrey.ac.uk

**Dr Hayley Leonard**
Lecturer
h.leonard@surrey.ac.uk

School of Psychology
AD Building
University of Surrey
Guildford
GU2 7XH

01483 686888

Thank you for taking the time to read this Information Sheet.
Appendix G: Parent consent form

A copy of the parent consent form is provided on the following page.
Parent Consent Form [version 1, date 26.09.2016]

Executive function and internalizing problems in adolescents with and without Developmental Coordination Disorder

Please initial each box

- I have read and understood the Information Sheet provided (version 2, date xx/xx/xx). I have been given a full explanation by the investigators of the nature, purpose, location and likely duration of the study, and of what my child and I will be expected to do. 

- I have been advised about the potential for some questions to be of a sensitive nature for my child and/or myself. I have been given the opportunity to ask questions on all aspects of the study and have understood the advice and information given as a result.

- I agree to comply with the requirements of the study as outlined to me to the best of my abilities. I shall inform the investigators immediately if I have any concerns.

- I agree for my child’s and my own anonymised data to be used for this study that will have received all relevant ethical approvals.

- I understand that all project data will be held for at least 6 years and all research data for at least 10 years in accordance with University policy and that my personal data is held and processed in the strictest confidence, and in accordance with the UK Data Protection Act (1998).

- I understand that my child and I are free to withdraw from the study at any time without needing to justify our decision, without prejudice and without our legal rights and school education being affected.

- I understand that I can request for my data and my child’s data to be withdrawn until publication of the data and that following my request all personal data already collected from us can be destroyed.

- Optional. I agree for the researchers to contact me to provide me with a study results summary.

- Optional. I agree for the researchers to contact me about future studies.
• I confirm that I have read and understood the above and freely consent to participating in this study. I have been given adequate time to consider my participation.  

Name of child:  
(BLOCK CAPITALS)  

Name of parent/guardian  
(BLOCK CAPITALS)  

Signed:  

Date:  

Name of researcher taking consent:  
(BLOCK CAPITALS)  

Signed:  

Date:  
Appendix H: Adolescent assent form

A copy of the adolescent assent form is provided on the following page.
Adolescent Assent Form [version 1, date 08.08.2016]

Executive function and internalizing problems in adolescents with and without Developmental Coordination Disorder

Please tick yes or no

- Have you read (or been read to) about this research study? Yes ☐ No ☐
- Has somebody explained this research study to you? Yes ☐ No ☐
- Do you understand what the research study is about? Yes ☐ No ☐
- Have you asked all the questions you want? Yes ☐ No ☐
- Have you had your questions answered in a way you understand? Yes ☐ No ☐
- Do you understand it’s okay to say you don’t want to take part? Yes ☐ No ☐
- Do you understand it’s okay to stop taking part at any time? Yes ☐ No ☐
- Are you happy to take part? Yes ☐ No ☐

If any answers are ‘no’ or you don’t want to take part, don’t sign your name on this form.

If you do want to take part, please write your name and today’s date.

Your name: ............................................................
Date: ............................................................

Name of researcher taking consent:
............................................................

Signed: ............................................................
Date: ............................................................
Appendix I: Eligibility screening questionnaire

Please answer the following questions about your child:

1. Date of birth: .................................................................

2. Gender: Male Female Other

3. a. Have they received a diagnosis of Developmental Coordination Disorder (sometimes also known as Dyspraxia); or have they been referred for an assessment of Developmental Coordination Disorder?
   Yes, diagnosed Awaiting assessment No

   b. If yes, are you able to provide a diagnostic report to the research team?
      Yes No

4. Have they received a diagnosis of an Attention Deficit Hyperactivity Disorder (ADHD); or have they been referred for an assessment of Attention Deficit Hyperactivity Disorder?
   Yes, diagnosed Awaiting assessment No

5. Have they received a diagnosis of an Autism Spectrum Disorder (ASD) (sometimes also known as Asperger’s syndrome); or have they been referred for an assessment Autism Spectrum Disorder?
   Yes, diagnosed Awaiting assessment No

6. Would you like to be contacted after the study to receive a summary of the overall findings?
   Yes, by email Yes, by post No
Appendix J: Favourable ethical opinion

Chair’s Action
Proposal Ref: 1214-PSY-16
Names of Student/Trainee: SERIF OMER
Title of Project: Executive function and internalizing problems in adolescents with and without Development Coordination Disorder
Supervisors: Dr Hayley Leonard

Date of submission: 16th August, 2016
Date of resubmission: 27th September 2016

The above Research Project has been re-submitted to the Faculty of Health and Medical Sciences Ethics Committee and has received a favourable ethical opinion on the basis described in the protocol and supporting documentation.

The final list of documents reviewed by the Committee is as follows:

Ethics Application Form
Detailed protocol for the project
Participant Information sheet
Consent Form
Risk Assessment
Insurance Documentation (If appropriate)

All documentation from this project should be retained by the student/trainee in case they are notified and asked to submit their dissertation for an audit.

Signed and Dated: 27/09/2016
Dr Anne Arber, Professor Bertram Opitz
Co-Chairs, Ethics Committee

Please note:
If there are any significant changes to your proposal which require further scrutiny, please contact the Faculty of Health and Medical Sciences Ethics Committee before proceeding with your Project.
Appendix K: Assumptions for independent samples \(t\)-tests

Data plots

Histograms, Q-Q plots, and boxplots were created for each of the study groups to explore the data for outliers and normality.

(i) Internalising symptoms (RCADS Total):

\(TD\) group
\[\text{Histogram for TD group}\]

\(DCD\) group
\[\text{Histogram for DCD group}\]

\[\text{Normal Q-Q Plot for TD group}\]
\[\text{Normal Q-Q Plot for DCD group}\]
(ii) Executive function difficulties (BRIEF GEC):

**TD group**

**DCD group**
Outliers

The data was explored for univariate outliers prior to further analyses. No outliers were identified on inspection of histograms or boxplots. Z-scores were also calculated for each variable, where 5% of the total sample ($n = 2.15$) were expected to exceed $\pm 1.98$, 1% ($n = 0.43$) were expected to exceed $\pm 2.58$, and 0% expected to exceed $\pm 3.29$ (Field, 2013). On the RCADS Total, only one data point exceeded a Z-score of $\pm 1.98$ (fewer than the 5% cut-off) and no data points exceeded $\pm 2.58$. On the BRIEF GEC, two data points exceeded $\pm 1.98$ (at the expected level) and no data points exceeded $\pm 2.58$. This suggests that no univariate outliers were present.

Normality

The data were explored for normality. Inspection of histograms and Q-Q plots indicated that the data was skewed for both the RCADS Total and BRIEF GEC in both
study groups. Skewness and kurtosis scores were calculated for each variable and divided by their standard error to obtain z-scores (see Table K1). All scores were within the cut-off for significance (1.96) suggesting the distributions did not differ significantly from a normal distribution. Both the Kolmogorov-Smirnov test and Shapiro-Wilk test were conducted to explore whether the sample differed significantly from a normal distribution. These tests identified that both the RCADS Total and BRIEF GEC differed significantly from a normal distribution in the TD group, but not in the DCD group. Considering all this information, the data for both the RCADS Total and BRIEF GEC were not considered to meet the assumption of normal distribution. As such, bootstrapping was used in the analyses which is considered robust to violations of this assumption (Field, 2013).

Table K1. Normality statistics for study variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>Skewness z-score</th>
<th>Kurtosis z-score</th>
<th>Kolmogorov-Smirnov test (p)</th>
<th>Shapiro-Wilk test (p)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>TD</td>
<td>DCD</td>
<td>TD</td>
<td>DCD</td>
</tr>
<tr>
<td>RCADS-25</td>
<td>1.53</td>
<td>0.040</td>
<td>-0.97</td>
<td>-1.142</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BRIEF GEC</td>
<td>1.8</td>
<td>-1.382</td>
<td>-0.37</td>
<td>-0.049</td>
</tr>
</tbody>
</table>

*Significantly different from a normal distribution (p < .05)

BRIEF GEC: Behaviour Rating Inventory of Executive Function – Global Executive Index; DCD: Developmental Coordination Disorder; RCADS-25: Revised Child Anxiety and Depression Scale – Short version; TD: Typically developing
Homogeneity of variance / homoscedasticity

The variance of the outcome variable was explored for homogeneity. The variance ratios between the two groups indicated that the variances were approximately equal (RCADS Total: 1.38; BRIEF GEC: 1.76). In support of this, Levene’s test was not significant for both the RCADS Total, $F(1, 41) = 1.033, p = .315$, and BRIEF GEC, $F(1, 41) = 1.720, p = .197$. This suggests that the variance was stable at each level of the independent variable (DCD Status).
Appendix L: Assumptions for simple mediation analysis

The main assumptions for linear regression were explored for each path in the mediation analysis (i.e. path $a$, $b$ and $c$).

**Multivariate outliers / influential cases**

For each regression, the standardised residuals (z-scores) were calculated. It was expected that 2.15 cases (5% of total sample) would have standardised residuals exceeding +/-1.96 standard deviations from the mean, 0.43 cases (1%) would exceed +/-2.58, and zero cases (0%) would exceed +/-3.29 (Field, 2013). Each regression was also explored for influential cases. Cook’s distance was calculated, whereby scores greater than 1 indicate the case is having an undue influence on the model (Cook, 1977). Leverage values were calculated, whereby values greater than three times the average leverage indicate undue influence (Stevens, 2002). Finally, Mahalanobis distances were calculated, whereby values exceeding 11 were deemed as having undue influence (the cut-off suggested for very small sample sizes with two predictors; Field, 2013).

**Path a:** One case (2.3%) exceeded +/-1.96 standard deviations from the mean and zero cases exceeded +/-2.58 standard deviations from the mean. This is within expected levels, given the small sample size. Further examination indicated that no cases had a Cook’s distance higher than 1 (highest value = 0.13). Zero cases had leverage values larger than three times the average Leverage (Average Leverage = 0.05; highest value = 0.05). No values had a Mahalanobis value greater than 11 (highest value = 2.02). Given the above, no cases were deemed to have had any undue influence on the model.
Path b: One case (2.3%) exceeded +/- 1.96 standard deviations from the mean and zero cases exceeded +/- 2.58 standard deviations from the mean. This is within expected levels, given the small sample size. Further examination indicated that no cases had a Cook’s distance higher than 1 (highest value = 0.14). Zero cases had leverage values larger than three times the average Leverage (Average Leverage = 0.07; highest value = 0.18). No values had a Mahalanobis value greater than 11 (highest value = 7.43). Given the above, no cases were deemed to have had any undue influence on the model.

