Rapid online control in children with Developmental Coordination Disorder (DCD)

A thesis submitted in fulfillment of the requirements for the degree of Doctor of Philosophy

Christian Edward Anthony Hyde
BSc., Grad. Dip. Psych.

Division of Psychology
School of Health Sciences
Science, Engineering and Technology (SET) Portfolio
RMIT University

December 2010
Declaration of Authorship

I certify that except where due acknowledgement has been made, the work is that of the author alone; the work has not been submitted previously, in whole or in part, to qualify for any other academic award; the content of the thesis is the result of work which has been carried out since the official commencement date of the approved research program; and any editorial work, paid or unpaid, carried out by a third party is acknowledged.

Aspects of the studies presented in this thesis have been accepted for publication:


Signed:

________________________

Christian Hyde

Date:
Acknowledgements

Firstly, I would like to acknowledge my primary supervisor Prof Peter Wilson for all his hard work and perseverance. Thank-you for the opportunities, support and guidance that you have provided me with over these years. I would also like to thank my co-supervisor, Prof Paul Maruff for his flexibility and assistance whenever it was required.

Thank-you to the primary schools who participated in this research, their staff, students and parents who were all so accommodating. Special thanks to James Quilty for being my go-between man.

Thank-you to all the RMIT staff and fellow postgraduate students who have helped me along the way. I have thoroughly enjoyed my years as a PhD student and that is largely due to the fact that you all made going to work and doing my research so much fun. I would especially like to thank Assoc. Prof. Andrew Francis and Assoc. Prof. Andrea Chester for their encouragement; Jan Elliot, for all her hard work and for listening to my waffle (though this probably extended my submission date by months); Scott Ruddock and Alana, for taking the time to help me proof read my thesis; and Ray Duckman, for his technical assistance throughout my research.

To my family and friends who have always supported me at every stage of my life, no matter what my choices, it is hard find the words to thank you all. Special thank-you to Mum, for your love and care; Chris, for your amazing strength; my grandparents, Noel and Margaret, who have been such an incredible support; Lindsey, Ian and Simon for the company, meals, drinks and even roof over my head for a brief period there; the Maniscalchi family, for being the wonderful people you are and for not trying to convince Cass to find someone with a real job; my siblings, for not
disowning me when I forgot your birthdays and didn’t visit for months at a time; my partners in crime, Ant and Austin for helping me enjoy the lighter side of life; and to Ali, Dale, Q, and Matt. To you, I’d just like to say… this thesis is a beautiful piece!

Finally, to Cass. I guess now is the time to tell you that I lied. PhD’s don’t make a lot of money. You will still be partnered to a poor man after all these years of study, albeit a slightly more educated one. Your unconditional friendship, patience and love through these years has left me with a debt that I will never be able to repay. I promise I will never stop trying though. Thank-you for everything.

To anyone I have forgotten to thank, I am sorry. However, it is just not possible to thank everyone who deserves it individually because there are so many of you. If this means you, I owe you a beer.
Table of contents

SUMMARY ........................................................................................................................................1

CHAPTER 1
AN OVERVIEW OF RESEARCH INTO DCD .................................................................6

INTRODUCTION .......................................................................................................................7

Overview of Chapter 1 ...........................................................................................................7

Developmental Coordination Disorder: Diagnosis, Presentation, and Associated Problems .................................................................................................................................8

Systems for identifying and diagnosing motor impairment in children .................9

*DSM-IV criteria for DCD.* ..................................................................................................10

*ICD system for diagnosing motor impairment.* ............................................................13

*Overview of the DSM-IV and ICD-10 systems.* .........................................................16

Presentation of the Disorder .............................................................................................17

*Secondary Social, Psychological, and Educational Issues.* .....................................18

Research into DCD ...........................................................................................................19

*Descriptive Research.* ...................................................................................................19

*Intervention Studies.* .....................................................................................................21

Aetiological Accounts of DCD .........................................................................................23

*General Maturational Delay.* .....................................................................................24

*Neurological Impairment.* ...........................................................................................25

*Atypical Brain Development (ABD) Theory.* ............................................................26

*Deficits in Attention, Motor Control and Perception (DAMP) Theory.* ..............27

*Automatization Deficit Hypothesis.* ........................................................................27

*Conclusion.* ....................................................................................................................28
Information Processing Accounts. ................................................................. 29
Cognitive Neuroscientific Approaches. .......................................................... 30
Timing Deficit Hypotheses. ............................................................................. 32
Conclusion. ........................................................................................................ 35
Disinhibition and Poor Motor Skill. ................................................................. 36
Conclusion. ........................................................................................................ 38
Predictive Control (viz internal modeling) and DCD ..................................... 39
Internal modeling and motor control............................................................... 39
Predictive modeling is impaired in children with DCD. ................................. 40
DCD and Motor Imagery: A window into an IMD ........................................... 41
Further evidence for impaired predictive modeling in DCD ......................... 43
Motor control and adaptation: the role of predictive modeling ................. 44
Motor control studies ...................................................................................... 46
Learning (adaptation) studies ......................................................................... 48
Conclusion. ........................................................................................................ 49
Summary of Cognitive Neuroscientific Accounts of DCD ............................ 50

Rapid online motor control in children with Developmental Coordination
Disorder (DCD) ................................................................................................. 51
Double-step reaching tasks and online control. ........................................... 51
Double-step reaching performance in DCD .................................................... 52
Performance on double-step tasks in DCD: preliminary insight into online
control ............................................................................................................... 54

Double-step reaching interpreted using computational models can clarify the
nature of ROC in DCD and issue surrounding the IMD hypothesis............... 56
Summary of Chapter 1 ..................................................................................... 58
CHAPTER 2

STUDY 1: DOUBLE-STEP REACHING PERFORMANCE IN CHILDREN WITH DEVELOPMENTAL COORDINATION DISORDER: CHRONOMETRIC

ANALYSIS OF REACHING ................................................................. 60

INTRODUCTION AND OVERVIEW .................................................. 61

  Impaired ROC and Optic Ataxia .................................................. 62
  Summary. ................................................................................. 62

METHOD ....................................................................................... 63

Participants .................................................................................... 63
Apparatus ...................................................................................... 64
  Double-step reaching paradigm (DSRP) ....................................... 64
Procedure ..................................................................................... 65
Design and Analysis ................................................................. 67

RESULTS ........................................................................................ 68

Reaction Time .............................................................................. 68
Movement Time ............................................................................ 69
  Analysis of Group Differences .................................................. 69
  Analysis of Individual Differences .......................................... 70
  Analysis of $MT_{\text{diff}}$ scores within each group (DCD and Control). ........ 71
Errors .......................................................................................... 72
Summary of results ....................................................................... 72

DISCUSSION ................................................................................ 73

Reaction Time .............................................................................. 74
Movement Time ............................................................................ 74
  Analysis of age differences within each motor group ................. 77
CHAPTER 3

STUDY 2: DOUBLE-STEP REACHING PERFORMANCE IN CHILDREN WITH DEVELOPMENTAL COORDINATION DISORDER: KINEMATIC ANALYSIS OF REACHING

INTRODUCTION AND OVERVIEW .................................................................................. 85

Summary. ......................................................................................................................... 87

METHOD ......................................................................................................................... 88

Participants ..................................................................................................................... 88

Apparatus ....................................................................................................................... 88

Double-step reaching paradigm (DSRP). ................................................................. 88

Procedure ....................................................................................................................... 89

Design and Analysis .................................................................................................... 90

RESULTS ....................................................................................................................... 91

Chronometric Analysis ............................................................................................... 91

Reaction Time. ............................................................................................................. 91

Movement Time. ........................................................................................................ 91

Analysis of Individual Differences. ........................................................................... 92
Predictive modeling and typical motor development........................................112

Research on the development of predictive control using non-reaching paradigms..........................................................113

Summary. ........................................................................................................115

METHOD ........................................................................................................116

Participants.....................................................................................................116

Apparatus ......................................................................................................117

Procedure ......................................................................................................117

Design and Analysis ......................................................................................117

RESULTS .......................................................................................................119

Chronometric Analysis ..................................................................................119

Reaction Time. ..............................................................................................119

Movement Time............................................................................................120

Developmental trends. ..................................................................................121

MT\text{\_diff} scores across typical development..........................................122

Children with and without DCD. .................................................................123

Comparison of MT\text{\_diff} scores for children with and without DCD. ....123

Kinematic Analysis .......................................................................................123

Early control parameters............................................................................123

tPA and tPV..................................................................................................123

Developmental trends. ................................................................................124

Children with and without DCD. .................................................................124

TC..................................................................................................................125

Late control parameters: PCT.................................................................126

Error ..............................................................................................................127
Anticipatory errors ................................................................. 127
Centre touch errors ............................................................. 128
Touch down errors ............................................................... 128
Summary of results .............................................................. 128

DISCUSSION ........................................................................... 130

Chronometric Analysis .......................................................... 132

Reaction Time ........................................................................ 132
Condition effect ...................................................................... 132
Developmental trends ............................................................ 132
Children with and without DCD .............................................. 132

Movement Time ..................................................................... 133
Condition effect ...................................................................... 133
Developmental trends ............................................................ 133
Children with and without DCD .............................................. 134

Kinematic Analysis ................................................................. 136

Early control parameters ...................................................... 136
tPA and tPV ........................................................................... 136
TC .......................................................................................... 137
Developmental trends ............................................................ 137
Children with and without DCD .............................................. 139

Late control parameters ....................................................... 139
PCT ....................................................................................... 139
Developmental trends ............................................................ 139
Children with and without DCD .............................................. 140

Summary of kinematic analysis .............................................. 140
Error .............................................................................................................. 141

Anticipatory Errors ....................................................................................... 141

Centre touch error. .......................................................................................... 142

Touch down error. ........................................................................................... 143

General discussion .......................................................................................... 144

Atypical ROC (viz predictive modeling) in DCD reflects a developmental
immaturity. ......................................................................................................... 144

Limitations ........................................................................................................ 145

Summary ........................................................................................................... 146

CHAPTER 5

GENERAL DISCUSSION ..................................................................................... 148

GENERAL DISCUSSION ..................................................................................... 149

Summary ........................................................................................................... 149

Summary of Studies 1, 2 and 3 ........................................................................ 149

Study 1 .............................................................................................................. 149

Study 2 .............................................................................................................. 150

Study 3 .............................................................................................................. 151

Summary ........................................................................................................... 151

Implications for understanding ROC ............................................................... 152

ROC across typical development. ................................................................... 152

Impaired ROC in DCD: Implications for the IMD hypothesis...................... 153

Toward a unified account of DCD: the neural basis of impaired predictive
modeling ........................................................................................................... 158

Cerebellar contributions to predictive control ............................................... 160

Cerebellar contributions to control and learning .......................................... 161
List of Figures