Path c: Zero cases (0%) exceeded +/- 1.96 standard deviations from the mean. Further examination indicated that no cases had a Cook’s distance higher than 1 (highest value = 0.13). Zero cases had leverage values larger than three times the average Leverage (Average Leverage = 0.05; highest value = 0.05). No values had a Mahalanobis value greater than 11 (highest value = 2.02). Given the above, no cases were deemed to have had any undue influence on the model.

Independence of errors

Durbin-Watson statistics were calculated for each regression model to explore the assumption of independent errors (i.e. the residuals within the model are uncorrelated). This assumption was met if values were close to 2 (Durbin & Watson, 1950, 1951).

Path a: The data met the assumption of independent errors as the Durbin-Watson statistic lay very close to 2 (Durbin-Watson = 1.904).

Path b: The data met the assumption of independent errors as the Durbin-Watson statistic lay very close to 2 (Durbin-Watson = 1.971).
Path c: The data met the assumption of independent errors as the Durbin-Watson statistic lay very close to 2 (Durbin-Watson = 1.771).

**Linearity and homoscedasticity**

A graph depicting the standardised residuals against the standardised predicted values was plotted for the model including both the independent variable (IV) and mediator (M) as predictors (i.e. path b). Data points were generally dispersed randomly and evenly throughout the plot indicate that linearity and homoscedasticity can be assumed. Homogeneity of variance was previously explored and assumed for paths a and c prior to conducting t tests (see Appendix K).
Normally distributed errors

Histograms and P-P plots of the standardised residuals were created for each regression model to explore the distribution of errors (see below). They show that the distribution of errors was approximately normally distributed, at least sufficiently for bootstrapping analyses.

Path a:

Path b:
**Path c:**

![Histogram](image1)

![Graph](image2)

**Homogeneity of regression**

The data were tested to check that the relationship between the mediator and dependent variable (DV) were not dependent on the IV. When including the interaction between DCD status and BRIEF GEC in the model, the interaction was not significant ($p = .192$).

**Multicollinearity**

The data was assessed for multicollinearity prior to conducting the mediation analysis. Pearson’s correlation coefficients between the key predictor variables indicated no correlations were higher than .90. Correlations of .90 or more are suggestive of multicollinearity (Field, 2013). Further tests also indicated that the VIF level was below 10 (Bowerman & O’Connell, 1990; Myers, 1990) and tolerance was above 0.2 (Menard, 1995) when entering both predictors (DCD status and BRIEF GEC) into the model (VIF = 4.182; tolerance = 0.239).
Appendix M: Assumptions for subscale analyses using independent samples $t$-tests

Data plots

Histograms, Q-Q plots, and boxplots were created for each subscale of the RCADS-25 and BRIEF across each study group to explore the data for outliers and normality.

(i) Depression symptoms (RCADS-25 Depression):

*TD group*

*DCD group*
Anxiety symptoms (RCADS-25 Anxiety):

TD group

DCD group
(ii) Behavioural regulation difficulties (BRIEF BRI):

**TD group**

**DCD group**
(iii) Metacognitive difficulties (BRIEF MCI):

**TD group**

**DCD group**
Outliers

The data was explored for univariate outliers prior to further analyses. No significant outliers were identified on inspection of histograms or boxplots. Z-scores were also calculated for each variable, where 5% of the total sample (n = 2.15) were expected to exceed +/-1.98, 1% (n = 0.43) were expected to exceed +/-2.58, and 0% expected to exceed +/-3.29 (Field, 2013). On the RCADS-25 Depression subscale, only one data point exceeded a Z-score of +/-1.98 (fewer than the 5% cut-off) and no data points exceeded +/-2.58. On the RCADS-25 Anxiety subscale, two data points exceeded +/-1.98 (at the expected level) and no data points exceeded +/-2.58. Zero cases exceeded a z-score of +/-1.96 on both the BRIEF BRI and BRIEF MCI. This suggests that no univariate outliers were present.

Normality

The data were explored for normality. Inspection of histograms and Q-Q plots indicated that the data was skewed for all subscales in both study groups. Skewness and
Kurtosis scores were calculated for each variable and divided by their standard error to obtain z-scores (see Table M1). The RCADS-25 Anxiety subscale in the TD group exceeded the cut-off for significance (+/-1.96), indicating that it was significantly negatively skewed. Both the Kolmogorov-Smirnov test and Shapiro-Wilk test were conducted to explore whether the samples differ significantly from a normal distribution. A combination of these tests indicated that all subscales differed significantly from a normal distribution in the TD group, but not in the DCD group. Considering all this information, the data for the subscales were not considered to meet the assumption of normal distribution. As such, bootstrapping was used in the analyses which is considered robust to violations of this assumption (Field, 2013).

Table M1. Normality statistics for subscale variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>Skewness z-score</th>
<th>Kurtosis z-score</th>
<th>Kolmogorov-Smirnov test (p)</th>
<th>Shapiro-Wilk test (p)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>TD</td>
<td>DCD</td>
<td>TD</td>
<td>DCD</td>
</tr>
<tr>
<td>RCADS-25 Depression</td>
<td>1.94</td>
<td>-1.05</td>
<td>-0.22</td>
<td>-0.48</td>
</tr>
<tr>
<td>RCADS-25 Anxiety</td>
<td>2.47*</td>
<td>0.94</td>
<td>0.63</td>
<td>-0.66</td>
</tr>
<tr>
<td>BRIEF BRI</td>
<td>0.74</td>
<td>-0.76</td>
<td>-1.40</td>
<td>-0.82</td>
</tr>
<tr>
<td>BRIEF MCI</td>
<td>1.83</td>
<td>-1.21</td>
<td>-0.33</td>
<td>0.67</td>
</tr>
</tbody>
</table>

*Significantly different from a normal distribution (p < .05)

BRI: Behavioural Regulation Index; BRIEF: Behaviour Rating Inventory of Executive Function; DCD: Developmental Coordination Disorder; MCI: Metacognitive Index; RCADS-25: Revised Child Anxiety and Depression Scale – Short version; TD: Typically developing
**Homogeneity of variance / homoscedasticity**

The variance of the outcome variable was explored for homogeneity. The variance ratios between the two groups indicated that the variances were approximately equal (RCADS-25 Depression: 1.07; RCADS-25 Anxiety: 1.40; BRIEF BRI: 1.11; BRIEF MCI: 1.60). In support of this, Levene’s test was not significant for all outcomes, largest: $F(1, 41) = 1.38$, $p = .247$. This suggests that the variance was stable at each level of the independent variable (DCD Status).
Appendix N: Assumptions for parallel multiple mediation analysis based on subscales

The assumptions for linear regression were explored for each path in the parallel multiple mediation analyses (i.e. paths $a_1, a_2, b_1, b_2, c$). This included separate analyses with the RCADS-25 Depression and RCADS-25 Anxiety scores as the DV.

Multivariate outliers / influential cases

For each regression, the standardised residuals (z-scored) were calculated. The models ranged from having 0 to 1 cases exceeding +/-1.96 standard deviations from the mean. Among all models, no cases had a Cook’s distance higher than 1 (highest values: 0.13 to 15). No cases had Leverage value greater than three times the average Leverage (highest values: 0.05 to 0.17). No cases had a Mahalanobis value greater than 11 (highest values: 2.02 to 7.02). Given the above, no cases were deemed to have had any undue influence on any of the models.

Independence of errors

Durbin-Watson statistics were close to 2 for all models (range: 1.693 to 2.147).

Linearity and homoscedasticity

A graph depicting the standardised residuals against the standardised predicted values was plotted for the models including all predictors. Data points were generally dispersed randomly and evenly throughout the plots indicating that linearity and
homoscedasticity can be assumed for both models. Homogeneity of variance was previously explored and assumed for paths a and c prior to conducting $t$-tests (see Appendix M).

**RCADS-25 Depression**

**RCADS-25 Anxiety**

![Histograms and P-P plots of the standardised residuals were created for each regression model. They show that the distribution of errors was approximately normally distributed. Plots of the models including all predictors are displayed below.](image)

**Normally distributed errors**

Histograms and P-P plots of the standardised residuals were created for each regression model. They show that the distribution of errors was approximately normally distributed. Plots of the models including all predictors are displayed below.

**RCADS-25 Depression**

**RCADS-25 Anxiety**
Homogeneity of regression

The data were tested to check that the relationship between the mediators and DV’s were not dependent on the IV. When including the interaction between IV and M’s, the interaction was not significant (range of $p = .068$ to .248).

Multicollinearity

The VIF level was below 10 (Bowerman & O’Connel, 1990; Myers, 1990) and tolerance was above 0.2 (Menard, 1995) for all variables (range, VIF= 3.77-4.27; tolerance = 0.234-2.65).
Appendix O: Assumptions for simple mediation analysis within the TD sample

The main assumptions for linear regression were explored for each path in the mediation analysis (i.e. path $a$, $b$ and $c$) with the DCDQ-07 as the IV (TD group only).

Multivariate outliers / influential cases

For each regression, the standardised residuals (z-scores) were calculated. It was expected that 1.60 cases (5% of total sample) would have standardised residuals exceeding +/-1.96 standard deviations from the mean, 0.32 cases (1%) would exceed +/- 2.58, and zero cases (0%) would exceed +/- 3.29 (Field, 2013). Cook’s distance, leverage values, and Mahalanobis distances were also calculated.

Path $a$: Two cases (6.25%) exceeded +/-1.96 standard deviations from the mean and zero cases exceeded +/- 2.58 standard deviations from the mean. No cases had a Cook’s distance higher than 1 (highest value = 0.12). Zero cases had leverage values larger than three times the average Leverage (Average Leverage = 0.03; highest value = 0.09). No values had a Mahalanobis value greater than 11 (highest value = 6.07). Given the above, no cases were deemed to have had any undue influence on the model.

Path $b$: One case (3.13%) exceeded +/- 2.58 standard deviations. No cases had a Cook’s distance higher than 1 (highest value = 0.21). One case had a leverage value larger than three times the average Leverage (Average Leverage = 0.06; highest value = 0.22). No values had a Mahalanobis value greater than 11 (highest value = 6.71). Given the above, no cases were deemed to have had any undue influence on the model.
**Path c:** One case (3.13%) exceeded +/-1.96 standard deviations from the mean and no cases exceeded +/-2.58 standard deviations. Further examination indicated that no cases had a Cook’s distance higher than 1 (highest value = 0.14). Two cases had leverage values larger than three times the average Leverage (Average Leverage = 0.03; highest value = 0.19). No values had a Mahalanobis value greater than 11 (highest value = 6.07). Given the above, no cases were deemed to have had any undue influence on the model.

**Independence of errors**

The data met the assumption of independent errors as the Durbin-Watson statistic lay very close to 2 (Durbin-Watson range = 1.792-2.14).

**Linearity and homoscedasticity**

A graph depicting the standardised residuals against the standardised predicted values was plotted for each path. Data points were dispersed randomly and approximately evenly throughout the plot indicating that linearity and homoscedasticity can be assumed.
Normally distributed errors

Histograms and P-P plots of the standardised residuals were created for each regression model to explore the distribution of errors (see below). They show that the distribution of errors was approximately normally distributed, at least sufficiently for bootstrapping analyses.

Path a:

Path b:
Path $c$:

Multicollinearity

The data was assessed for multicollinearity prior to conducting the mediation analysis. Pearson’s correlation coefficients between the key predictor variables indicated no correlations were higher than .65. Further tests also indicated that the VIF level was below 10 (Bowerman & O’Connell, 1990; Myers, 1990) and tolerance was above 0.2 (Menard, 1995) when entering both predictors (DCDQ-07 and BRIEF GEC) into the model (VIF = 1.00; tolerance = 1.00).
Part 2: Major Research Project Literature review

Internalising symptoms in Developmental Coordination Disorder: A systematic review and meta-analysis

Word count: 6175

Statement of Journal Choice

The Journal of Child Psychology and Psychiatry

‘The Journal of Child Psychology and Psychiatry’ publishes research that advances the understanding of developmental psychopathology and that informs theory and clinical practice. It is a leading journal for child and adolescent psychology and psychiatry. The journal has confirmed interest in this paper for submission to their ‘Research Reviews’ section. See Appendix for more details.
Abstract

**Background:** Developmental Coordination Disorder (DCD) affects 5-6% of children. There is growing evidence that DCD is associated with greater levels of internalising symptoms (i.e. depression and anxiety). This is the first systematic review and meta-analysis to explore the magnitude of this effect, the quality of the evidence, and potential moderators. **Methods:** A systematic search was conducted to identify studies reporting a comparison between individuals with DCD/probable DCD and typically developing (TD) individuals on measures of internalising symptoms. A pooled effect size (Hedges $g$) was calculated using random effects meta-analysis. Study quality, publication bias and potential moderators of the effect were explored. **Results:** Twenty studies, including a total of 23 subsamples, met the inclusion criteria, of which 22 subsamples were included in the meta-analysis (DCD: $n = 1123$; TD: $n = 7346$). A significant, moderate effect of DCD on internalising symptoms was found ($g = 0.61$). This effect remained robust after accounting for publication bias and excluding lower quality studies. The effect was significantly larger in studies utilizing a cross-sectional design (vs. longitudinal), convenience sampling (vs. population screening) and a majority male sample. **Conclusions:** The findings demonstrate that individuals with DCD experience greater levels of internalising symptoms than their peers. This highlights the importance of routine screening for emotional difficulties in DCD, raising awareness of the condition in mental health services, and developing psychosocial interventions that extend beyond a focus on motor impairments. However, there is a need for higher quality, longitudinal studies to better understand the causal relationship between DCD and internalising symptoms.
Keywords: Developmental Coordination Disorder, DCD, Internalising symptoms, depression, anxiety, mental health, meta-analysis.
Introduction

Developmental Coordination Disorder (DCD) is a neurodevelopmental disorder affecting between 5-6% of children and is characterised by significant impairment to an individual’s ability to perform everyday motor tasks (American Psychiatric Association [APA], 2013). This can include difficulties with self-care (e.g. tying shoelaces, brushing teeth), academic tasks (e.g. handwriting), and leisure activities (e.g. catching a ball, balancing). A diagnosis of DCD is based on four criteria (APA, 2013): (a) performance in motor coordination tasks is substantially below expectation given the person’s age and opportunities; (b) the motor coordination difficulties significantly interfere with activities of daily living or academic achievement; (c) difficulties began in the early developmental period; and (d) the difficulties cannot be attributed to an intellectual disability or neurological condition (e.g. cerebral palsy). DCD is sometimes referred to as ‘dyspraxia’. However, there is no internationally agreed definition for the term dyspraxia, it is not included in diagnostic manuals and it is sometimes used to refer to a broader range of difficulties beyond those characterising DCD. As such, the term DCD is used hereafter.

Despite its prevalence, DCD often goes unrecognised and is poorly understood among healthcare and education professionals (Gaines, Missiuna, Egan, & McLean, 2008; B. N. Wilson, Neil, Kamps, & Babcock, 2013). This is of concern given that DCD has been found to have a significant impact not just on an individual’s motor abilities, but across a wide range of psychological, cognitive, physical and social domains (Zwicker, Harris, & Klassen, 2013). For example, children with DCD have been found to engage in less physical activity (Cairney, Hay, Veldhuizen, Missiuna, & Faught,
to be at an increased risk of obesity (Cairney & Veldhuizen, 2013), to underachieve in academic performance (Gomez et al., 2015), to be more socially isolated (Chen & Cohn, 2003) and to have lower self-esteem (Miyahara & Piek, 2006) compared to their typically developing (TD) peers. There is also evidence that the impact of DCD across these domains persists through childhood and into adulthood (Cousins & Smyth, 2003; Hill, Brown, & Sorgardt, 2011; Kirby, Sugden, Beveridge, & Edwards, 2008). DCD also often co-occurs with other developmental disorders, including Attention Deficit Hyperactivity Disorder (ADHD) and Autism Spectrum Disorder (ASD; Pieters, et al. 2012).

One area that has received increasing attention is the impact of DCD on mental health, specifically internalising symptoms. ‘Internalising’ is a broad construct referring to symptoms of both depression and anxiety, which have been found to commonly overlap in childhood and adolescence (Eaton et al., 2013; Kovacs & Devlin, 1998). There is growing evidence that individuals with DCD have elevated levels of internalising symptoms compared to their TD peers (Mancini, Rigoli, Cairney, Roberts, & Piek, 2016). Research has also found associations between motor abilities and internalising symptoms in community samples of TD children and adults (Poole et al., 2016; Rigoli, Piek, & Kane, 2012; A. Wilson, Piek, & Kane, 2013), an increased risk of psychiatric disorders in individuals with DCD (Rasmussen & Gillberg, 2000), and impaired motor abilities in individuals with common psychiatric disorders (Damme, Simons, Sabbe, & van West, 2015). The Environmental Stress Hypothesis is a framework that was introduced to account for this relationship between DCD and mental health (Cairney, Rigoli, & Piek, 2013). It suggests that the motor impairments in DCD
can expose an individual to a variety of secondary psychosocial stressors which over time can lead to poorer mental health (see Figure 1). These proposed mediators include physical (e.g. physical inactivity), social (e.g. interpersonal conflict, reduced social resources), and personal factors (e.g. reduced self-esteem).

Figure 1. Diagram depicting the Environmental Stress Hypothesis as an explanation for internalising symptoms in DCD (from Mancini et al. 2016).

Understanding the link between DCD and mental health has important implications for assessment and intervention with this population, including at schools, physical health services and mental health services. To date, several reviews have summarised the findings on internalising symptoms in DCD (Caçola, 2016; Mancini et al., 2016; Missiuna & Campbell, 2014). However, they consist of narrative summaries only. There are also inconsistent findings, with some studies finding no significant effect
(Davis, Ford, Anderson, & Doyle, 2007; King-Dowling, Missiuna, Rodriguez, Greenway, & Cairney, 2015) and others a large effect (Dewey, Kaplan, Crawford, & Wilson, 2002; Pratt & Hill, 2011). A systematic search, synthesis and critical appraisal of the evidence can provide a more rigorous understanding of this relationship. A meta-analysis to pool the findings would provide a more accurate understanding of whether individuals with DCD do indeed experience greater internalising symptoms than their peers and would provide insight into the magnitude of this difference.

Studies also vary greatly in their design, participants, measures, and methodological quality. Whereas some studies have recruited participants with a confirmed diagnosis of DCD, others have included only those identified as ‘probable DCD’ based on screening measures. Studies also differ in how well they controlled for confounding variables, whether they employed a longitudinal or cross-sectional design, and whether they recruited participants through population-based screening or convenience sampling. There is also some evidence to suggest the impact may be greater in adolescents compared to younger children (Piek et al., 2007; Skinner & Piek, 2001), in males compared to females (Sigurdsson et al., 2002), and in individuals with comorbid ADHD (Missiuna et al., 2014; Piek et al., 2007). A meta-analytic approach allows for an investigation into the potential sources of heterogeneity across studies, which could help to guide future research and intervention.

The aim of this paper was, therefore, to conduct a systematic review and meta-analysis of studies that compared individuals with DCD to TD individuals on measures of internalising symptoms; to appraise the quality of the evidence; and to explore which factors moderate the effect. The focus was on severity levels of internalising symptoms,
as opposed to rates of actual diagnosis, given that most studies have adopted severity outcome measures, and given that data on diagnostic rates may obscure important differences in actual symptoms.

**Method**

The review was protocol-driven and carried out in accordance with recommended guidelines for systematic reviews (Moher, Liberati, Tetzlaff, Altman, & The Prisma Group, 2009) and meta-analyses of observational studies (Stroup et al., 2000).

**Eligibility criteria**

In line with recommended guidelines, broad inclusion criteria were used with the aim to later explore the impact of specific design features. Articles were eligible if they:

(a) included participants, of any age, with a confirmed diagnosis of DCD according to DSM-IV or DSM-5 criteria; or who were identified as having motor coordination difficulties consistent with DCD (i.e. ‘probable DCD’); (b) included a comparison group of TD individuals, as defined by the absence of diagnosed or suspected developmental disorders; (c) measured levels of internalising symptoms (i.e. depression and/or anxiety) for each group using self-report, parent-report, teacher-report, direct observation or clinical interview; (d) reported statistics that could be transformed into a standardised mean difference (e.g. mean and standard deviations); and (e) were available in full text in English. Studies involving participants with a comorbid diagnosis (e.g. ADHD) were also eligible if the motor coordination difficulties were clearly described and used as the
basis for group comparison (as the first criterion above). Studies were excluded if participants’ motor difficulties were attributed to another developmental difficulty or medical diagnosis (e.g. cerebral palsy). For studies using the label ‘dyspraxia’, they were required to refer to overall motor impairment and not just oro-motor difficulties and gesture. Studies were also excluded if the outcome only included rates of psychiatric diagnoses (i.e. they did not report on a measure of the severity of internalising symptoms).