<table>
<thead>
<tr>
<th>Figure</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>FIGURE 1.1. A GRAPHICAL REPRESENTATION OF A ‘NON- JUMP’ AND JUMP TRIAL</td>
<td>52</td>
</tr>
<tr>
<td>FIGURE 2.1. A GRAPHICAL REPRESENTATION OF THE DOUBLE-STEP REACH TASK PARAMETERS</td>
<td>67</td>
</tr>
<tr>
<td>FIGURE 2.2. MEAN REACTION TIME (RT +/- SE) FOR DCD AND CONTROL GROUPS</td>
<td>69</td>
</tr>
<tr>
<td>FIGURE 2.3. MEAN MOVEMENT TIME (MT +/- SE) FOR DCD AND CONTROL GROUPS</td>
<td>70</td>
</tr>
<tr>
<td>FIGURE 2.4. MEAN MT_{DIFF} SCORES FOR DCD AND CONTROL CHILDREN</td>
<td>71</td>
</tr>
<tr>
<td>FIGURE 2.5. MEAN NUMBER OF ERRORS (+/- SE) FOR EACH GROUP</td>
<td>72</td>
</tr>
<tr>
<td>FIGURE 3.1. A TYPICAL REACHING TRAJECTORY PLOT FROM A CONTROL AND DCD CHILD FOR NON-JUMP AND JUMP TRIALS</td>
<td>89</td>
</tr>
<tr>
<td>FIGURE 3.2. MEAN REACTION TIME (RT +/- SE) FOR DCD AND CONTROL GROUPS</td>
<td>91</td>
</tr>
<tr>
<td>FIGURE 3.3. MEAN MOVEMENT TIME (MT +/- SE) FOR DCD AND CONTROL GROUPS</td>
<td>92</td>
</tr>
<tr>
<td>FIGURE 3.4. MEAN MT_{DIFF} SCORES TRIALS FOR DCD AND CONTROL CHILDREN</td>
<td>93</td>
</tr>
<tr>
<td>FIGURE 3.5. MEAN TIME TO CORRECTION OF MOVEMENT TRAJECTORY FOR DCD AND CONTROL CHILDREN</td>
<td>94</td>
</tr>
<tr>
<td>FIGURE 4.1. MEAN GROUP REACTION TIMES (RT, +/- SE)</td>
<td>119</td>
</tr>
<tr>
<td>FIGURE 4.2. MEAN GROUP MOVEMENT TIME (MT, +/- SE)</td>
<td>120</td>
</tr>
<tr>
<td>FIGURE 4.3. INDIVIDUAL MEAN MT_{DIFF} SCORES FOR EACH GROUP</td>
<td>122</td>
</tr>
<tr>
<td>FIGURE 4.4. MEAN GROUP TIME TO CORRECTION OF MOVEMENT TRAJECTORY (TC, +/-SE)</td>
<td>126</td>
</tr>
<tr>
<td>FIGURE 4.5. MEAN GROUP AE AND CTE (+/- SE)</td>
<td>127</td>
</tr>
<tr>
<td>FIGURE 4.5. MEAN GROUP AE AND CTE (+/- SE)</td>
<td>127</td>
</tr>
</tbody>
</table>
List of Tables

<table>
<thead>
<tr>
<th>Table</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>TABLE 1.1. DIAGNOSTIC CRITERIA FOR DCD</td>
<td>11</td>
</tr>
<tr>
<td>TABLE 1.2. RESEARCH CRITERIA FOR SDDMF</td>
<td>15</td>
</tr>
<tr>
<td>TABLE 5.1. NEURAL REGIONS MORE ACTIVELY EMPLOYED BY DCD AND CONTROL CHILDREN RESPECTIVELY DURING A SIMPLE DRAWING TASK</td>
<td>164</td>
</tr>
</tbody>
</table>
## List of Acronyms

<table>
<thead>
<tr>
<th>Acronym</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>ABD</td>
<td>Atypical brain developmental</td>
</tr>
<tr>
<td>ADHD</td>
<td>Attention deficit hyperactivity disorder</td>
</tr>
<tr>
<td>AE</td>
<td>Anticipatory Errors</td>
</tr>
<tr>
<td>AMC</td>
<td>Age-matched control</td>
</tr>
<tr>
<td>Auto-D</td>
<td>Automatization deficit</td>
</tr>
<tr>
<td>CO-OP</td>
<td>Cognitive orientation to daily occupational performance</td>
</tr>
<tr>
<td>COVAT</td>
<td>Covert orienting of visual-spatial attention task</td>
</tr>
<tr>
<td>CP</td>
<td>Cerebral Palsy</td>
</tr>
<tr>
<td>CSAPPA</td>
<td>Children self-perception of adequacy in, and predilection for, physical activity</td>
</tr>
<tr>
<td>CTE</td>
<td>Centre touch errors</td>
</tr>
<tr>
<td>DAMP</td>
<td>Deficits in attention, motor control, and perception</td>
</tr>
<tr>
<td>DCD</td>
<td>Developmental coordination disorder</td>
</tr>
<tr>
<td>DSRP</td>
<td>Double-step reaching paradigm</td>
</tr>
<tr>
<td>DSST</td>
<td>Double-step saccade task</td>
</tr>
<tr>
<td>EEG</td>
<td>Electroencephalography</td>
</tr>
<tr>
<td>EMG</td>
<td>Electromyography</td>
</tr>
<tr>
<td>FOB</td>
<td>Flock of Birds</td>
</tr>
<tr>
<td>fMRI</td>
<td>Functional magnetic resonance imaging</td>
</tr>
<tr>
<td>GCM</td>
<td>Growth curve modeling</td>
</tr>
<tr>
<td>HREC</td>
<td>Human research ethics committee</td>
</tr>
<tr>
<td>IMD</td>
<td>Internal modeling deficit</td>
</tr>
<tr>
<td>IP</td>
<td>Information processing</td>
</tr>
<tr>
<td>LD</td>
<td>Learning disabilities</td>
</tr>
<tr>
<td>MABC</td>
<td>Movement assessment battery for children</td>
</tr>
<tr>
<td>MAND</td>
<td>McCarron assessment of neuromuscular development</td>
</tr>
<tr>
<td>MBD</td>
<td>Minimal brain dysfunction</td>
</tr>
<tr>
<td>MEP</td>
<td>Motor evoked potential</td>
</tr>
<tr>
<td>MI</td>
<td>Motor imagery</td>
</tr>
<tr>
<td>MT</td>
<td>Movement time</td>
</tr>
<tr>
<td>MT_{diff}</td>
<td>Movement time difference</td>
</tr>
<tr>
<td>PAT</td>
<td>Prism adaptation task</td>
</tr>
<tr>
<td>PCT</td>
<td>Post-correction time</td>
</tr>
<tr>
<td>PDD</td>
<td>Pervasive developmental disorder</td>
</tr>
<tr>
<td>PPC</td>
<td>Posterior parietal cortex</td>
</tr>
<tr>
<td>RD</td>
<td>Reading disorder/disability</td>
</tr>
<tr>
<td>ROC</td>
<td>Rapid online control</td>
</tr>
<tr>
<td>RT</td>
<td>Reaction time</td>
</tr>
<tr>
<td>SDDMF</td>
<td>Specific developmental disorder of motor function</td>
</tr>
<tr>
<td>SI</td>
<td>Sensory Integration</td>
</tr>
<tr>
<td>SLD</td>
<td>Specific language disorder</td>
</tr>
<tr>
<td>TC</td>
<td>Time to correction of movement trajectory</td>
</tr>
<tr>
<td>TDE</td>
<td>Touchdown errors</td>
</tr>
<tr>
<td>TMS</td>
<td>Transcranial magnetic stimulation</td>
</tr>
<tr>
<td>tPA</td>
<td>Time to peak acceleration</td>
</tr>
<tr>
<td>tPV</td>
<td>Time to peak velocity</td>
</tr>
<tr>
<td>Abbreviation</td>
<td>Description</td>
</tr>
<tr>
<td>--------------</td>
<td>---------------------------</td>
</tr>
<tr>
<td>VGPT</td>
<td>Visually guided pointing task</td>
</tr>
<tr>
<td>VI</td>
<td>Visual imagery</td>
</tr>
<tr>
<td>YC</td>
<td>Younger control</td>
</tr>
</tbody>
</table>
SUMMARY

Developmental coordination disorder (DCD) is characterized by impaired motor performance in the absence of any identifiable neurological or medical condition. Despite affecting roughly 6% of children, the aetiology of the disorder remains the source of some debate. In recent years, the cognitive neurosciences have provided an exciting new framework from which to investigate the cause/s of DCD. This field aims to identify the neuro-cognitive basis of function/dysfunction by mapping cognition and behavior to neural structures. The accounts to have emerged have been varied and interesting, most prominently, impaired motor timing related to the cerebellum (Castelnau, Albaret, Chaix, & Zanone, 2007; Williams, Woollacott, & Ivry, 1992); poor movement inhibition implicating fronto-striatal networks (Mandich, Buckolz, & Polatajko, 2002; Wilmut, Brown, & Wann, 2007), and, impaired predictive control, possibly implicating parieto-cerebellar networks [viz, the internal modeling deficit (IMD) hypothesis, Wilson and colleagues, 1999, 2004, 2006, 2008)]. Though each theory enjoys a degree of empirical validity, no unified account of the disorder has emerged. This disparity highlights the need for continued experimental work. One aspect of motor control that shows great promise in clarifying the nature of DCD is rapid online control (ROC) of reaching. Current computational accounts of online control provide a framework for investigating DCD and clarifying one of the more prominent cognitive neuroscientific accounts of the disorder- the IMD hypothesis. To date, however, the findings on ROC in DCD have been conflicting: some work suggests preserved online control (Plumb et al., 2008) while others suggest impairment (Wilmut, Brown, & Wann, 2006).
The aim of this thesis was to clarify the nature of online motor control in children with DCD using the well-validated double-step reaching paradigm (Dubrowski, Bock, Carnahan, & Jüngling, 2002; Farnè et al., 2003; Grèa et al., 2002). Here participants reach and touch one of three potential targets. For the majority of trials (80%), the target remains stationary for the duration of movement (non-jump); while for the remaining trials (20%) the target ‘jumps’ unexpectedly at movement onset thus requiring participants to correct their reaching ‘on the fly’. Initially interpreted using traditional dual-component models of motor control, more recent examples have adopted computational models as an interpretive framework. The former view movement as occurring in distinct predictive (forward) and feedback-based stages (Desmurget & Grafton, 2003), while the latter propose an integrated system, a view supported by current consensus on motor control (Desmurget & Grafton, 2000; Desmurget & Sirigu, 2009; Shadmehr & Krakauer, 2008) and empirical evidence (Saunders & Knill, 2003; 2005). In accordance with such modeling, the working assumption of this thesis is that ROC is only viable to the extent that predictive (forward) and feedback models can be integrated seamlessly. The following thesis consists of three studies exploring rapid online motor control in DCD, each investigating various aspects of performance on the double-step reaching task. In Study 1, chronometric analysis of double-step reaching was conducted to determine the integrity of ROC in children with DCD. No differences between DCD and control groups were evident for simple (non-jump) reaching yet the performance of children with DCD was more affected by unexpected target perturbation at movement onset than controls, manifest by increased movement time and error. Interpreted from a computational perspective, I argue that this decreased capacity for
accounting for unexpected target perturbation may reflect a decreased capacity for correcting predictive models of movement. The demonstrated interaction effect for movement time stands in contrast to previous work investigating double-step reaching in DCD which showed slower performance overall but no differential effect for target condition (Plumb et al., 2008). I argue that key methodological and theoretical limitations limit the reliability of group comparison in Plumb and colleagues experiment. For example, DCD and control groups performed different versions of the double-step task and performance was interpreted using the superseded dual-component approach. By controlling task parameters across groups and using computational models as an interpretive framework, I propose that Study 1 offers a more valid account of ROC in DCD. These results suggested impaired ROC in children with DCD that may be attributable to poor predictive control.