If separate studies included overlapping samples, priority was given to the study with the best control of important confounders (i.e. age and gender) or the study that allowed for the most detailed exploration of moderating factors (e.g. outcomes reported separately by gender or age group). Where the same participants were included but different subtests reported, data were combined (Borenstein, Hedges, Higgins, & Rothstein, 2009).

**Search strategy**

Studies were identified through a systematic search of Medline, PsychInfo, CINAHL, ERIC, and Web of Science. To minimise the impact of publication bias, unpublished studies were also searched using ProQuest Dissertations and Theses and Open Grey. The latest search was completed on 3rd March 2018.

The search included terms related to DCD (*developmental coordination disorder*, *DCD*, *dyspraxi*, *motor skills disorder*, *coordination difficult*, *coordination problem*, *clumsy*, *clumsiness*, *motor proficiency*, *motor competence*, *motor difficult*, *motor impairment*, *motor dysfunction*, *perceptual motor difficult*, *perceptual motor*...
impairment, motor skills disorder, motor learning difficult*, motor learning problem*, motor problem*, movement disorder, psychomotor disorder) combined with terms related to internalising symptoms (internali*, anxi*, depress*, mood, mental health, mental illness, mental disorder, emotional problem*, psychopatholog*). Titles and abstracts were screened by one reviewer (SO), with 25% cross-checked by a second (AMN; percent agreement = 97.5%, Cohen’s kappa = 0.65). The full texts of all potentially relevant articles were then screened independently by two reviewers (SO & AMN), with disagreement resolved by consensus and discussion with a third (HL; percent agreement = 94.5%, kappa = 0.87). The bibliographies of the included studies and relevant review articles were screened and their citations tracked to identify additional studies. The first authors of the included articles were also contacted to identify any further eligible studies or to clarify missing information.

**Data extraction**

Data on each study were extracted into an electronic pro forma. Two researchers (SO & AMN) performed data extraction for all included studies and inconsistencies were discussed until consensus was reached. Interrater reliability was good for both categorical (percent agreement = 91-100%; kappa = 0.82-1.00) and continuous (intraclass correlation coefficient = 1.00) data.

The following information was extracted for each study: author, publication year, country, design (cross-sectional; longitudinal), population, sampling procedure (population-based screening; selective/convenience sample), criteria for DCD (confirmed DCD; probable DCD), criteria for TD, number of participants, gender
(percentage male), age (mean and range), comorbid ADHD diagnosis (ADHD assessed and excluded from the sample; ADHD assessed and included; ADHD not assessed), measure of internalising symptoms, internalising construct (depression; anxiety; overall internalising), reporter (self-report; parent-report; teacher-report; clinician/researcher-report), and scores (means and standard deviations, other relevant statistics).

If multiple informants or measures were used to assess internalising symptoms, they were extracted separately so that they could be pooled (see Statistical Analysis). Preference was given to data adjusted for important confounders (e.g. gender, age) if not matched by design. However, where studies also adjusted for additional variables (e.g. intelligence), the unadjusted scores were preferred to ensure comparability across studies (Voils, Crandell, Chang, Leeman, & Sandelowski, 2011). Where findings were reported separately for subgroups (e.g. gender, age groups) these data were extracted separately as subsamples to facilitate the moderation analysis. Where separate groups were included for confirmed and probable DCD, only the confirmed DCD group was extracted. Where separate groups were included for comorbid DCD/ADHD and DCD only, the DCD only group was extracted. This allowed for a better understanding of the specific effect of DCD on internalising problems.

**Study quality**

An adapted version of the Newcastle-Ottawa Scale (NOS) was used to assess study quality (Wells et al., 2011). Each study was rated on representativeness of the DCD group (i.e. population screening), selection of the control group (i.e. same population as DCD), ascertainment of DCD diagnosis (i.e. confirmed DCD); control for
baseline internalising (for longitudinal studies), comparability of groups (i.e. control for confounders), measurement of internalising symptoms (i.e. validated measures), length of follow-up (i.e. at least one year follow-up), and completeness of follow-up (i.e. ≥80% follow-up rate or <80% but unlikely to introduce bias).

For the DCD criteria to be rated as ‘confirmed DCD’, the study must have assessed motor skills as being below the 15th percentile using performance-based measures (Criterion A; Blank, Smits-Engelsman, Polatajko, & Wilson, 2012), as having a significant impact on activities of daily living or academic achievement (e.g. questionnaires or interview; Criterion B), and ruled out intellectual disability and other neurological conditions (e.g. by interview, performance measures, medical reports; Criterion D). Alternatively, they could have cross-checked medical records for diagnosis. Given that many studies were published prior to publication of the DSM-5 and the introduction of Criterion C, it was not essential that studies established whether difficulties began in the early developmental period. The confounders considered most important for comparability of study groups were age and gender (Twenge & Nolen-Hoeksema, 2002). The NOS satisfies relevant guidelines for quality assessment of non-randomised studies (Sanderson, Tatt, & Higgins, 2007) and is recommended for systematic reviews of observational studies (Deeks et al., 2003).

**Statistical Analysis**

The main analysis was performed using Review Manager 5 software.

*Summary effect:* The standardised mean difference (SMD; Hedges g) between the DCD group and the TD group and its 95% confidence interval were calculated for
each study (or subsample) separately. SMD’s around 0.2 can be considered small, 0.5 moderate, and 0.8 large. Where studies reported data on multiple measures of internalising, a pooled effect size and variance was calculated (assuming a correlation between measures of 1, which provides a conservative estimate of the variance; Borenstein, Hedges, Higgins, & Rothstein, 2009). Effect sizes were weighted according to the inverse of their variance to ensure that more precise estimates influence overall effect size most heavily and to reduce the effect of the upwardly biased estimates of smaller studies (Hedges & Olkin, 1985). Random-effects meta-analysis was used to calculate a summary effect for total internalising symptoms across all studies and its 95% confidence interval.

**Heterogeneity:** Q-statistics were used to assess for heterogeneity and the $I^2$ statistic to quantify the proportion of the variance due to heterogeneity. Moderators were explored to identify potential sources of heterogeneity. The following moderators were explored: year of publication (pre-2010 vs. post-2010), design (longitudinal vs. cross-sectional), gender (>50% male vs. ≤50% male in the DCD group), age (included adolescents ≥12 vs. no adolescents), comorbid ADHD (assessed and excluded vs. not assessed, or assessed but included), sampling strategy (population screen versus selective/convenience); selection of DCD group (confirmed vs. probable DCD), controlled for confounders (age and gender controlled vs. uncontrolled), reporter (self-report versus informant-report), and type of internalising (overall internalising vs. depression vs. anxiety). The significance of moderators was tested using Q-statistics.

**Publication bias:** Publication bias was assessed visually using a funnel plot, where an asymmetrical distribution indicates the presence of publication bias. Egger’s
test was used to statistically check for publication bias (Egger, Smith, Schneider, & Minder, 1997). Duval and Tweedie’s trim-and-fill procedure was used to compute an adjusted effect size by imputing the effect of smaller, unpublished studies (Duval & Tweedie, 2000). Finally, Rosenthal's (1979) Fail-safe $N$ was calculated to determine the number of studies with an average effect size of 0 that would have to be included to produce a non-significant result. This number should exceed $5k + 10$ (where $k$ is the number of studies).

*Sensitivity analysis:* A sensitivity analysis was conducted to calculate a pooled effect size excluding lower quality studies (i.e. those not meeting at least five criteria on the NOS).

**Results**

**Search results**

The search identified a total of 20 studies meeting the inclusion criteria, consisting of 23 eligible subsamples (two studies reported outcomes separately for males and females, one study reported separate outcomes for children and adolescents; hereafter these are treated as separate studies). The search process is summarised in Figure 2.

It should be noted that two articles reported outcomes at multiple time points for the same longitudinal study (Harrowell, Hollén, Lingam, & Emond, 2017; Lingam et al., 2012). The data from the latter time point was used (Harrowell et al., 2017) because separate outcomes were reported for males and females, allowing for better exploration of moderators. Two articles reported data on the same cross-sectional study (Pearsall-
Figure 2. PRISMA flow diagram summarising the search process.
Jones, Piek, Rigoli, Martin, & Levy, 2011; Piek et al., 2007), so the larger sample was included (Piek et al., 2007). One eligible study reported extreme values for the outcome, raising concerns around its accuracy, and was subsequently excluded (Tseng, Howe, Chuang, & Hsieh, 2007).

**Characteristics of the included studies**

The characteristics of the included studies are summarised in Table 1. A total of 8469 participants were included (1123 DCD, 7346 TD). The studies were published between 1994 and 2018. Most were from developed countries, with one study from Taiwan. Three prospective cohort studies were identified that screened for DCD in a cohort and assessed their internalising symptoms at a later follow-up (Harrowell et al., 2017, male & female samples; Wagner, Jekauc, Worth, & Woll, 2016). The remaining 20 studies adopted a cross-sectional design.

Sixteen studies identified individuals with and without DCD/probable DCD via population-based screening of community samples (Campbell, Missiuna, & Vaillancourt, 2012; Chen, Tseng, Hu, & Cermak, 2009; Dewey et al., 2002; Francis & Piek, 2003; Harrowell et al., 2017; King-Dowling et al., 2015; Li et al., 2018; Missiuna et al., 2014; Piek, Bradbury, Elsley, & Tate, 2008; Schoemaker & Kalverboer, 1994; Skinner & Piek, 2001; van den Heuvel, Jansen, Reijneveld, Flapper, & Smits-Engelsman, 2016; Wagner et al., 2016). Of the remaining studies, five recruited DCD participants through selective or convenience samples such as clinical referrals or
support groups (Crane, Sumner, & Hill, 2017; Hill & Brown, 2013; Pratt & Hill, 2011; Wagner, Bös, Jascenoka, Jekauc, & Petermann, 2012; Watson & Knott, 2006), one recruited participants through screening a clinical population of children born with extremely low birth weight (Davis et al., 2007), and one sampled from a monozygotic twin population (Piek et al., 2007).

The studies varied in their operationalisation of the DCD group. Ten studies confirmed a DCD diagnosis via independent assessment of diagnostic criteria (Chen et al., 2009; Harrowell et al., 2017; Missiuna et al., 2014; van den Heuvel et al., 2016; Wagner et al., 2012) or via clinical reports (Crane et al., 2017; Hill & Brown, 2013; Pratt & Hill, 2011; Watson & Knott, 2006). Thirteen studies identified those as probable DCD based on performance-based tests of motor function (Davis et al., 2007; Dewey et al., 2002; Francis & Piek, 2003; King-Dowling et al., 2015; Li et al., 2018; Piek et al., 2008; Schoemaker & Kalverboer, 1994; Skinner & Piek, 2001; Wagner et al., 2016) or by parent-report questionnaires (Campbell et al., 2012; Piek et al., 2007).

Most studies recruited children and adolescents, with only one study conducted with adults (Hill & Brown, 2013). Of the child and adolescent studies, ten included adolescents aged 12 or over (Dewey et al., 2002; Harrowell et al., 2017; Li et al., 2018; Missiuna et al., 2014; Piek et al., 2007; Pratt & Hill, 2011; Skinner & Piek, 2001, adolescent sample; Wagner et al., 2016; Watson & Knott, 2006). There was a mix of male and female participants across the studies, with fourteen having majority male participants (Campbell et al., 2012, male sample; Crane et al., 2017; Davis et al., 2007; Dewey et al., 2002; Francis & Piek, 2003; Harrowell et al., 2017, male sample; King-
Table 1. Summary of characteristics of the included studies

<table>
<thead>
<tr>
<th>Study</th>
<th>Year</th>
<th>Population</th>
<th>DCD assessment</th>
<th>DCD sample, n</th>
<th>TD sample, n</th>
<th>DCD age, mean (range)</th>
<th>TD age, mean (range)</th>
<th>DCD gender, % male</th>
<th>TD gender, % male</th>
<th>Excluded ADHD? (measure)</th>
<th>Confounders controlled</th>
<th>Internalising measures, (type)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Harrowell et al. (male sample) 2017</td>
<td>Population screening: Birth cohort in south-west England, motor skills assessed age 7.</td>
<td>Confirmed DCD</td>
<td>SDQ: 109; SMFQ: NR (total sample)</td>
<td>NR</td>
<td>SDQ: 1780; SMFQ: NR (total sample collected at)</td>
<td>17.5 years; 17.5 years;</td>
<td>100%</td>
<td>0%</td>
<td>No</td>
<td>Age, gender,*</td>
<td>SMFQ (self-rated depression); SDQ - emotional subscale (self-rated internalising)</td>
<td></td>
</tr>
<tr>
<td>Harrowell et al. (female sample) 2017</td>
<td>Population screening: Birth cohort in south-west England, motor skills assessed age 7.</td>
<td>Confirmed DCD</td>
<td>SDQ: 59; SMFQ: NR (total sample)</td>
<td>NR</td>
<td>SDQ: 1970; SMFQ: NR (total sample collected at)</td>
<td>17.5 years; 17.5 years;</td>
<td>0%</td>
<td>0%</td>
<td>No</td>
<td>Age, gender,*</td>
<td>SMFQ (self-rated depression); SDQ - emotional subscale (self-rated internalising)</td>
<td></td>
</tr>
<tr>
<td>Wagner et al. 2016</td>
<td>Population screening: Cohort of 6-10 year olds across Germany: Motor skills assessed age 6-10.</td>
<td>Probable DCD (performance-based test)</td>
<td>114</td>
<td>823</td>
<td>14.35 (12-16)</td>
<td>14.38 (12-16)</td>
<td>47.8%</td>
<td>49.3%</td>
<td>No</td>
<td>Gender, age, baseline internalising</td>
<td>SDQ (parent-rated internalising)</td>
<td></td>
</tr>
</tbody>
</table>
### Cross-sectional Studies

<table>
<thead>
<tr>
<th>Study</th>
<th>Sample Description</th>
<th>Age, Gender</th>
<th>Screening Method</th>
<th>Age Mean (Range)</th>
<th>Depression (Range)</th>
<th>Measure Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Campbell et al.</td>
<td>Female sample; Fifth grade students from previous longitudinal study in Ontario, Canada.</td>
<td>10.9 (10-11)</td>
<td>No (assessed)</td>
<td>100%</td>
<td>BASC-2 – depression subscale (self-rated depression)</td>
<td></td>
</tr>
<tr>
<td>Chen et al.</td>
<td>1st-3rd grade children in greater Taipei area, Taiwan.</td>
<td>7.93 (NR)</td>
<td>No (measured, not excluded)</td>
<td>59.0%</td>
<td>CBCL – Chinese version - withdrawn, somatic complaints, depressed/anxious subscales (parent-rated internalising)</td>
<td></td>
</tr>
<tr>
<td>Davis et al.</td>
<td>Screening of clinical sample: Cohort of children born with ELBW or very preterm in Victoria, Australia.</td>
<td>8.61 (7-10)</td>
<td>Yes (self-/parent-report)</td>
<td>70.0%</td>
<td>SDQ-Teacher - Emotional subscale (teacher-rated internalising)</td>
<td></td>
</tr>
<tr>
<td>Crane et al.</td>
<td>Convenience sample: children with DCD via primary schools and charity; TD via primary schools in South London, UK.</td>
<td>8.12 (7-10)</td>
<td>None</td>
<td>74.0%</td>
<td>BASC-parent: internalising subscale (parent-rated internalising); BASC-teacher rated (teacher-rated internalising)</td>
<td></td>
</tr>
<tr>
<td>Study</td>
<td>Population screening:</td>
<td>Probable DCD (performance-based tests, screening questionnaires)</td>
<td>N</td>
<td>75th percentile</td>
<td>Prevalence</td>
<td>Measured/Excluded</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>-----------------------</td>
<td>---------------------------------------------------------------</td>
<td>---</td>
<td>----------------</td>
<td>-------------</td>
<td>------------------</td>
</tr>
<tr>
<td>Dewey et al. 2002</td>
<td>public and private schools in Calgary, Canada.</td>
<td>45</td>
<td>78</td>
<td>11.8 (NR)</td>
<td>11.4 (NR)</td>
<td>No</td>
</tr>
<tr>
<td>Francis &amp; Piek 2003</td>
<td>Grades 3-5 in primary schools in Perth, Australia.</td>
<td>42</td>
<td>42</td>
<td>NR (7-11)</td>
<td>NR (7-11)</td>
<td>No</td>
</tr>
<tr>
<td>Hill &amp; Brown 2013</td>
<td>Convenience sample: DCD adults via support groups and higher education institutions. TD adults via higher education institutions and local community centres across UK.</td>
<td>Confirmed</td>
<td>36</td>
<td>49</td>
<td>29.28 (19-59)</td>
<td>27.84 (18-56)</td>
</tr>
<tr>
<td>King-Dowling et al. 2015</td>
<td>community organizations in Southern Ontario, Canada.</td>
<td>Probable DCD (performance-based tests)</td>
<td>37</td>
<td>117</td>
<td>4.92 (NR)</td>
<td>4.92 (NR)</td>
</tr>
<tr>
<td>Li et al. 2018</td>
<td>Participants from the Physical Health and Activity Study Team project from schools in Canada.</td>
<td>Probable DCD (performance-based test)</td>
<td>79</td>
<td>1127</td>
<td>13.45 (12-14)</td>
<td>13.40 (12-14)</td>
</tr>
<tr>
<td>Study and Year</td>
<td>Population screening:</td>
<td>Confirmed</td>
<td>Total mean</td>
<td>Total mean</td>
<td>Yes (parent/school-report)</td>
<td>No (not assessed)</td>
</tr>
<tr>
<td>---------------</td>
<td>-----------------------</td>
<td>-------------</td>
<td>------------</td>
<td>------------</td>
<td>---------------------------</td>
<td>-----------------</td>
</tr>
<tr>
<td>Missiuna et al. 2014</td>
<td>Population screening: Grades 4-8 from schools within two health boards regions in Canada.</td>
<td>Confirmed DCD (researcher assessment)</td>
<td>68</td>
<td>91</td>
<td>11.8 (NR)</td>
<td>12.0 (NR)</td>
</tr>
<tr>
<td>Piek et al. 2007</td>
<td>Screening of a twin sample: Monozygotic twins discordant for DCD on the Australian Twin Registry.</td>
<td>Probable DCD (screening questionnaire)</td>
<td>24</td>
<td>24</td>
<td>11.91 (6.45-16.99)</td>
<td>11.91 (6.45-16.99)</td>
</tr>
<tr>
<td>Piek et al. 2008</td>
<td>Population screening: Kindergarten children in a primary school in Western Australia.</td>
<td>Probable DCD (performance-based tests)</td>
<td>14</td>
<td>26</td>
<td>NR; Total sample mean = 4.3 (3.75-5.33)</td>
<td>NR; Total sample mean = 4.3 (3.75-5.33)</td>
</tr>
<tr>
<td>Pratt &amp; Hill 2011</td>
<td>Convenience sample: DCD from support groups, existing research, schools within London and south-east England; TD from existing research and schools within London and south-east England.</td>
<td>Confirmed DCD (medical report)</td>
<td>27</td>
<td>35</td>
<td>10.08 (6-15)</td>
<td>9.38 (6-15)</td>
</tr>
<tr>
<td>Schoemaker &amp; Kalverboer 1994</td>
<td>Population screening: Dutch mainstream schools.</td>
<td>Probable DCD (performance-based test)</td>
<td>18</td>
<td>18</td>
<td>7.3 (6.1-9.0)</td>
<td>7.3 (6.0-9.1)</td>
</tr>
<tr>
<td>Study</td>
<td>Year</td>
<td>Population screening</td>
<td>Method of diagnosis</td>
<td>Sample Size</td>
<td>Range of Age</td>
<td>Range of Performance-based Tests</td>
</tr>
<tr>
<td>------------------</td>
<td>------</td>
<td>--------------------------------------------</td>
<td>-----------------------------------------</td>
<td>-------------</td>
<td>--------------</td>
<td>----------------------------------</td>
</tr>
<tr>
<td>Skinner &amp; Piek (child sample)</td>
<td>2001</td>
<td>Primary schools in Western Australia.</td>
<td>Probable DCD (performance-based tests)</td>
<td>58</td>
<td>NR (8-10)</td>
<td>NR (8-10)</td>
</tr>
<tr>
<td>Skinner &amp; Piek (adolescent sample)</td>
<td>2001</td>
<td>Population screening: High schools in Western Australia.</td>
<td>Probable DCD (performance-based tests)</td>
<td>51</td>
<td>NR (12-14)</td>
<td>NR (12-14)</td>
</tr>
<tr>
<td>van den Huevel et al.</td>
<td>2016</td>
<td>Population screening: Primary schools in middle and eastern Netherlands.</td>
<td>Confirmed DCD (researcher assessment)</td>
<td>TRF: 20; SDQ 22</td>
<td>TRF: 307; SDQ: 339</td>
<td>NR; For total sample (not all included in analysis): 7.0 (4.3-10.3)</td>
</tr>
<tr>
<td>Wagner et al.</td>
<td>2012</td>
<td>DCD: Clinical sample from occupational therapy groups in Germany; TD: elementary schools in Germany.</td>
<td>Confirmed DCD (researcher assessed)</td>
<td>35</td>
<td>7.69 (5-11)</td>
<td>NR “matched”</td>
</tr>
<tr>
<td>Watson &amp; Knott 2006</td>
<td>DCD: Clinical sample from Occupational therapy department in West of Scotland; TD: Primary schools in West of Scotland.</td>
<td>Confirmed DCD (medical report)</td>
<td>15</td>
<td>30</td>
<td>10.2 (8-12)</td>
<td>10.25 (NR)</td>
</tr>
</tbody>
</table>