While chronometric analysis provides important information about the integrity of ROC at a global level, it does not inform the subtle transitions in control that support the un-folding movement. Importantly, I was unable to determine whether the double-step reaching of children with DCD was affected early in the reaching cycle or later. For double-step reaching, demands on predictive modeling are greatest early in the reaching cycle where large-scale trajectory corrections are implemented. Alternatively, demands on predictive modeling are comparatively minimal following these changes since the target remains stationary for the remainder of movement. Hence, in order to better identify the motor control properties responsible for the slower, less accurate double-step reaching shown by children with DCD, Study 2 involved both kinematic and chronometric analysis of double-step reaching.
In Study 2, the pattern of performance on chronometric variables shown in Study 1 was replicated. Importantly, early kinematic markers (i.e. time to peak acceleration and velocity) failed to discriminate between groups. However, children with DCD took significantly longer to correct the trajectory of their reaching towards the new target on jump trials. Time to correction is thought to reflect the stage in reaching when error information which arises following a mismatch between the expected (according to the predictive model) and actual sensory consequences of reaching has been successfully integrated with the on-going motor command. By comparison, the post correction phase did not differ between groups suggesting that once trajectory corrections had been implemented, children with DCD were able to complete movement as efficiently as their same-age peers (i.e. when demands on predictive modeling were minimal). In summation, this pattern of results was consistent with the view that children with DCD experience difficulty correcting predictive models of movement online; specifically, they may have difficulty using error information to rapidly correct the on-going motor command.

What remained unclear from Studies 1 and 2 was whether the atypical pattern of performance (and apparent deficit in predictive modeling) reflects a developmental immaturity or deviance from the typical developmental path. Study 3 was designed to clarify this issue. Here, double-step reaching in older children with DCD (8 to 12 years of age) was compared with both a group of age-matched controls and younger control children (5 to 7 years of age). A group of young adults were also assessed to provide a benchmark for mature motor function. These comparison groups enabled me to interpret the performance of children with DCD relative to a normal developmental continuum. Should the performance of children with DCD mirror that
of younger neurologically immature control children, developmental immaturity would be inferred. A pattern of responding in DCD that did not fit along the normal “continuum” would indicate developmental deviance. Importantly, chronometric analysis revealed that the movement time of children with DCD and younger controls was equally affected by target perturbation, while kinematic analysis showed overlap in their early kinematic profiles specifically time to trajectory correction on jump trials. This supports the view that difficulties in ROC (and predictive modeling) in DCD may reflect a developmental immaturity.

In Chapter 5, the implications of findings from the three studies are discussed in relation to ROC in children and the quest to forge a unified account of DCD. I argue that this group of studies makes a compelling case that ROC is impaired in children with DCD. Interpreted from a computational perspective, this impairment appears to reflect a disruption to predictive control, specifically the ability to use the error signal which arises when there is disparity between the expected (according to the predictive model) and actual sensory consequences of movement to adjust movements in-flight. Study 3 showed that these difficulties are likely to reflect developmental immaturity possibly at the level of the posterior parietal cortex (PPC) or cerebellum. Importantly, these results clarify some of the inconsistencies in the literature on ROC in clumsy children. I also discuss the contribution that this thesis makes to the validation of the IMD account of DCD, one of the more promising neuro-cognitive accounts of DCD currently available. Clinical implications of this research and directions for future research are then discussed.
CHAPTER 1

AN OVERVIEW OF RESEARCH INTO DCD
Introduction

Overview of Chapter 1

In Chapter 1, a review of research investigating Developmental Coordination Disorder (DCD) is provided: a brief history of DCD as a diagnostic entity is discussed, followed by diagnostic criteria and key clinical features. A review of the main categories of investigation is then provided. An important theme that arises in the developmental literature is the need for a unifying aetiological account of DCD to form the basis of effective remediation. A brief narrative review of different theoretical accounts of DCD will show that the cognitive neurosciences offer a promising framework for developing a unified aetiological account of DCD. Several accounts have emerged, including impaired movement timing associated with the cerebellum (Castelnau et al., 2007; Lundy-Ekman, Ivry, Keele, & Woollacott, 1992; Williams et al., 1992); disinhibition of movement initiation implicating fronto-striatal circuitry (Mandich et al., 2002; Mandich, Buckolz, & Polatajko, 2003; Wilson & Maruff, 1999); and impaired predictive control (viz internal modeling deficit; Williams, Thomas, Maruff, Butson, & Wilson, 2006; Williams, Thomas, Maruff, & Wilson, 2008; Wilson, Maruff, Butson, Lum, & Thomas, 2004). It is shown that while each enjoys a degree of empirical validity, no one theory has provided a unifying account of the disorder. This disparity highlights the importance of continued experimental work to clarify the issue.

One area that holds promise in clarifying the aetiology of DCD is rapid online control (ROC), an aspect of movement crucial to mature fluid action. In recent decades, double-step reaching paradigms have been the cornerstone of the investigation into ROC, however their interpretation has been varied, usually based on
superseded theories of motor control (i.e. dual-component models). As a result, data on DCD has been conflicting. While some recent work using double-step reaching suggests no difficulty in ROC (e.g., Plumb et al., 2008), others suggest deficits (based on sequential pointing, for example; Wilmut et al., 2006); this work is reviewed. To help resolve this debate, it is proposed that current cognitive neuroscience models be used as a framework for investigating double-step reaching performance in DCD and a more controlled study of double-step reaching be employed. It is argued that investigated using this framework, the area of ROC shows great potential in addressing inconsistent lines of evidence surrounding one of the more prominent cognitive neuroscientific account of DCD- the IMD hypothesis. These issues are addressed in the studies presented in Chapters 2, 3 and 4 which aim to clarify the nature of ROC in children with DCD.

Developmental Coordination Disorder: Diagnosis, Presentation, and Associated Problems

Over the years, there has been considerable conjecture surrounding the appropriateness of diagnostic labelling for impaired motor development. This has hindered cross-research comparison and the establishment of a sound understanding of the disorder. Important developments in recent decades have seen greater continuity in terms and diagnostic criteria used across studies. The following section compares and critiques the two dominant systems for identifying motor impairment in children; the World Health Organization’s (WHO) International Classification System (ICD-10) and the American Psychiatric Association’s (APA) Diagnostic and Statistical manual of Mental Disorder (DSM). Despite some limitations, in accordance with international consensus statements (see Sugden, 2006), I argue for
the use of the DSM system and label- Developmental Coordination Disorder (DCD). Symptom presentation and associated issues are then outlined.

**Systems for identifying and diagnosing motor impairment in children**

Impairment in the ability to acquire movement skills in children, in isolation of any detectable neurological or medical condition, has been extensively reported in the developmental literature since the turn of last century (Orton, 1937; Walton, Ellis, & Court, 1962). Throughout this period, a number of terms have been used to describe this “syndrome”: ‘developmental dyspraxia’ (Cermak, 1985; Dewey, 1995), ‘clumsiness’ (Gubbay 1975; Henderson & Hall, 1982; Losse et al., 1991), ‘Minimal Brain Damage’ (Forssström & van Hofsten, 1982), ‘physically awkward’ (Marchiori, Wall, & Bedingfield, 1987) and ‘perceptuo-motor dysfunction’ (Laszlo, Bairstow, & Bartrip, 1988). For a review see Henderson and Barnett (1998) and Geuze, Jongmans, Schoemaker, and Smits-Engelsman, (2001). Historically, the lack of a unifying label for the disorder has compromised comparison of research across studies and the development of a sound knowledge base of the disorder. In 1994, following a consensus meeting of the world’s leading clinicians and researchers it was decided that the term ‘Developmental Coordination Disorder’ (DCD) should be applied (Polatajko, Fox, & Missiuna, 1995). This term has been widely adopted, although there continues to be some variation in the terms used; a recent review showing that just above half of 319 research papers reviewed adopted the term ‘DCD’ (Magalhães, Missiuna, & Wong, 2006).

The late 1980’s and early 90’s saw a number of seminal longitudinal studies emerge illustrating that the movement difficulties experienced by *clumsy* children were often pervasive and tended not to dissipate with age. In 1987, this research
culminated in acknowledgement by the American Psychological Association (APA) that the disorder was a distinct diagnostic entity- i.e. in the Diagnostic and Statistical manual of Mental Disorder III-R (DSM-III-R; APA, 1987). This preliminary identification was followed by entries for developmental motor impairment in the WHO’s *International Classification System* (ICD-10) in 1992 and the APA’s subsequent edition of the *Diagnostic and Statistical manual of Mental Disorder* (the DSM-IV) in 1994. These were labeled Specific Developmental Disorder of Motor Function (SDDMF) and Developmental Coordination Disorder respectively. Overlap and distinctions between the two schemes are described below.

**DSM-IV criteria for DCD.** The DSM-IV (APA, 1994) states that the defining feature of DCD is “a marked impairment in the development of motor coordination” (pg. 56). Four criterion, two of which are inclusionary (Criterion A and B) and two exclusionary (Criterion C and D), are outlined (Table 1.1). As demonstrated below, these criteria have been the subject of rigorous debate because of the level of interpretation involved in their application (see Geuze, et al., 2001 for a good review). Although the Leeds international censuses statement of 2006 (Sugden, 2006) addressed a number of these concerns, the literature continues to debate the merit of the DSM-IV diagnostic criteria and the Leeds consensus statement recommendations (Barnett, 2008; Peters & Henderson, 2008; Sugden, Kirby, & Dunford, 2008). A short overview of these issues is provided below:
Table 1.1. Diagnostic Criteria for DCD (APA, 2000, pg 58)

A. Performance in daily activities that require motor coordination is substantially below that expected given the person’s chronological age and measured intelligence. This may be manifested by marked delays in achieving motor milestones (e.g., walking, crawling, and sitting), dropping things, “clumsiness”, poor performance in sports, or poor handwriting.

B. The disturbance in Criterion A significantly interferes with academic achievement or activities of daily living.

C. The disturbance is not due to a general medical condition (e.g., cerebral palsy, hemiplegia, or muscular dystrophy) and does not meet criteria for a Pervasive Developmental Disorder.

D. If Mental Retardation is present, the motor difficulties are in excess of those usually associated with it.

Criterion A states that an individual must demonstrate difficulties conducting daily activities that require movement. Difficulties operationalizing the term ‘daily activities’ have led to varied interpretation of this criteria (Henderson & Barnett, 1998). The 2006 Leeds censuses statement did little to address this issue, providing only a general overview of the core features of the condition (Sugden, 2006). Additionally, no “gold standard” measure of motor control analogous to that of Wechsler’s’ IQ measures of the cognitive domain exists to gauge motor competence (Henderson & Barnett, 1998) and no quantitative cut-off for diagnosis is stipulated, further contributing to the range of applications and interpretations of Criteria A.

Criterion B states that motor impairment must significantly interfere with academic achievement and/or daily living activities. Again, the DSM-IV does little to aide clinicians’ and researchers in operationalizing the terms ‘academic achievement’ or ‘daily living skills’ (Chambers, Sugden, & Sinani, 2005). Although the Leeds consensus statement suggests a number of general activities to which this criteria should be applied [including self-care, leisure and schoolwork (i.e. handwriting, PE
and tools use), there continues to be variation in the application of Criteria B.

Criterion C, the first of the exclusionary criterion, states that motor problems must not be due to a general medical condition or pervasive developmental disorder (PDD). Case studies of children suffering from motor impairment often report difficulties associated with applying this criteria when the source of the motor impairment is unclear. For example, Peters and Henderson (2008) report the case of ‘Kevin’ who displayed the soft neurological signs symptomatic of both mild cerebral palsy (CP) and severe DCD. While in many cases the cause of motor impairment may be clear (i.e. muscular dystrophy, severe CP or cerebral lesioning) for ‘borderline’ cases such as Kevin’s, applying Criteria C presents as a greater challenge.

Finally, Criterion D states that if intellectual disability is present, motor impairment must be in excess of that usually associated with it. Yet, there is still no clear evidence, either for against, the relationship between intellectual functioning and motor development (Henderson & Barnett, 1998; Sadock, Sadock, & Levin, 2007). This has led to differing application of this criteria depending on the degree of acceptance of the link between IQ and motor competence. Some suggest that motor impairment increases markedly once IQ falls below 70 and as such diagnosis of DCD should not be given when IQ drops below this level (Sugden, 2006). Others suggest that DCD and intellectual disability should be considered as discrete, yet potentially co-morbid, disorders (Geuze et al., 2001).

**Applying the DSM-IV Criteria in Research Settings.** Since official recognition of its existence in 1987, there has been a surge in research investigating various aspects of DCD. As is the case in clinical settings, there has been great variability in how the diagnostic criteria and terminology have been applied in the research literature (Geuze et al., 2001; Magälhaes et al., 2006). For example,
psychometric cut-offs vary between the 5th to 15th percentile when standardized motor screening tools are used to identify motor impairment, consistent with Criteria A. Similarly, a recent review of the DCD literature found that 40% of studies did not specify how the DMS-IV exclusionary criteria (i.e. Criterion C and D) were applied (Geuze et al., 2001). These issues have compromised comparison and review of related articles.

**Conclusion.** Given ambiguities in the DSM-IV diagnostic criteria for DCD and cultural differences across disciplines and internationally, it seems unlikely that the issues surrounding inconsistent application of DSM-IV diagnostic criteria will be resolved in the near future. For example, when applying Criteria A, the McCarron Assessment of Neuromuscular Development (MAND) (McCarron, 1997) is frequently used as a standardized measure of motor competence in Australia, while the Movement Assessment Battery for Children (MABC; Henderson & Sugden, 1992; Henderson, Sugden & Barnett, 2007) tends to be adopted in Europe. The reasoning underlying these differences appears to be fairly arbitrary. Hence, as is argued elsewhere (Geuze et al., 2001), it is important that authors and clinicians report and justify their inclusionary and exclusionary diagnostic criteria clearly to allow reliable cross-laboratory comparison and inter-clinician communication.

**ICD system for diagnosing motor impairment.** The ICD-10 (WHO, 1992, F82) states that the principle feature of Specific Developmental Disorder of Motor Function (SDDMF) is “a serious impairment in the development of motor coordination”. Diagnosis of SDDMF is reserved for those children:
“whose motor coordination on fine or gross motor tasks is significantly below that expected on the basis of his or her age and general intelligence. This is best assessed on the basis of an individually administered standardized tests of fine and gross motor coordination. The difficulties in coordination should have been present since early in development (i.e. they should not constitute an acquired deficit). It is usual for the motor clumsiness to be associated with some degree of impaired performance on visuospatial cognitive tasks.”

A year later, four additional criterion were added to guide the application of the above criteria in research settings- these are outlined in Table 1.2. Debate surrounding the ICD-10 diagnostic criteria for SDDMF has been less rigorous than for the DSM-IV criteria for DCD. The most likely reason for this is that 'SDDMF' label is used far less frequently than 'DCD' in research and clinical settings, particularly outside of Europe. This is no doubt at least partly attributable to international consensus statements suggesting the use of the DSM-IV system for diagnosing developmental motor impairment (Polatajko et al. 1995; Sugden, 2006) (discussed below). Nonetheless there are a number of similarities between the DSM-IV and ICD-10 classification systems and consequently similar issues surround their application. These are outlined next.
Table 1.2. Research criteria for SDDMF (WHO, 1993, pg. 179)

| A. | A score on a standardized test of fine or gross motor coordination that is at least two standard deviations below the level expected for the child's chronological age. |
| B. | The disturbance in A significantly interferes with academic achievement or activities of daily living. |
| C. | No diagnosable neurological disorder. |
| D. | Most commonly used exclusion criterion: IQ below 70 on an individually administered standardized test. |

In accordance with the WHO’s suggestion that a child’s motor difficulties must be significantly below that expected for their age, Criterion A of the research criteria (above) states that motor impairment must be at least two standard deviations below that expected according to a child’s chronological age. Similarly to the DSM-IV criteria, no standardized measure of motor competence is proposed to assess skill level. In contrast to the DSM-IV criteria, however, the ICD-10 criteria does suggest a quantitative cut-off- at least two standard deviations below the mean- though no justification is provided for why this cut-off is deemed appropriate.

Further, Criterion B of the research criteria states that motor impairment must impact on academic achievement or activities of daily living. Similarly to the DSM-IV criteria, no efforts are made to operationalize the constructs ‘academic achievement’ or ‘activities of daily living’ allowing for a variety of interpretations. However, where consensus statements have provided some additional guidance for the corresponding DSM-IV criterion, no such guidance is available for applying Criterion B for SDDMF.

Criterion C states that motor impairment must be in isolation of a diagnosable neurological disorder. This criterion is again similar to Criterion C of the DSM-IV
diagnostic system which indicates that motor impairment must be in isolation of medical condition or PDD. Consequently, similarly to the DSM-IV, difficulties arise in the application of this criteria when the cause/s of motor impairment are not entirely clear- for example, in cases where motor difficulties might reflect mild neurological impairment (viz. mild CP) or severe DCD.

Finally, Criteria D implies that motor impairment must occur in isolation of intellectual deficit. As noted above, the link between motor impairment and intellectual disability remains contentious. The decision to exclude a diagnosis of SDDMF on the basis of intellectual disability assumes an acceptance of this link and is at odds with DSM-IV criteria which instead states that in the case of intellectual disability motor impairment must be beyond that usually associated with it. Until stronger evidence exists either supporting or refuting the link between motor skill and intellectual abilities, the DSM-IV criteria arguably provides a better middle ground to work with on the issue.

**Overview of the DSM-IV and ICD-10 systems.** The diagnostic criteria for DCD and Specific Developmental Disorder of Motor Function are largely analogous; they both acknowledge motor impairment as the requisite feature of the disorder, it’s impact on academic functioning and everyday living and excludes diagnosis on the basis of neurological and (to a degree) intellectual impairment. However, international consensus statements have recommended that the APA’s DSM-IV terminology and diagnostic criteria be applied (Polatajko et al., 1995; Sugden, 2006). Though, as previously eluded to, terminology used in reference to developmental motor difficulties continues to vary, it would appear that this recommendation has been largely adhered to, with a recent review finding that not one of 176 publications reviewed used the ICD-10 terminology (Geuze et al. 2001).
The term DCD has also been adopted by a bi-annual international conference on the disorder since 1995. Accordingly, the following thesis adopted the DSM-IV classification for DCD.

**Presentation of the Disorder**

Clumsiness (viz Developmental coordination disorder- DCD) affects roughly 6% of children between the ages of 5 and 11 years and is characterized by an overall impairment in fundamental motor skills. Impairment presents in a variety of gross and/or fine motor tasks such as walking, running, balancing, standing, jumping, reaching, writing and/or grasping (Miller, Missiuna, Macnab, Malloy-Miller, & Polatajko, 2001; Missiuna et al., 2008a). While children often demonstrate a degree of competence when performing these tasks, they experience difficulty performing them to a functional standard (Sugden & Chamber, 2003). Consequently, they are unable to acquire, refine and conduct their movements at age appropriate levels and many seemingly trivial activities become the source of much frustration and anguish. Similarly to other neuro-developmental disorders, incidence in males outnumbers females approximately 2 to 1 (Missiuna, Gaines, Soucie, & McLean, 2006a). The debate as to whether this reflects actual gender differences in prevalence or is the result of the stereotyped view that males tend to undertake activities where motor impairment is more likely to be noticed remains unresolved.

Symptom expression is heterogeneous and as such the presentation and severity of symptoms vary considerably between cases and throughout development, often complicating diagnosis and intervention planning. For example, where a child may present with postural control difficulties, another may present with handwriting issues ranging from messy to illegible handwriting (Missiuna, Rivard, & Pollock,
2004). Further, these difficulties may persist (or exacerbate) into adolescence and adulthood or dissipate with time (Sugden, 2006). DCD is an idiopathic disorder and despite efforts to develop measures of early identification (for example, the Children Self-Perception of Adequacy in, and Predilection for, Physical Activity, CSAPPA-Hay, Hawes, & Faught, 2004) it is typically not diagnosed before 5 years of age. DCD presents irrespective of race, culture or socio-economic status (APA, 2000; Sugden, 2006), though incidence tends to be higher in children of low socio-economic background (Lingam, Hunt, Golding, Jongmans, & Emond, 2009). As is outlined next, identification and treatment of DCD is also complicated by the fact that the negative impact of DCD is not confined to motor performance, commonly being linked with poorer social, mental health and educational outcomes in comparison to same-age children.

**Secondary Social, Psychological, and Educational Issues.** The co-occurrence of motor impairment and secondary mental health issues has been commonly reported in the literature, especially in the past decade: poor psychosocial functioning (Cummins, Piek, & Dyck, 2005; Dewey, Kaplan, Crawford, & Wilson, 2002; Kristensen & Torgersen, 2008), risk of obesity (Cairney, Hay, Faught, & Hawes, 2005), anxiety and affective disorders (Kristensen & Torgersen, 2008; Moruzzi et al., 2010; Piek et al., 2007b), risk of coronary vascular disease (Faught, Hay, Cairney, & Flouris, 2005) and personality disorders, alcohol abuse and criminal offending (Rasmussen & Gillberg, 2000) have all been associated with impaired movement, though the relationship between motor impairment and these secondary disorders is generally though to be indirect (Cummins et al., 2005). By way of illustration, children with DCD often report poorer self-perceptions of their motor capabilities and subsequently tend to prefer a sedentary lifestyle (Cairney et al., 2005;
Misiuna et al., 2008a; Wocadlo & Rieger, 2008) which has been linked to increased loneliness (Poulsen, Ziviani, Cuskelly, & Smith, 2007), risk of obesity (Cairney et al., 2005) and risk of coronary vascular disease (Faught et al., 2005).

A number of authors have also highlighted the association between motor impairment and poorer educational performance and outcomes (Rasmussen & Gillberg, 2000; Wocadlo & Rieger, 2008). While research suggests lower perceptions of scholastic competence in DCD compared to same-age peers (Watson & Knott, 2006), others have shown that affected children experience difficulties with reading comprehension and basic reading skills compared to control children (Dewey et al., 2002). While these effects may also be considered as indirect, Miller and colleagues (2001) demonstrated that between 71 and 90% of children identified as exhibiting DCD symptoms experienced handwriting and written communication difficulties highlighting the direct impact that atypical motor development may have on academic performance.

Research into DCD

As noted above, research into the different aspects of DCD has increased considerably in recent years. Depending on the purpose of the research, these studies can be broadly categorized into one of three approaches (excluding review papers): descriptive studies, aimed at describing key features of the disorder, it’s associated problems and common co-morbidities; intervention studies, which investigate the effectiveness of remedial methods; and aetiological studies, which aim to establish the causal basis of impaired movement. These approaches are discussed next.