BASC: Behaviour Assessment System for Children; BDI: Beck Depression Inventory; CBCL: Child Behaviour Checklist; CDI: Children’s Depression Inventory; ELBW: Extremely Low Birth Weight; IDS: Intelligence and Developmental Scales; NR: Not reported; SCARED: Screen for Child Anxiety Related Disorders; SCAS: Spence Children’s Anxiety Scale; SDQ: Strengths and Difficulties Questionnaire; SMFQ: Short Moods and Feelings Questionnaire; STAI: State-Trait Anxiety Inventory for Adults; STAIC: State-Trait Anxiety Inventory for Children; TRF: Teacher Report Form.

a Additional confounders included in adjusted analysis: maternal depression, family adversity, IQ, social communication.
b Based on reported age for combined male and female subsamples.
c Study reports two alternative performance-based measures for determining DCD. Criteria based on the MABC were used for comparability with other studies.
d Adjusted for gender and IQ, but unadjusted data used in meta-analysis.
Six studies explicitly excluded DCD participants with ADHD based on self-/parent-/school-reported diagnoses (Crane et al., 2017; Hill & Brown, 2013; Missiuna et al., 2014; Watson & Knott, 2006) or based on scores on standardised screening questionnaires (Piek et al., 2007) or either of the two (Pratt & Hill, 2011). The remaining studies either did not assess for ADHD (Campbell et al., 2012; Davis et al., 2007; Francis & Piek, 2003; King-Dowling et al., 2015; Li et al., 2018; Piek et al., 2008; Schoemaker & Kalverboer, 1994; Skinner & Piek, 2001; Wagner et al., 2012) or measured symptoms but did not exclude those above the clinical cut-off (Chen et al., 2009; Dewey et al., 2002; Harrowell et al., 2017; van den Heuvel et al., 2016; Wagner et al., 2016).

All outcome measures of internalising symptoms were based on questionnaires. Most studies measured overall internalising symptoms using the Child Behaviour Checklist (Chen et al., 2009; Dewey et al., 2002; King-Dowling et al., 2015a), Strengths and Difficulties Questionnaire (Crane et al., 2017; Harrowell et al., 2017; van den Heuvel et al., 2016; Wagner et al., 2016), Behaviour Assessment System for Children (BASC; Davis, Ford, Anderson, & Doyle, 2007), Teacher Report Form (van den Heuvel et al., 2016), Kessler-6 (Li et al., 2018), or the Intelligence and Developmental Scales (Wagner et al., 2012). Nine studies specifically measured depressive symptoms using the Children’s Depression Inventory (Francis & Piek, 2003; Missiuna et al., 2014), Beck Depression Inventory (Hill & Brown, 2013), Short Mood and Feelings Questionnaire (Harrowell et al., 2017), BASC – Depression subscale (Campbell et al., 2012), Twin and
Sibling Questionnaire – Sad Affect subscale (Piek et al., 2007), or the Birleson Depression Measure (Watson & Knott, 2006). Six studies specifically measured levels of anxiety symptoms using the State-Trait Anxiety Inventory for Children (Schoemaker & Kalverboer, 1994; Skinner & Piek, 2001), State-Trait Anxiety Inventory for Adults (Hill & Brown, 2013), Screen for Child Anxiety Related Disorders (Missiuna et al., 2014), or Spence Children's Anxiety Scale (Pratt & Hill, 2011). Overall, the outcome measures were based on parent-report in seven studies (Chen et al., 2009; Dewey et al., 2002; King-Dowling et al., 2015; Piek et al., 2007, 2008; Pratt & Hill, 2011; Wagner et al., 2016; Wagner et al., 2012), teacher-report in two studies (Crane et al., 2017; van den Heuvel et al., 2016), self-report in eleven studies (Campbell et al., 2012; Francis & Piek, 2003; Harrowell et al., 2017; Hill & Brown, 2013; Li et al., 2018; Schoemaker & Kalverboer, 1994; Skinner & Piek, 2001; Watson & Knott, 2006), and a combination in two studies (Davis et al., 2007; Missiuna et al., 2014).

Risk of bias

The risk of bias in the included studies is summarised in Table 2. Selection bias was variable. The use of a population screening method in 16 studies ensured the DCD sample was somewhat representative of the population studied. However, seven studies recruited the DCD sample from a selective group or convenience sample, which may be at greater risk of bias. This included children born very preterm or with extremely low birth weight (Davis et al., 2007), volunteer samples from DCD support groups (Crane et al., 2017; Hill & Brown, 2013; Pratt & Hill, 2011), monozygotic twin samples (Piek et al., 2007), or clinical samples from occupational therapy services (Wagner et al., 2012;
Watson & Knott, 2006). In most studies, the TD group was drawn from the same community (e.g. school, geographical location) as the DCD group, except for four studies that were from a different source (Crane et al., 2017; Hill & Brown, 2013; Pratt & Hill, 2011; Wagner et al., 2012) and thus had an increased risk from selection bias. Additionally, while 10 studies confirmed diagnoses of DCD by independent assessment or clinical reports (Chen et al., 2009; Crane et al., 2017; Harrowell et al., 2017; Hill & Brown, 2013; Missiuna et al., 2014; Pratt & Hill, 2011; van den Heuvel et al., 2016; Wagner, 2017; Watson & Knott, 2006), 13 did not confirm key diagnostic criteria (Campbell et al., 2012; Davis et al., 2007; Dewey et al., 2002; Francis & Piek, 2003; King-Dowling et al., 2015; Li et al., 2018; Piek et al., 2007, 2008; Schoemaker & Kalverboer, 1994; Skinner & Piek, 2001; Wagner et al., 2016). Caution should be taken, therefore, when attributing differences in internalising symptoms in these studies to DCD. Most studies were cross-sectional and, therefore, unable to account for the stability of internalising symptoms over time. Of the longitudinal studies, one controlled for baseline internalising symptoms (Wagner et al., 2016).

The studies varied in the comparability of study groups and control for important confounders. Age and gender were controlled in 11 studies through either matched groups (Campbell et al., 2012; Francis & Piek, 2003; Piek et al., 2007; Schoemaker & Kalverboer, 1994; Skinner & Piek, 2001; Wagner et al., 2012; Watson & Knott, 2006) or adjusted analyses (Harrowell et al., 2017; Wagner et al., 2016). The study by Piek et al. (2007) adopted a monozygotic twin design which also controls for a wide range of genetic and shared environmental factors. The remaining studies failed to sufficiently
Table 2. Summary of risk of bias

<table>
<thead>
<tr>
<th>Study</th>
<th>DCD representative of population</th>
<th>TD from the same population</th>
<th>DCD diagnosis confirmed</th>
<th>Prior internalising symptoms controlled</th>
<th>Controls for gender</th>
<th>Controls for age</th>
<th>Standardised outcome measure</th>
<th>Sufficient length of follow-up</th>
<th>Sufficient follow-up rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Campbell et al. (2012, male)</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>Campbell et al. (2012, female)</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>Chen et al. (2009)</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td></td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>Crane et al. (2017)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>*</td>
<td></td>
<td></td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>Davis et al. (2007)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>*</td>
<td></td>
<td></td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>Dewey et al. (2002)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>*</td>
<td></td>
<td></td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>Francis and Piek (2003)</td>
<td>*</td>
<td>*</td>
<td></td>
<td></td>
<td>*</td>
<td></td>
<td></td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>Harrowell et al. (2017, male)</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>Harrowell et al. (2017, female)</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>Hill and Brown (2013)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>*</td>
<td></td>
<td></td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>King-Dowling et al. (2015)</td>
<td>*</td>
<td>*</td>
<td></td>
<td></td>
<td>*</td>
<td></td>
<td>*</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>Li et al. (2018)</td>
<td>*</td>
<td>*</td>
<td></td>
<td></td>
<td>*</td>
<td></td>
<td>*</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>Missiuna et al. (2014)</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>Piek et al. (2007)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>*</td>
<td></td>
<td></td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>Piek et al. (2008)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>*</td>
<td></td>
<td></td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>Pratt and Hill (2011)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>*</td>
<td></td>
<td></td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>Schoemaker and Kalverboer (1994)</td>
<td>*</td>
<td>*</td>
<td></td>
<td></td>
<td>*</td>
<td></td>
<td>*</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>Skinner &amp; Piek (2001, adolescent)</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>Skinner &amp; Piek (2001, child)</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>van den Hovevel (2016)</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td></td>
<td>*</td>
<td></td>
<td></td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>Wagner et al. (2012)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>*</td>
<td></td>
<td></td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>Wagner et al. (2016)</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>Watson and Knott (2016)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>*</td>
<td></td>
<td></td>
<td>*</td>
<td>*</td>
</tr>
</tbody>
</table>
control for age and gender. Although one such study did adjust for differences in gender in the analysis (Piek et al., 2008), it also included intellectual ability as a covariate, and so the unadjusted difference between the groups was used in the meta-analysis to ensure comparability (Voils et al., 2011).

All studies utilised outcome measures with established validity and reliability psychometrics. It should be noted that three studies only reported specific narrow-band depression or anxiety subscales, as opposed to using the broad-band internalising scales (Campbell et al., 2012; Piek et al., 2008). These might raise concerns around selective reporting.

Finally, only three studies included a longitudinal follow-up (Harrowell et al., 2017, male & female sample; Wagner et al., 2016). This was over a year in all three studies. However, there were high rates of attrition in all three.