Descriptive Research. The purpose of descriptive research is to describe the key attributes of DCD. This research is not concerned with the ‘why’ or
‘how’ of the disorder, but rather the ‘what’. Descriptive studies investigate a variety of characteristics of the disorder including its general presentation, prevalence and prognosis (Losse et al., 1991; Miller et al., 2001; Missiuna et al., 2008b; Missiuna, Moll, King, King, & Law, 2007; Rasmussen & Gillberg, 2000). Others have explored the impact of motor impairment on daily living (Peters & Henderson, 2008; Summers, Larkin, & Dewey, 2008), academic performance (Dewey et al., 2002; Wocadlo & Rieger, 2008), immediate and long-term health (Cairney et al., 2005; Hands, 2008), psychosocial functioning (Cummins et al., 2005; Dewey et al., 2002; Missiuna et al., 2007; Rodger et al., 2003), the family of those affected (Missiuna, Moll, Law, King, & King, 2006b) and participation in activities (Engel-Yeger & Kasis, 2010). Also, researchers have focused on describing the common co-occurrence of DCD and developmental disorders such as attention deficit hyperactivity disorder (ADHD) and reading and learning difficulties (Dewey et al., 2002; Kaplan, Wilson, Dewey, & Crawford, 1998; Martin, Piek, & Hay, 2006) while others have tried to establish the existence of sub-types of the disorder (Hoare, 1994; Macnab, Miller, & Polatajko, 2001; Tsai, Wilson, & Wu, 2008). This research has provided clinicians with important insight into the motor (and associated psycho-social) difficulties commonly faced by sufferers of the disorder, it’s highly heterogeneous nature and the knowledge that, if untreated, motor impairment frequently persists into adolescence and adulthood (Cantell, Smyth, & Ahonen, 2003; Losse et al., 1991; Missiuna et al., 2007). Furthermore, it is now understood that co-occurrence of DCD and other developmental disorders (i.e. ADHD, dyslexia etc.) is the rule rather than the exception (Kaplan et al., 1998). This knowledge has formed the basis of aetiological accounts of DCD and guided intervention development.
**Intervention Studies.** The range of available causal accounts of DCD is reflected in the variety of approaches to intervention. Despite this variety, efforts to develop effective remediation have thus far failed to provide clinicians with a universally preferred or effective program. Intervention methods can be broadly categorized as one of two general types: *process-oriented* approaches and *task oriented* approaches (for good reviews see Sugden, 2007; Wilson, 2005): these are discussed next.

Process-oriented approaches are based on information processing accounts of action (discussed in greater detail below) and neuromaturational models of motor control and development. These presume that motor impairment is the result of deficit or impairment at the level of putative processes or functions thought to be essential to age-appropriate movement (i.e. sensorimotor integration, visuo-spatial processing, memory etc.). Intervention is based on the assumption that remediation of these underlying deficits will result in improved motor function. Examples of these types of intervention include sensory integration therapy (SI) and perceptuo-motor approaches. As a general rule, these approaches have failed to generate support. In a recent meta-analysis of 13 intervention studies, Pless and Carlsson (2000) found little empirical evidence for the efficacy of SI methods compared to top-down (task-oriented) approaches. They attributed this lack of effect to difficulties motor impaired children experience generalizing improvement in one motor domain to other (similar) actions. Similarly, a review conducted by Mandich, Polatajko, Macnab, and Miller (2001) reported indifferent results for SI and process-oriented approaches with both being supported initially by key studies reporting positive outcomes yet subsequent investigations largely failing to differentiate their effects from other intervention methods such as tutoring, physical education, perceptual motor training and even no
treatment. The same meta-analysis failed to find support for the efficacy of perceptuo-motor training.

More recently, task-oriented approaches have emerged which emphasize ameliorating the difficulties that children experience with specific tasks (e.g. handwriting). In accordance with contemporary accounts of motor control and learning, they emphasize the role of the interaction between the child, task and environment in skill acquisition. Intervention focuses on developing cognitive or problem-solving skills so that individuals are able to employ the most appropriate strategy to complete a given task (Barnhart, Davenport, Epps, & Nordquist, 2003). Examples include task-specific strategies and cognitive methods such as the Cognitive Orientation to Daily Occupational Performance (CO-OP). While initial investigation has provided promising support for the CO-OP approach (Miller, Polatajko, Missiuna, Mandich, & Macnab, 2001; Sangster, Beninger, Polatajko, & Mandich, 2005), validation of such top-down approaches to intervention has relied on a limited number of studies with small sample sizes. Notwithstanding, they are supported by contemporary approaches to skill acquisition and carry with them an ecological validity beyond that offered by process-oriented approaches.

**Conclusion.** Despite the availability of a number of intervention approaches, no single method has proven to be universally effective. What is clear, however, is that intervention is better than no intervention. Though initial evidence is supportive of task-oriented approaches to intervention, some review papers have also failed to differentiate them from process-oriented models (Mandich et al., 2001; Hillier, 2007). Rather than one approach being more effective than the other, it may be that common features account for treatment effects (Hillier, 2007). For example, positive feedback, parent involvement in treatment planning and delivery, education of those involved in
caring for the motor impaired child and personalized treatment planning are some generic themes which have been implicated in positive intervention outcomes (Hillier, 2007). Given the likely heterogeneity of DCD, it may also be the case that sub-types of DCD respond best to different treatment approaches. If it is indeed the case that sub-types of DCD exist, for which there is a growing body of supportive evidence (Hoare, 1994; Macnab et al. 2001; Tsai et al., 2008), it is likely that the aetiology of each sub-type differs, if only subtly, and that they might respond differently to targeted intervention. This would explain why, as will be discussed below, no comprehensive aetiological account or intervention strategy exists for DCD. It also highlights the importance of investigation into the casual basis of DCD to the development of effective intervention. Accordingly, research investigating the aetiology of DCD is discussed next.

**Aetiological Accounts of DCD**

Theoretical and technological advances have seen causal accounts of atypical motor development (i.e. DCD) evolve considerably in the last two to three decades. Aetiological research attempts to identify the underlying cause/s of the disorder. The following section describes a number of the more prominent theories to have emerged from this research.

Initially, clumsiness in children was thought to result from a general maturational delay from which children would spontaneously recover (Geuze & Börger, 1993). This model was later challenged by those proposing a generalized non-specific deficit at a neurological level (Gillberg, 2003; Kaplan, Crawford, Cantell, Kooistra, & Dewey, 2006; Visser, 2003). Though once prominent, these theories have since given way to information processing accounts (Wilson & McKenzie, 1998), and
more recently, cognitive neuroscientific models of DCD (Castelnau et al., 2007; Mandich et al., 2003; Williams et al., 2006; 2008). The latter approach concerns itself with determining the perceptuo-motor mechanisms subserving DCD and identifying the neural locus of function/dysfunction. As will be argued, this approach provides the most promising framework for developing a unified causal model of DCD. These theories are discussed chronologically below.

**General Maturational Delay.** Initially clinicians considered the motor deficits experienced by children with DCD to be transitory, something that the child would ‘grow out of’ (Geuze & Börger, 1993). Since then, a number of longitudinal studies have illustrated that symptoms often do not dissipate in time, resulting in pervasive motor and psychosocial difficulties. In their seminal study, Losse et al., (1991) showed that motor impairment persists well into teenage years. Conducting a follow-up study of 16 children aged 15 to 17 years of age who at between five and seven years had been identified as suffering from DCD, they found that over 85% still experienced coordination difficulties, as well as secondary academic and psychosocial difficulties. Though their sample size was modest, these effects have since been replicated in more recent work. Cantell, Smyth and Ahonen (1994) re-assessed 15 year olds who had been identified as experiencing delayed motor development at 5 years of age and showed that 47% of the original sample had persistent motor problems, described as the ‘DCD group’; whereas 53% had minor movement difficulties, described as the ‘intermediate group’. While the motor performance of the ‘DCD group’ was poorer than both the ‘intermediate’ and ‘control’ groups, the performance of the latter two groups were similar. These results were largely replicated in a follow up of the same children at age 17 and 18 years (Cantell et al., 2003) (see also Cousins & Smyth, 2003; Missiuna et al., 2007). Thus, there is
compelling evidence that a substantial proportion of children do not outgrow their movement difficulties, a factor which may be mediated by the initial severity of impairment. How these difficulties manifest in later adulthood is less well understood. Nonetheless, these findings do not support the general maturational delay account of DCD.

**Neurological Impairment.** Motor coordination difficulties frequently co-occur with developmental disorders such as ADHD, ADD and Learning Disorders (Kaplan et al., 2006). Similarities between symptoms of these disorders and those caused by cerebral damage (i.e. mild tremor, motor impairment, attention deficits, hyperactivity and learning difficulties) have in the past led to the view that they may reflect a form of mild brain damage (Kaplan et al., 2006; Visser, 2003). Furthermore, the common co-occurrence of these disorders led to the proposition that rather than reflecting separable disorders, they may be symptoms of a generalized syndrome, initially labeled as Minimal Brain Dysfunction (MBD). The MBD hypothesis gained prominence in the 1960’s, however, in light of a lack of supportive evidence, clinicians and researchers questioned the validity of the assumption that the disorder resulted from brain damage or impairment, and the popularity of the view began to wane in the 1980s and early 1990s (Gilger & Kaplan, 2001; Green & Baird, 2005). Nonetheless, the core principle of the MBD (namely, that a group of developmental disorders are best conceptualized as a single condition resulting from non-specific widespread neural impairment) is clearly evident in a number of contemporary accounts of DCD. These include the theories of Atypical Brain Developmental (ABD) (Gilger & Kaplan, 2001; Kaplan et al., 1998), deficits in attention, motor control, and perception (DAMP) (Gillberg, 2003) and the Automatization deficit (Auto-D) hypothesis (Visser, 2003) which are discussed next.
Atypical Brain Development (ABD) Theory. The ABD theory was initially proposed by Kaplan and colleagues (1998) to explain the common co-occurrence of ADHD, reading disorder (RD) and DCD. The authors investigated children who had been referred for attentional and motor problems and found that of 115 children who met the criteria for one of the disorders of interest, only 53 were identified as ‘pure’ cases: DCD (n=23), RD (n= 19) and ADHD (n= 8). Of the remaining 62 co-morbid cases, 23 met the criteria for all three disorders. The authors then proposed a common underlying cause. They argued that neuroimaging and neuropsychological evidence did not support a one-to-one relationship between cortical regions and symptoms of developmental disorders, rather reflecting a more general pattern of atypical function. Hence, the ABD theory proposed that these frequently co-occurring symptoms resulted from diffuse, rather than localized, brain dysfunction.

Like the MBD hypothesis, however, the idea of non-specific diffuse damage is vague; causal mechanisms that could be a focus for intervention are difficult to identify. Further, technological and theoretical advances over the decade since Kaplan and colleagues formative study have provided more reliable evidence of the relationship between neural pathways and the symptoms of specific development disorders. Though there is a degree of overlap between the suggested neural regions subserving these disorders, this work supports the idea of dissociable disorders. For example, there is now strong evidence for a role of frontal-striatal-cerebellar circuits in ADHD (for a review see Krain & Castellanos, 2006), while specific movement deficits have been consistently linked to neural regions using neuropsychological evidence; movement timing impairment has been associated with the cerebellum (Castelnau et al., 2007; Williams et al., 1992), and compromised predictive control of
movement with the posterior parietal cortex (PPC) and cerebellum (Kagerer, Contreras-Vidal, Bo, & Clark, 2006; Katschmarsky et al., 2001; Wilson et al., 2004; Williams et al., 2006).

**Deficits in Attention, Motor Control and Perception (DAMP) Theory.** In a series of longitudinal follow-up studies in Sweden in the early 1980’s, Gillberg and colleagues highlighted the frequent co-morbidity of developmental disorders (for a good review, see Gillberg, 2003). To account for this overlap, Gillberg coined the term, *deficits in attention, motor control and perception* (DAMP). Like MBD, the DAMP theory proposed that rather than viewing the symptoms of what are now recognized as developmental disorders (i.e. DCD, ADHD, Dyslexia etc.) as discrete it is more useful to conceptualize them as a unitary disorder subserved by a generalized deficit. Like MBD, however, the nature and source of this impairment is unclear (Visser, 2003) and thus its usefulness in providing a focus for intervention methods for movement difficulties is limited.

**Automatization Deficit Hypothesis.** Initially, the automotization deficit (Auto-D) hypothesis proposed that the motor deficits frequently linked with dyslexia may result from a deficit in the ability to automatize behavior. Where novel tasks require a greater level of conscious attention for successful completion, automatized skills require little or no conscious attention, irrespective of additional processing demands (Nicolson & Fawcett, 1990). Dyslexia data showed that the motor difficulties that frequently accompany the disorder became pronounced when a motor task was undertaken concurrently with another non-motor task (i.e. dual–task condition) (Fawcett, Nicolson, & Dean, 1996). These findings led to the proposition that automatization, or lack thereof, may contribute to the motor impairments seen in dyslexia (see Visser, 2003). Since then, a number of studies have used this paradigm
to show similar effects in individuals with motor problems and ADHD symptoms (Raberger & Wimmer, 1999) leading to the suggestion that the Auto-D framework may explain the movement difficulties seen in DCD and other developmental disorders and, in part, their common co-occurrence (Visser, 2003).

In contrast to the DAMP and MBD theories, the Auto-D has been linked to a neural substrate, the cerebellum which subserves motor learning and other cognitive functions (Fawcett & Nicolson, 1999). Importantly, since the theory isolates a causal mechanism and neural basis, it provides a locus for remedial effort. Interestingly though, to my knowledge no effort has been made to develop such intervention.

Recent research on DCD samples has generally been supportive (Cherng, Liang, Chen, & Chen, 2009; Laufer, Ashkenazi, & Josman, 2008; Tsai, Pan, Cherng & Wu, 2009a). For example, recently, Tsai and colleagues (2009a) compared the performance of children with DCD with balance problems (controlling for co-morbid ADHD) and controls under five dual-task conditions. Each condition involved a primary balancing task and one of five cognitive tasks varying in oral or auditory complexity. Children in the DCD group performed significantly worse than controls on three of the five dual-task conditions, providing partial support for the Auto-D hypothesis. Other work has shown this dual-task effect to increase with task complexity in DCD (Cherng et al., 2009).

**Conclusion.** Theories suggesting generalized brain impairment in DCD tend to lack independent empirical support and, with the exception of the Auto-D hypothesis, offer little scope for the identification of causal mechanisms or for developing targeted approaches to remediation. What is clear from this research, however, is that ‘pure cases’ of DCD are the exception rather than the rule (Kaplan et al., 1998). Hence, there is a need to screen for co-morbid disorders when DCD is
suspected or diagnosed - and vice versa. This is particularly pertinent in light of research relating co-morbidity to poorer psychosocial outcomes (Rasmussen & Gillberg, 2002).

**Information Processing Accounts.** The information processing (IP) framework has provided researchers with a valuable framework for investigating DCD. The development of the computer in the 1950’s and 60s led to the information processing (IP) model of perception and action which proposed that like computers, human’s process perceptual and motor information in a fixed serial order (Lachman, Lachman, & Butterfield, 1979). Using this framework, researchers proposed that a deficit at one or more stages of processing may account for DCD symptomatology (Wilson & McKenzie, 1998). The main experimental methods of the IP approach include the additive-factor method (Sternberg, 1969) and the differential loading approach (Wickens, 2002). Briefly, these involved manipulating the processing demands on putative cognitive stages and noting changes in speed, efficiency and accuracy of movement that result. The effect of this manipulation on motor output was thought to indicate the integrity of these functions and their processing capacity. The IP framework has allowed researchers of DCD to try to isolate the causal processes responsible for atypical motor performance and development. For example, using a choice reaction time task, Smyth (1991) showed the movement times of children with DCD to be slower than controls for complex but not simple movements. He argued that this reflected a deficit at the stage of movement programming.

From an IP framework, a number of perceptual and motor processes have been implicated in DCD (Wilson & McKenzie, 1998). For example, in their seminal meta-analysis of the IP deficits associated with DCD, Wilson and McKenzie demonstrated the prevalence of deficits in visual spatial processing (Hulme, Biggerstaff, Moran, &
McKinlay, 1982; Lord & Hulme, 1987), kinesthetic perception (Bairstow & Laszlo, 1981; Laszlo & Bairstow, 1983) and cross modal perception (Newnham & McKenzie, 1993) in children with DCD.

One major criticism of IP theory is its apparent lack of ecological validity and failure to capture the real-time, interactive function of neural networks. Contemporary accounts of motor control now acknowledge the interaction of the individual, task and the environment (see below) in movement rather than it simply being controlled in a top-down fashion. This key limitation, at least in part, likely explains why IP theory has failed to provide a unifying account of the disorder and why it has largely failed to inform effective intervention programs (Wilson, 2005).

Cognitive Neuroscientific Approaches. In recent years, the cognitive neurosciences have provided a new and vital framework for understanding DCD. In keeping with developments in the cognitive sciences, this emerging multi-disciplinary field has seen a shift away from the stage-like processing espoused by traditional IP theory towards the function of multiple interacting networks in movement. Specifically, connectivist approaches to cognition now encapsulate the human brain’s capacity to undertake numerous processes in parallel supported by widespread, yet distinct, neural networks (for a good review see Roy, 2008). More recent examples have taken an embodied view of cognition borrowing key principles from dynamical systems theory (Castelnau et al., 2007). The product of this theoretical advancement is an understanding that cognition cannot be viewed in isolation of environmental and task constraints. Further, technological advances (such as functional magnetic resonance imaging (fMRI), electroencephalography (EEG) and transcranial magnetic stimulation (TMS) in recent years have substantially increased the fields understanding of the neural underpinnings of motor processes and the dynamic
interplay between brain and environment over the course of development.

From a developmental perspective, recent experimental work has examined the function of motor control networks that might be compromised in DCD. While theoretically espousing the ideals of embodied cognition, the main experimental methods of the cognitive neurosciences continue to rely on the manipulation the processing demands on putative mechanism and observing changes in behavioral output (as per IP theory). In the case of DCD, the neural basis of function/dysfunction has been directly investigated using EEG (Lust, Geuze, Wijers, & Wilson, 2006) and more recently with imaging techniques such as fMRI (Kashiwagi, Iwaki, Narumi, Tamai, & Suzuki, 2009; Zwicker, Missiuna, Harris, & Boyd, 2010) and structural MRI (Mariën, Wackenier, De Surgeloose, De Deyn, & Verhoeven, 2010). More frequently though, the structural basis of impairment is inferred through neuropsychological comparisons. Here, the structural loci of impairment is inferred by comparing the performance profiles of children with DCD and adults with damage to cortical regions which have resulted in a specific motor impairment [i.e. the PPC and internal representations of action and online control; and the cerebellum and movement timing]. Impairment is inferred when the atypical performance pattern demonstrated by children with DCD mirrors that of lesion patients; the greater the number of tasks used to corroborate the finding, the more reliable the inference. The hypotheses to emerge are both interesting and varied: most prominently, impaired movement timing related to the cerebellum (Castelnau et al., 2007; Lundy-Ekman et al., 1991; Volman & Geuze, 1998a); disinhibition of movement related to fronto-striatal circuitry (Mandich et al., 2002; Wilmut et al., 2007; Wilson, Maruff, & McKenzie, 1997) and impaired predictive control (viz internal modeling deficit, William et al., 2006; 2008, Wilson, Maruff, Ives, & Currie, 2001) involving parieto-
cerebellar networks. The following section provides a brief review of these models.

Timing Deficit Hypotheses. Atypical performance on measures of movement timing has been a well-documented feature of DCD, though their interpretation has varied. From an IP perspective, impairment at the level of a putative motor timing mechanism has been suggested (Williams et al., 1992) with contemporary work implicating the cerebellum (Lundy-Ekman et al., 1991) *ala* the cognitive neurosciences. More recently, others have adopted a dynamical systems interpretation, instead proposing a deficit in dynamic control (Volman & Geuze, 1998a,b). Recent examples of this dynamical approach have embraced the structuralist (neural) approach espoused by the cognitive neurosciences, proposing a role for the cerebellum, and even a central time keeping component (Castelnau et al., 2007). This work is discussed next.

Continuous tapping paradigms have been used extensively to investigate movement timing in children with DCD. Participants tap in time with an auditory (i.e. a beep) stimulus. Upon synchrony, the sound ceases and participants are required to maintain the original tapping frequency. From an IP perspective, a number of studies have employed this paradigm in children with DCD and demonstrated increased timing variability. For example, applying Wing and Kristofferson’s (1973) model using a continuous tapping task, Williams and colleagues showed that timing control in younger (6-7 years old) and older (9-10 years old) children with DCD was significantly more variable than controls on a uni-manual tapping task. They attributed this increased timing variability to a dysfunctional central timing mechanism.

Other work investigating tapping performance in children with DCD has also shown impaired performance and attributed it to an impaired central timing
mechanisms implicating impairment at the level of the cerebellum as per a cognitive neuroscientific framework. For example, Lundy-Ekman and colleagues (1991) divided a group of clumsy children into those who showed mild signs of cerebellar dysfunction and those with mild signs of basal ganglia damage and compared their performance on a continuous tapping task with age-matched controls. They found a double dissociation between the clumsy groups: the cerebellar group displayed increased inter-tap interval variability and force variability, whereas the timing variance of the basal ganglia group was within normal limits. Conversely, the basal ganglia group displayed increased force variance compared to the cerebellar group. When they partitioned this variance into central timing and movement implementation components as per Wing and Kristofferson’s (1973) model, the authors attributed the increased variance in the cerebellar group to an impaired central timing mechanism.

In more recent years, an alternative theoretical framework based on dynamical systems theory has been adopted to investigate temporal control of movement in DCD (Volman & Geuze, 1998a,b). According to dynamical systems theory, movement is the emergent property of the interaction between the physical constraints of the individual (i.e. neuro-developmental, biomechanical etc.) and informational constraints (i.e. previous task experience, instructions, motivation etc.) (Newell, 1986). Stable motor patterns are thought to be the product of effective self-organization of these sub-components rather than an a priori motor command or putative motor process or stage (as per IP models).

With respect to the timing of tapping, Kelso (1984) demonstrated that coordination between two fingers is characterized by two stable patterns: in-phase, relating to the activation of agonist muscles in each finger leading to synchronized
flexion and extension movements; and anti-phase patterns, where one finger flexes and the other extends simultaneously. Kelso found in-phase patterns to be more stable than anti-phase patterns, to the extent that when anti-phase oscillations reach a critical frequency, a transition to in-phase coordination is observed. In a study requiring adult participants to initiate finger flexion-extensions upon perception of metronomic flashes or to tap “on-beat” (synchronize) or “off-beat” (syncopate) to flashes, Engström, Kelso and Holroyd, (1996) showed these principles to hold for bimanual tapping to an external stimulus in adults.

Recent work adopting this framework has suggested that the movement timing deficits seen in DCD may result from unstable coordination patterns reflecting difficulties with the self-organization of movement sub-components. Volman & Geuze (1998a) measured children with DCD and age-matched controls ability to produce smooth rhythmic flexions/extensions of their index fingers either under in-phase or anti-phase conditions. Children with DCD showed significantly increased variability for anti-phase coordination patterns denoting less stable coordination patterns. The authors suggested that these results could not be accounted for by a central timing mechanism (viz IP accounts, i.e. Lundy-Ekman et al., 1991; Williams et al., 1992) as such a model was unable to explain coordination stability properties such as stability loss or phase transitions. Further, in-keeping with the phenomenological approach to movement endorsed by dynamical systems theory, they did not attribute the increased variability to a neural substrate because of the difficulties with linking abstract stability patterns to cortical structures.