**Internalising symptoms**

Since only one study based on an adult sample was identified (Hill & Brown, 2013), that study was excluded from the remaining analyses to minimise heterogeneity. Across the 22 studies with children and adolescents, those with DCD or probable DCD were found to have higher levels of internalising symptoms than TD controls with a medium effect size ($g = 0.61; 95\% \text{ CI: } 0.48-0.74; \text{ see Figure 3 for forest plot}$). There was significant moderate heterogeneity among the studies ($I^2 = 56\%; \chi^2 = 47.84; p = 0.0007$).
**Figure 3.** Forest plot for total internalising symptoms across all studies
Moderator analysis

The results of the moderator analyses are summarised in Table 3. The results revealed that the effect size was significantly larger in studies that utilised a cross-sectional design (versus longitudinal), that included a majority male sample in the DCD group (versus majority female) and that recruited a selective or convenience sample of participants (as opposed to population-based screening). There was also a trend ($p = .05$) towards a greater effect size in studies that did not control for important confounders and in studies that excluded individuals with a diagnosis of ADHD. No significant effect was found for publication year, age, confirmation of DCD diagnosis, outcome respondent, or type of internalising measure.

Sensitivity analysis

A sensitivity analysis was conducted to include only those studies meeting five or more criteria on the NOS. A moderator analysis identified a significant difference between the high-quality and low-quality studies ($Q = 4.42; p = 0.04$). Analysis of the higher quality studies ($k = 10$) found a smaller, but still moderate, effect of DCD on internalising symptoms ($g = 0.46; 95\%$ CI: 0.34 to 0.58). There was also no evidence of significant heterogeneity among these ten studies ($Q = 5.95; p = 0.55; I^2 = 0\%$).

Publication bias

The funnel plot (see Figure 4) displayed some asymmetry, with smaller studies tending to report larger effect sizes, possibly indicative of publication bias. Eggers test was statistically significant, supporting the presence of publication bias (Egger’s bias =
Table 3. Summary of results for moderators

<table>
<thead>
<tr>
<th>Moderator</th>
<th>k</th>
<th>Total n</th>
<th>g</th>
<th>95% CI</th>
<th>Q</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Year of publication</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.31</td>
<td>0.58</td>
</tr>
<tr>
<td>- Post-2010</td>
<td>12</td>
<td>3388</td>
<td>0.58</td>
<td>0.39 to 0.77</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Pre-2010</td>
<td>10</td>
<td>1074</td>
<td>0.65</td>
<td>0.49 to 0.81</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Design</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>9.33</td>
<td>0.002</td>
</tr>
<tr>
<td>- Longitudinal</td>
<td>3</td>
<td>937</td>
<td>0.29</td>
<td>0.09 to 0.49</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Cross-sectional</td>
<td>20</td>
<td>3645</td>
<td>0.67</td>
<td>0.54 to 0.80</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.02</td>
<td>0.88</td>
</tr>
<tr>
<td>- Included adolescents</td>
<td>10</td>
<td>2682</td>
<td>0.60</td>
<td>0.39 to 0.80</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Not included adolescents</td>
<td>12</td>
<td>1780</td>
<td>0.62</td>
<td>0.44 to 0.79</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>4.72</td>
<td>0.03</td>
</tr>
<tr>
<td>- &gt;50% male</td>
<td>15</td>
<td>1889</td>
<td>0.71</td>
<td>0.52 to 0.91</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- &gt;50% female</td>
<td>7</td>
<td>2573</td>
<td>0.46</td>
<td>0.33 to 0.58</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Confirmed diagnoses</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2.30</td>
<td>0.13</td>
</tr>
<tr>
<td>- Confirmed DCD</td>
<td>9</td>
<td>998</td>
<td>0.75</td>
<td>0.48 to 1.01</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Probable DCD</td>
<td>13</td>
<td>3434</td>
<td>0.52</td>
<td>0.39 to 0.65</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Population-based design</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>5.06</td>
<td>0.02</td>
</tr>
<tr>
<td>- Population screening</td>
<td>17</td>
<td>4176</td>
<td>0.52</td>
<td>0.41 to 0.62</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Selective sample</td>
<td>4</td>
<td>286</td>
<td>1.02</td>
<td>0.59 to 1.45</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ADHD</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3.70</td>
<td>0.05</td>
</tr>
<tr>
<td>- Excluded ADHD</td>
<td>5</td>
<td>379</td>
<td>0.97</td>
<td>0.53 to 1.41</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Did not exclude ADHD</td>
<td>17</td>
<td>4083</td>
<td>0.52</td>
<td>0.41 to 0.63</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Controlled for confounding</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3.81</td>
<td>0.05</td>
</tr>
<tr>
<td>- Age and gender controlled</td>
<td>12</td>
<td>1752</td>
<td>0.48</td>
<td>0.36 to 0.60</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Age and gender not controlled</td>
<td>10</td>
<td>2710</td>
<td>0.74</td>
<td>0.51 to 0.97</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Outcome respondent</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2.18</td>
<td>0.14</td>
</tr>
<tr>
<td>- Self-report</td>
<td>10</td>
<td>1907</td>
<td>0.50</td>
<td>0.39 to 0.62</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Parent/teacher-report</td>
<td>12</td>
<td>2396</td>
<td>0.71</td>
<td>0.46 to 0.97</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Outcome measure type

- Total internalising 12 3492 0.59 0.39 to 0.78
- Depression only 5 495 0.54 0.36 to 0.72
- Anxiety only 4 316 0.77 0.38 to 1.16

Sensitivity analyses

Study quality 4.42 0.04
- High quality 10 1638 0.46 0.34 to 0.58
- Low quality 12 2800 0.72 0.51 to 0.93

ADHD: Attention Deficit Hyperactivity Disorder.

*Missiuna et al. (2014) excluded from analysis due to using both self-and observer-report. Inclusion of each type of measure from this study, independently, did not significantly change the results.

Figure 4. Funnel plot of effect sizes and standard error
2.38; 95% CI: 0.20 to 4.56; \( p = 0.02 \)). However, Duval and Tweedie’s Trim and Fill procedure did not impute additional studies and, therefore, the effect size adjusted for publication bias was identical to the non-adjusted effect size. Rosenthal’s Fail-safe \( N \), suggested that the number of studies with null results that would have to be included to produce a non-significant combined effect size is 1076. This is substantially larger than the minimum required when applying Rosenthal’s (1979) formula (i.e. 120).

**Discussion**

The present systematic review and meta-analysis indicates that children and adolescents with DCD or probable DCD experience greater levels of internalising symptoms compared to their TD peers. The magnitude of this difference suggests a moderate effect size, with individuals with DCD scoring over half a standard deviation higher. This moderate effect, although reduced slightly, remained robust after excluding lower quality studies. Publication bias is also unlikely to have substantially influenced the result. Several methodological and participant factors that may moderate the magnitude of this effect have also been identified.

**DCD and internalising symptoms**

The findings are in line with the emerging consensus that DCD can have a significant impact on an individual’s mental health (Caçola, 2016; Mancini et al., 2016; Missiuna & Campbell, 2014). Notably, the magnitude of the effect identified is comparable, if not greater, than that found in meta-analyses of a wide range of chronic
physical health conditions including fibromyalgia, cleft lip and palate, migraine sufferers, diabetes, spina bifida, and epilepsy (Pinquart & Shen, 2011a, 2011b).

The Environmental-Stress Hypothesis was proposed to account for this relationship between DCD and internalising symptoms (Cairney, Rigoli, & Piek, 2013). It suggests that the motor impairments in DCD can expose an individual to a variety of secondary stressors, which over time can lead to poorer mental health. Although potential mediators were not explored in this review, they have been outlined previously (Mancini et al., 2016). They include peer victimisation (Campbell et al., 2012), reduced leisure activities (Raz-Silbiger et al., 2015), impaired social skills (A. Wilson et al., 2013), poorer self-esteem (Rigoli et al., 2012), physical inactivity (Li et al., 2018), reduced social support (Rigoli et al., 2017), and lower perceived academic competence (Lingam et al., 2012). Individuals with DCD may also experience impairments to various cognitive abilities, including executive functions (Wilson, Ruddock, Smits-Engelsman, Polatajko, & Blank, 2013) and social cognition (Cummins, Piek, & Dyck, 2005). This may further impact on self-regulation and mental wellbeing (Lantrip, Isquith, Koven, Welsh, & Roth, 2016; Letkiewicz et al., 2014). Future meta-analyses regarding the magnitude of the effects for these potential mediators will be important, providing further opportunities for intervention.

**Moderating factors**

The present review has identified several methodological factors which might moderate the degree to which DCD is associated with internalising symptoms. As expected, the effect size was likely over-estimated in cross-sectional compared to
longitudinal studies, and in convenience or clinic-referred samples compared to samples recruited via population-based screening. Such methodologies have less control of confounding factors and a less representative selection of DCD participants (e.g. more severe impairments in clinical samples). There was also a trend towards larger effect sizes in studies that failed to control for age and gender. Again, the magnitude of the effect sizes in these studies were likely inflated by confounding (Deeks et al., 2003). No significant effect was found for the DCD criteria used. This suggests that, although establishing all DCD diagnostic criteria is important for the quality of research in this area (Zwicker et al., 2013), failing to do this might not have a substantial impact on the results. Population-based screening, longitudinal design and control for confounders should take priority.

Participant factors that might moderate the effect of DCD on internalising symptoms were also identified. The effect was larger in studies with a majority male sample. This would suggest that DCD has a greater impact on the mental health of males and is in line with the findings of Sigurdsson et al. (2002). This is particularly important given the prevalence of DCD may be greater in males (Kirby & Sugden, 2007; Missiuna, 1994). It has been suggested that male children attribute greater value to physical activity and sports compared to females, which might account for the larger impact of DCD on their wellbeing (Poulsen, Ziviani, & Cuskelly, 2006; Poulsen, Ziviani, Cuskelly, & Smith, 2007). However, although significant, the difference in magnitude of the effect sizes were minimal. Additionally, only two of the included studies reported direct comparisons of the impact of DCD on males and females, with one suggesting no difference (Campbell et al., 2012) and the other suggesting a greater
impact for females (Harrowell et al., 2017). Regardless of which gender experiences the larger effect, there is evidence that DCD can impact on the mental health of both and perhaps it is the mechanisms by which this occurs that differ (Li et al., 2018).

There was also a trend towards larger effect sizes in studies that specifically excluded participants with ADHD. This contradicts what might have been expected from previous research (Martin, Piek, & Hay, 2006; Missiuna et al., 2014; Rasmussen & Gillberg, 2000). One possible explanation for this finding is that children with comorbid ADHD are more likely to be diagnosed and to subsequently receive support for their difficulties (Heath, Toste, & Missiuna, 2005; Rivard, Missiuna, Hanna, & Wishart, 2011). Participants with DCD only, on the other hand, may have continued through childhood for a long time with the motor difficulties going unrecognised and unsupported (Gaines et al., 2008). However, it should also be noted that there were only five studies included in the meta-analysis that specifically excluded participants with ADHD. All five studies were also of a lower quality. It is a more plausible explanation that methodological limitations inflated their combined effect size and, therefore, caution should be taken when drawing any conclusions about the moderating effect of comorbid ADHD.

Contrary to what might be expected, no significant moderating effect was found for age. This is somewhat surprising, given that previous research has suggested the impact of DCD on internalising symptoms may increase as children transition into adolescence (Missiuna, Moll, King, King, & Law, 2007; Skinner & Piek, 2001). However, only one study included in the present review reported separate outcomes for adolescents and children. The moderator categories for the meta-analysis were based on
somewhat arbitrary criteria (i.e. studies that included adolescents in their sample, as opposed to studies with a pure adolescent sample) which may have prevented the detection of differences between the age groups.

Finally, no significant effect was found for the type of outcome measure or respondent, suggesting that DCD may be associated with elevated levels of depression, anxiety, and overall internalising symptoms, regardless of the person rating it.

**Strengths and limitations**

There are several limitations of this review that should be considered when interpreting the findings. First, the quality of the included studies was variable. Most studies were based on cross-sectional data only, which makes it difficult to establish causality. Although three longitudinal studies were included, only one controlled for baseline measures of internalising symptoms and all reported a high rate of attrition. Many of the studies also failed to control for important confounders (i.e. age and gender) and to establish all DCD diagnostic criteria.

The moderator analysis should also be interpreted with caution. As outlined above, there were an insufficient number of studies within some of the moderator categories to reliably explore their impact (including age and comorbid ADHD). Most of the studies were also conducted in western, developed countries and, therefore, generalisation to other countries is limited. Additionally, only one study with adults was identified and this study failed to meet many of the quality criteria. Therefore, the extent
to which elevated internalising symptoms persist into adulthood in people with DCD is unclear.

However, this review is the first attempt to systematically synthesise the evidence on internalising symptoms in individuals with DCD and provide a pooled summary of the effect size. Publication bias is unlikely to have a substantial impact on the findings. Additionally, despite methodological limitations of the included studies, potential moderating factors have been identified. The effect size also remained substantial, and heterogeneity reduced, after excluding lower quality studies.

**Implications and conclusion**

Despite some limitations, the findings of this review have important clinical and research implications. It can be concluded that individuals with DCD may have an increased risk of developing elevated levels of internalising symptoms. The difference of half a standard deviation between individuals with DCD and their peers could be considered clinically important (Norman, Sloan, & Wyrwich, 2003). This would support the practice of routine screening of mental health difficulties in individuals with DCD and motor impairment. Given that DCD is poorly understood among professionals (Gaines et al., 2008; B. N. Wilson et al., 2013) and that families often report difficulties obtaining support (Stephenson & Chesson, 2008), such routine screening could be useful across a range of services (including schools, occupational therapy, physical healthcare, and mental healthcare). The findings also highlight the need for professionals in mental health services to be aware of the disorder and how it impacts their patients. Additionally, the findings support the need for the development of psychosocial
interventions for DCD with a focus on the secondary stressors that might mediate the link between motor difficulties and emotional wellbeing (Missiuna et al., 2012).

Future research should focus on high-quality longitudinal studies to better understand the causal link between DCD and internalising symptoms, including the role of important mediators. It is recommended that studies include probability sampling strategies and control for confounders and the stability of internalising symptoms over time. This review has also highlighted the need for more research investigating mental health in adults with DCD, especially given that the impact of DCD has been found to continue into adulthood, including negative effects on higher education, employment and mental wellbeing (Cousins & Smyth, 2003; Hill et al., 2011; Kirby, Williams, Thomas, & Hill, 2013). Research investigating the effectiveness of routine screening for mental health difficulties in DCD and psychosocial interventions would also provide insight into the improved management of DCD. Given the major economic impact of poor mental health (Trautmann, Rehm, & Wittchen, 2016) and the increasing focus on improving psychological wellbeing in government policy (Department of Health, 2011), the need to identify and support those individuals most at risk of mental health difficulties is crucial.

References


and intervention of developmental coordination disorder (long version).

*Developmental Medicine & Child Neurology, 54*(1), 54–93.


Appendix: Guidelines for authors

[Appendix removed for E-Thesis submission]
Part 3: Summary of Clinical Experience

**Adult placement**

*Service:* Community Mental Health Team  
*Dates:* October 2015 – September 2016  
*Experience:* Conducting psychological assessments and interventions with adults presenting with a range of mental health difficulties. I predominantly worked within a Cognitive-Behavioural Therapy (CBT) model. This included CBT for depression, generalised anxiety disorder, social anxiety, obsessive compulsive disorder, health anxiety and panic disorder. I gained experience applying third-wave CBT models (e.g. Acceptance and Commitment Therapy) in one-to-one sessions and facilitating a Dialectical Behaviour Therapy (DBT) group for people diagnosed with borderline personality disorder. I gained some experience administering neuropsychological tests. I also conducted a service evaluation of a multidisciplinary referrals meeting.

**Child and adolescent placement**

*Service:* Tier 2 Child and Adolescent Mental Health Team  
*Dates:* October 2016 – March 2017  
*Experience:* Working within a Choice and Partnership Approach to conduct psychological assessments and intervention with young people presenting with mild-moderate mental health difficulties and their families. I predominantly worked within the CBT model with some experience applying systemic models. Presenting concerns included depression, anxiety and behavioural difficulties. I gained experience working
with young people with Attention Deficit Hyperactivity Disorder and Autism Spectrum Conditions. I facilitated a CBT group for anxiety. I administered neuropsychological tests. I had the opportunity to conduct consultation work for local schools and provided a teaching session to health professionals on Developmental Coordination Disorder.

**Older people placement**

*Service:* Memory Assessment Service  
*Dates:* April 2017 – September 2017  
*Experience:* Conducting detailed cognitive assessments and formulations with adults presenting with possible dementia. I gained experience administering and scoring a range of neuropsychological tests and working within a neurocognitive model to assist with diagnosis. I delivered brief interventions with people adjusting to a recent diagnosis of dementia and their carers. This included one-to-one, family, and group sessions focused on psychoeducation, CBT, systemic therapy, neurorehabilitation, and cognitive stimulation therapy. Presenting diagnoses included Alzheimer’s disease, vascular dementia, frontotemporal dementia, Lewy-bodies dementia, Parkinson’s disease, progressive supranuclear palsy, and mild cognitive impairment. I also delivered a training session to the multidisciplinary team on delivering a diagnosis of dementia.

**People with Learning Disabilities placement**

*Service:* Community Team for People with Learning Disabilities  
*Dates:* October 2017 – March 2018
Experience: Adapting psychological models and interventions for adults with a learning disability. This included working within an integrated framework, informed by CBT, systemic, neurocognitive, and behavioural models. Presenting concerns included low mood, anxiety, anger, and behaviours that challenge. I gained experience conducting functional assessments and developing Positive Behaviour Support plans to help clients and their carers manage challenging behaviours. I gained experience with consultation and joint working with other professionals. I also conducted relaxation groups within an inpatient service. I had the opportunity to supervise assistant psychologists.

Specialist placement: Neuropsychology

Service: Epilepsy neuropsychology outpatients / inpatient acute traumatic brain injury

Dates: April 2018 – September 2018

Experience: Conducting neuropsychological assessment with people with a diagnosis of epilepsy. This included individuals concerned by cognitive difficulties or as part of a presurgical screening assessment. I gained experience working within the neurocognitive model and writing detailed neuropsychological reports. I also worked in an inpatient acute neurorehabilitation service for people with a traumatic brain injury. I gained experience with neuropsychological assessment; delivering brain injury education groups; working with families; conducting mood assessments and intervention; delivering insight-raising interventions; and introducing cognitive rehabilitation strategies. I obtained experience working closely with other healthcare professionals, taking on a leadership role within the team, and supervising assistant psychologists.
Part 4: Table of Assessments Completed During Training

### Year I Assessments

<table>
<thead>
<tr>
<th>ASSESSMENT</th>
<th>TITLE</th>
</tr>
</thead>
<tbody>
<tr>
<td>WAIS</td>
<td>WAIS Interpretation (online assessment)</td>
</tr>
<tr>
<td>Practice Report of Clinical Activity</td>
<td>A cognitive-behavioural assessment and initial formulation of a female in late adolescence experiencing anxiety, low mood, and non-epileptic seizures</td>
</tr>
<tr>
<td>Audio Recording of Clinical Activity with Critical Appraisal</td>
<td>Audio recording and critical appraisal of a Cognitive Behaviour Therapy session with a male with depression and social anxiety</td>
</tr>
<tr>
<td>Report of Clinical Activity N=1</td>
<td>A cognitive-behavioural assessment and intervention for a man in his twenties experiencing depression and social anxiety</td>
</tr>
<tr>
<td>Major Research Project Literature Survey</td>
<td>Mental health in Developmental Coordination Disorder: Literature Survey</td>
</tr>
<tr>
<td>Major Research Project Proposal</td>
<td>Executive function and internalizing problems in children with and without Developmental Coordination Disorder</td>
</tr>
<tr>
<td>Service-Related Project</td>
<td>Clinicians’ views on a weekly meeting dedicated to discussing new referrals in a Community Mental Health &amp; Recovery Service</td>
</tr>
</tbody>
</table>

### Year II Assessments

<table>
<thead>
<tr>
<th>ASSESSMENT</th>
<th>TITLE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Report of Clinical Activity/Report of Clinical Activity – Formal Assessment</td>
<td>A neuropsychological assessment of a male in his mid-teens, presenting with academic and behavioural difficulties at school</td>
</tr>
<tr>
<td>PPLD Process Account</td>
<td>A reflective process account of a trainee’s experiences of a Personal and Professional Development group during the first two years of training</td>
</tr>
</tbody>
</table>

### Year III Assessments

<table>
<thead>
<tr>
<th>ASSESSMENT</th>
<th>TITLE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Presentation of Clinical Activity</td>
<td>A post-diagnostic intervention for a woman in her 70’s recently diagnosed with vascular dementia: A cognitive-behavioural and systemic approach</td>
</tr>
<tr>
<td>Major Research Project Literature Review</td>
<td>Internalising symptoms in Developmental Coordination Disorder: A systematic review and meta-analysis</td>
</tr>
<tr>
<td>----------------------------------------</td>
<td>--------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Major Research Project Empirical Paper</td>
<td>Internalising symptoms and executive function difficulties in adolescents with and without Developmental Coordination Disorder</td>
</tr>
<tr>
<td>Final Reflective Account</td>
<td>On becoming a clinical psychologist: A retrospective, developmental, reflective account of the experience of training</td>
</tr>
</tbody>
</table>