In a more recent study applying the dynamical systems framework to Engström and colleagues’ (1996) paradigm in children with DCD, Castelnau and colleagues (2007) found that children with DCD were significantly more variable
when required to syncopate and synchronize finger movements to metronomic flashes, with a more pronounced effect observed during syncopation. Interestingly, in contrast to Volman and Geuze, (1998a) who also applied a dynamical systems approach to their investigation of timing in DCD, the authors suggested a role for cerebellar impairment based on the association between impaired anti-phase coordination (i.e. syncopation) and lesioning to the cerebellum in adults. In further contrast, they went as far as to suggest a possible role for a central timing deficit.

**Conclusion.** Motor timing impairment is a principle feature of motor difficulties in DCD but the issue of whether deficit at the level of a putative time-keeper or dynamic control best explains these temporal control issues remains unclear. While early work using a dynamical systems approach took a more traditional stance and questioned the validity of applying a structural (neural) approach to understanding movement in favor of a phenomenological account (Volman & Geuze, 1998a), more recent application of the dynamical systems theory (i.e. Castelnau et al., 2007) acknowledges the possible role of the cerebellum (and even the possibility of specific control mechanisms- i.e. a central time keeper) in these timing deficits. Since dynamical systems theory’s greatest asset is its ecological validity, and it’s greatest limitation is arguably the difficulty it presents in identifying specific structures or functions with which to focus intervention, the latter (Castelnau and colleagues) approach appears to provide a workable middle ground and hence offers an exciting foundation from which to investigate motor timing deficits in DCD. Finally, although there is converging neuropsychological evidence for a role of the cerebellum in impaired motor timing in DCD, there is limited neuroimaging evidence. Indeed, just one structural MRI study exists which showed atypical cerebellar morphology in a DCD case study (Mariën et al., 2010). Thus, further neuroimaging
research is required to verify the role of this neural region in the movement timing deficits seen in atypical motor development.

**Disinhibition and Poor Motor Skill.** The co-occurrence of inhibitory and motor performance issues has been well established in both younger (Livesey, Keen, Rouse, & White, 2006) and older typically developing children (Piek, Dyck, Francis & Conwell, 2007a) as well as in developmental disorders such as ADHD (Barkely, 1997). Given the often co-morbid nature of DCD and ADHD, it is perhaps unsurprising that recent evidence has highlighted a possible role for impaired inhibition in clumsiness. Specifically, recent work has demonstrated that children with DCD experience difficulty inhibiting or shifting attention (Wilmut et al., 2007) and inhibiting the initiation of inappropriate movement (Mandich et al., 2002). This research is discussed below.

Though not interpreted from the perspective of inhibitory control, possibly the first group of studies to find evidence for disinhibition in DCD using a cognitive neuroscientific framework were Wilson and colleagues (1997) and Wilson and Maruff (1999) who investigated visuo-spatial attention in children with DCD using the covert orienting of visual-spatial attention task (COVAT). Participants were required to respond manually to the presence of a stimuli in one of two peripheral locations which was preceded by a spatial cue that directed their attention either to the target location (valid cue) or away from the target location (invalid cue). After an invalid cue, participants had to disengage their attention from the incorrect target location and re-direct it to the correct target location, thus increasing reaction time (Posner, 1988). Wilson and Maruff presented two types of spatial cue eliciting two forms of attentional orienting: endogenous and exogenous. Where exogenous cues are thought to attract attention automatically, endogenous cues elicit voluntary attentional
shifts through visual space. Both Wilson and Maruff and Wilson and colleagues found that children with DCD took significantly longer to shift attention following invalid endogenous cueing compared to controls. This effect was not evident for exogenous cueing. Initially interpreted as a deficit in shifting of voluntary attention, an alternative explanation that was later investigated by Mandich and colleagues (2003) was that this pattern of performance may reflect a decreased capacity for disengaging or inhibiting voluntary attention from an invalid to a valid target. In their investigation, Mandich and colleagues employed the COVAT in a group of children with DCD, but this time as a specific indicator of inhibitory control, and replicated the findings of Wilson and colleagues. That is, children with DCD experienced difficulty shifting their attention following invalid endogenous cueing yet showed preserved performance following exogenous cueing. Interestingly, this profile of impairment appears to be a stable characteristic of DCD performance on the COVAT (Tsai, 2009; Tsai, Yu, Chen, & Wu, 2009b) and has shown to be responsive to intervention (Tsai, 2009). Research from adult groups linking this profile of deficit to dysfunction at the level of fronto-parietal attentional networks (Posner, Rothbart, & Sheese, 2007; Tsai, 2009) suggests that this region may be involved in the difficulties that children with DCD show inhibiting voluntary attention from an invalid goal.

Interestingly, inhibiting involuntary (automatic) attentional shifts might also be an issue for children with DCD, but only when a significant motoric component of behavior is involved. Wilmut and colleagues (2007) compared the performance of children with DCD and controls on a modified COVAT involving a ‘motor-free’ and ‘motor’ condition. The former measured shifts of attention during eye movements, the latter during eye and hand movements. The authors showed that contrary to the earlier findings of Wilson and colleagues who suggested that impaired attentional
shifts were selective to voluntary shifts of attention; children with DCD took longer to disengage involuntary attention from incorrect targets, but only under the motor condition. Thus, it appears that the ability of children with DCD to disengage voluntary and involuntary attention from inappropriate stimuli may be task dependent.

Finally, the inhibitory deficits seen in children with DCD appear to extend beyond attentional deficits to include difficulties inhibiting the initiation of movement. Mandich and colleagues (2002) recently demonstrated that children with DCD display a reduced ability to suppress initiation of an inappropriate manual response during the ‘Simon Task’, a pattern of deficit that has been linked to the basal ganglia-frontal cortex network (Praamstra, Kleine, & Schnitzler, 1999).

**Conclusion.** The above group of studies provides promising preliminary evidence that children with DCD may experience inhibitory difficulties when a response to a goal compels action. Clearly, additional work is required to better establish those aspects of the multifaceted construct of inhibition that might be compromised and the specific conditions under which impaired inhibition might contribute to poor motor control in DCD. To date, there have been a limited number of studies testing the disinhibition model of DCD, and in the case of attentional control, evidence has been predominantly based on performance on the COVAT, or a variant thereof. Further, establishing the neural basis of poor movement inhibition is complicated by a lack of neuroimaging evidence in children with DCD and adult work suggesting that different inhibitory mechanisms may be subserved by distinct neural loci. For example, while difficulties voluntarily disengaging attention from an invalid cue have been linked to fronto-parietal networks (Posner et al., 2007), difficulties inhibiting movement initiation towards a compelling target may involve fronto-basal ganglia networks (Praamstra et al., 1999). Thus, continued experimental
work is required to clarify the role of dishinhibiton in atypical motor development, and its neural substrate/s.

Predictive Control (viz internal modeling) and DCD. Wilson and colleagues provide converging data that the motor difficulties experienced by children with DCD may, at least in part, be the result of an impaired ability to generate and/or monitor internal (predictive) models of movement. This hypothesis is based on evidence from measures of motor imagery (MI) (Williams et al., 2006, 2008; Wilson, Butson, Lum, & Thomas, 2004), oculomotor paradigms (Wilson et al., 1997; Wilson & Maruff, 1999) and an intervention study (Wilson, Thomas, & Maruff, 2002). More recently, the IMD hypothesis has also generated support outside Wilson and colleagues’ laboratory (Deconinck, Spitaels, Fias, & Lenoir, 2009). This work is discussed in detail in the following section.

Internal modeling and motor control. Recent computational modeling suggests that there are two types of internal model important to the control of purposive action: forward models use a copy of the motor command (viz efference copy) to the “plant” to predict the future state of the moving limb (Desmurget & Grafton, 2003; Jeannerod, 2006; Wolpert, 1997). The inverse model (or controller) generates the motor command necessary to achieve the desired goal state. With respect to the former, adaptive movement is predicated on the ability of the nervous system to predict the future location of the moving limbs using a forward (predictive) model (Shadmehr, Smith, & Krakauer, 2010). This forward estimate of limb position provides a means of rapidly integrating efferent and afferent signals - sometimes referred to as an internal feedback loop – thereby speeding responses to any changes in the environment during the course of movement (Desmurget & Grafton, 2003). This type of control circumvents delays associated with processing sensory feedback
– which can be up to 250 ms (Frith, Blakemore, & Wolpert, 2000)- since the position of a moving limb has changed appreciably by the time sensory feedback can be used to alter the unfolding motor command. Forward models are thought to contribute to movement control by anticipating the sensory consequences of movement, allowing the motor system to detect discrepancies between predicted movement and that indicated by actual sensory inflow. Error signals are thusly generated to correct the ongoing motor command (Desmurget & Sirigu, 2009). These error signals can then be used to correct and/or re-train both the predictive (forward) and inverse models of movement. A deficit in this process may account for at least some of the movement difficulties experienced by children with DCD, as discussed below.

**Predictive modeling is impaired in children with DCD.** A group of studies investigating visuo-spatial processing in children with DCD using the COVAT were the first to suggest that an internal modeling deficit may contribute to poor motor skills in children with DCD (Wilson et al., 1997; Wilson & Maruff, 1999). As noted above, these studies revealed that children with DCD display selective impairments in the voluntary control of covert attention, shown by delays in disengaging attention from invalidly cued targets. Based on the assumption that voluntary control of attention under such conditions relies on internal representations of behaviour (i.e. internal models) these results suggested an underlying problem in generating or monitoring internal models of oculomotor plans.

Soon after this work, Katschmarsky and colleagues (2001) compared the performance of children with DCD and age-matched controls on the double-step saccade task (DSST) arguing that impaired forward modeling in DCD would see dysmetria on the second saccade. This task requires participants to visually track two targets that are presented sequentially and then disappear. The first appears for 140
ms followed by the second for 100 ms. Participants are required to generate sequential saccades from the first to the second target. It is suggested that since the second target appears in a location different from the initial target, a spatial dissonance between the retinal coordinates of the second target and the required saccadic motor coordinates arises. Hence, in order to make an accurate second saccade, a forward model of the eye-movement must be generated to predict the endpoint of the first eye movement. As predicted, the authors found that children with DCD were dysmetric on second saccades. Their performance suggested a reduced ability to program saccade sequences using a forward model, unlike age-matched controls. Interestingly, this pattern of performance was similar to patients with lesions to the PPC. Since these earlier studies, Wilson and colleagues have shown impaired performance on measures of predictive modeling in DCD using a number of MI paradigms; these are discussed next.

**DCD and Motor Imagery: A window into an IMD.**

Children with DCD have shown consistent impairments in motor imagery (MI) (Williams et al., 2006; 2008; Wilson et al., 2001, 2004), an activity thought to directly correspond to the efference copy of the forward model. Motor imagery, the process of mentally rehearsing, or simulating, a proposed movement without overt execution (Jeannerod, 2006), has shown to be bound to the same temporal (Cerritelli, Maruff, Wilson, & Currie, 2000; Decety, Jeannerod, & Prablanc, 1989; Sirigu et al., 1996), biomechanical (Parsons, 1994; Petit, Pegna, Mayer, & Hauert, 2003) and environmental constraints as actual movement (Cerritelli et al., 2000; Decety et al. 1989) in normal adults and typically developing children. Further, the neural networks subserving the two processes overlap considerably (Jeannerod, 2001; Jeannerod, 2006). Thus, it is widely accepted that MI is the efference copy used by the forward
model which only comes to conscious awareness because movement has been inhibited (Crammond, 1997; Decety & Grèzes, 1999; Jeannerod, 1997). The integrity of the predictive (forward) model is therefore inferred from performance profiles during MI.

Performance during the visually guided pointing task (VGPT) has revealed atypical execution in children with DCD when compared to age-matched controls and adults (Wilson et al., 2001). The VGPT involves participants moving their hand either physically or mentally between targets of differing size (Sirigu et al., 1996). Wilson and colleagues (2001) found that the movement time of children with DCD did not conform to Fitts' Law (Fitts, 1954) as was the case for controls and had previously been shown in adults (Decety & Jeannerod, 1996). Further, the imagined movement of children with DCD did not increase with the addition of weight as was the case for control children and healthy adults (Decety et al., 1989) suggesting an impaired ability to generate, or employ, predictive (forward) models of movement. This pattern of performance resembled that of patients with lesions to the PPC during the same task (Sirigu et al., 1996).

In an ensuing group of mental rotation studies, Wilson and Colleagues showed that children with DCD display atypical reaction times (Wilson et al., 2004) and increased errors (Williams et al., 2006) during the mental rotation of limbs. Mental rotation paradigms require participants to determine the handedness of single hands presented at 45º increments from upright either clockwise or anti-clockwise. Wilson et al., (2004) found that the reaction time of control children increased linearly as the hand rotated away from 0º, a profile previously seen in adults (Kosslyn, Digirolamo, Thompson, & Alpert, 1998). This trade-off was much smaller in children with DCD, a pattern of performance evidenced in PPC patients (Sirigu & Duhamel, 2001). It was
suggested that children with DCD experienced difficulty employing MI and may have instead relied on visual imagery (VI) to complete the task, an alternative imagery strategy that is not bound by the same biomechanical and physical constraints as MI and actual movement. This proposition was subsequently supported in a follow-up investigation (Williams et al., 2006) which demonstrated that children with DCD did not benefit from explicit MI instructions (c.f. controls) during the mental rotation of limbs, yet displayed preserved imagined alphanumeric rotation (i.e. VI). Later work showed these MI difficulties to be more pronounced in children with more severe motor impairment (Williams et al. 2008). Interestingly, in contrast to Wilson and colleagues’ study where reaction time was atypical, Williams and colleagues (2006) showed that difficulties conducting MI in DCD manifest as increased error yet preserved reaction time.

Finally, the IMD hypothesis was challenged by Wilson and colleagues (2002) by evaluating an imagery training protocol designed to train the ability to generate predictive models for action in children with DCD. The protocol, which was administered using interactive DVD, showed to be as effect as traditional perceptuo-motor training in facilitating skill development.

Further evidence for impaired predictive modeling in DCD. The suggestion that predictive modeling might be compromised in DCD has also found direct and indirect support outside Wilson and colleagues laboratory. In a recent study, Deconinck and colleagues (2009) showed that children with DCD were significantly slower and less accurate when mentally rotating images of a hand than controls, though the pattern of movement met the typical speed-accuracy trade-offs. While this finding differs somewhat from Wilson and colleagues (2004) work that demonstrated an abnormal RT profile in DCD, it nonetheless provides evidence independent of
Wilson and colleagues laboratory that MI may be impaired in children with DCD suggesting difficulty generating and/or monitoring predictive models. Next I will discuss evidence that atypical predictive control might explain some of the motor control and motor learning (adaptation) difficulties that children with DCD commonly experience. Before I do this I will briefly outline the important distinction between motor control and motor adaption, and the putative role that predictive modeling plays in each process.

Motor control and adaptation: the role of predictive modeling. As I will discuss in greater detail below, according to computational models successful control of movement in-flight (and correction) is dependent on the nervous systems capacity to generate an accurate predictive model of the impending movement which encapsulates the expected sensory consequences of the movement and compare it to the actual sensory consequences of action throughout the movement cycle (Desmurget & Grafton, 2003; Gritsenko, Yakovenko, & Kalaska, 2009; Shadmehr et al., 2010). Should discrepancy arise (i.e. following unexpected visual perturbation) an error signal must be generated and used to re-calibrate the on-going motor command with minimal time-lag.

The process of combining predictive and feedback-based mechanisms to control movement is, however, only an effective control mechanism if the initial prediction is accurate (Shadmehr et al. 2010). Continuous error over repeated trials such as when trying to touch a target or grasp an item without success, indicates that the initial prediction may be imprecise and must be updated or adapted. This issue is particularly pertinent for the developing child where neuro-muscular maturation sees continual change in the goal-limb relationship, rendering predictive models obsolete on an almost continuous basis. For motor control to remain stable, and indeed refine,
the motor system must be able to maintain the accuracy of predictive models. Computational modeling posits that just as the error signal that arises following disparity between the expected (according to the predictive model) and actual consequences of movement can be used to re-calibrate the on-going motor command for the purpose of motor control, so too might it serve to adapt predictive (internal) and inverse models of movement (Magescas, Urquizar, & Prablanc, 2009; Shadmehr & Krakauer, 2008; Shadmehr et al., 2010). Indeed, the latter has been proposed as a plausible framework for motor maturation and skill acquisition (Wolpert & Ghahramani, 2000).

In the last four decades, prism adaptation tasks (PAT) have been the cornerstone of investigation into visuomotor adaptation. Traditionally, these require participants to make goal directed movements (i.e. drawing, pointing, throwing a ball) firstly with normal vision and then following undetectable visual displacement to the left or right (for example 10-15°) usually using prism goggles or glasses. Vision is then restored to normality for the final stages of assessment. During the first stage (normal vision), participants generate a predictive model which incorporates sensory-motor reference frames of the relationship between the limb and goal (Redding, Rossetti, & Wallace, 2005). Imperceptible visual displacement alters these relationships slightly, resulting in discordance between the expected consequences of the motor plan (according to the predictive model) and that indicated by sensory inflow during movement. Accordingly, in healthy adults, following prism displacement, the first few trials tend to miss their target in the direction of prismatic displacement. Over a number of trials the resultant error signal is used to update or adapt the predictive and inverse models incrementally to account for the change in limb to goal relationships. In behavioral terms, this process is reflected in a return to pre-prism
accuracy levels (for a good review see Redding et al., 2005). Successful adaptation of the predictive and inverse models is further indicated by the presence of performance ‘after-effects’ when vision is returned to normal. That is, throwing again misses the target, but this time to the opposite direction of the prismatic displacement. Interestingly, cerebellar patients show either impaired or absent motor adaptation during PATs (Martin, Keating, Goodkin, Bastian, & Thach, 1996; Morton & Bastian, 2004) suggesting that the cerebellum may be important in the adaptation of predictive models, a notion supported by neuroimaging work (Diedrichsen, Hashambhoy, Rane, & Shadmehr, 2005).

In the coming paragraphs, I will discuss evidence supporting the view that impaired predictive modeling may account for some of the common motor control difficulties experienced by children with DCD, for example, during drawing (Smits-Engelsman et al., 2003), postural control (Jover, Schmitz, Centelles, Chabrol, & Assaiante, 2010; Przysucha et al., 2007) and grip-force control (Pereria et al., 2001). I will then discuss evidence suggesting that impaired predictive modeling might also explain some of the difficulties children with DCD experience adapting movement over longer time-scales (i.e. motor learning).

Motor control studies. Recently, Smits-Engelsman and colleagues (2003) found that children with DCD and learning disabilities (LD) made significantly more errors than age-matched controls when drawing cyclically between two points. Based on the assumption that cyclical movements of this kind rely predominantly on the predictive (internal) model and in the case of error, the integration of sensory feedback to correct the internal model, the authors proposed that the increased error shown by the DCD-LD group might reflect difficulty controlling movement under feed-forward (predictive) control. An alternative
explanation is that this group experienced difficulty correcting the motor command by integrating predictive estimates of limb position with sensory feedback.

With respect to postural control, Przysucha and colleagues (2007) showed that postural control during leaning was less efficient in children with DCD compared to controls. Here, movement was parsed into feedfoward (predictive) and feedback-based control stages according to time taken to reach peak velocity as per dual-component models of motor control (which I discuss below). Briefly though, these posit that temporal constraints prevent sensory feedback from influencing the early stages of movement (i.e. until peak velocity) which is thought to be under the control of the motor command. This phase is followed by a feedback-based control stage where sensory feedback is used to guide the moving limb to improve terminal accuracy. The authors showed that children with DCD spent 54% of movement time using the more efficient online (feedback based) control strategy to guide leaning compared to 78% for control children. Since, from a computational perspective online control of this kind depends on the nervous systems capacity to integrate sensory afference with predictive estimates of limb and body position, these results support the notion that children with DCD may experience difficulty using this integrated system to control posture.

Similarly, in a recent experiment conducted by Jover and colleagues (2010), 16 DCD and control children held their arm out horizontally with a weight attached to their forearm and were required to maintain arm position following involuntary or self-induced load removal. Postural stabilization following the latter depends on the nervous systems ability to anticipate and compensate for the motor consequences of load removal. This process is inferred from decreased elbow flexion and reduced EMG activity in the flexor muscles [commencing prior to load removal] (Schmitz,
Martin, & Assaiante, 2002). Jover and colleagues showed that arm stabilisation was poorer in children with DCD following self-induced load removal suggesting poor anticipatory (viz predictive) control. Specifically, children with DCD showed delayed inhibition of flexor muscles and no correlation was shown between their inhibition and arm stabilization. On this latter point, for control children earlier flexor inhibition was correlated with improved arm stabilization indicating more efficient use of predictive control strategies to maintain arm stability.

Finally, Pereria and colleagues’ (2001) investigated control of grip force during lifting in children with DCD, DAMP and controls. Participants were required to grasp and lift a small object repetitively. Under these conditions, efficient grip force adjustments are required to ensure that the object does not slip from the participants’ grasp yet unnecessary force is not exerted. For example, if you want to eat a ripe strawberry you must grasp it with enough force to ensure that it does not slip from your hand, yet take care not to grasp so powerfully as to damage it. Successful motor output in these conditions is dependent on the nervous systems ability to anticipate the force and load fluctuations associated with the speed and duration of movement according the objects weight. The heavier the object and faster the movement, the greater the inertial load and the greater the grip-force necessary to prevent the item slipping. The authors showed that children with DCD employed greater grip force and safety margins than controls suggesting a poorly defined predictive model of the impending movement.

Learning (adaptation) studies. Kagerer and colleagues (2006) demonstrated that children with DCD failed to show clear after-effects on centre-out drawing following removal of 60º visual-feedback rotations implemented gradually in 10º increments. Since these after-effects are assumed to reflect adaptation of the
internal model of the visuo-spatial relationship between the individual and target (which have been altered by visual-feedback rotation), the lack of after effects shown by children with DCD indicates difficulty adapting internal models by way of sensory feedback mechanisms. Interestingly, cerebellar patients display similar profiles of deficit during adaptation tasks (Martin et al., 1996; Morton & Bastian, 2004).

Partial support for the notion that children with DCD experience difficulties adapting internal models was also provided by an investigation of ball throwing following prismatic displacement of vision in 9 children with DCD control children (Cantin, Polatajko, Thach, & Jaglal, 2007). Interestingly no overall group differences on adaptation were detected. However, analysis of individual differences showed that only 3 of the 9 children with DCD showed normal adaptation following prismatic displacement and its removal; conversely, all control children were able to do so. While this pattern of results does suggest some difficulty adapting internal models in children with DCD, the small sample size and mixed results make it difficult to draw reliable inferences from the data. Particularly difficult to reconcile is the fact that most children with DCD were able to adapt their throwing either after prismatic displacement or after it’s removal (i.e. in one of two adaptive stages).

Conclusion. In comparison to other plausible aetiological accounts that have emerged from a cognitive neuroscientific framework, the IMD hypothesis has arguably withstood the greatest level of scrutiny. Though the notion of impaired predictive modeling presents as one of the more exciting explanatory models from which to investigate DCD, there is clearly some disparity in the available lines of evidence. With respect to data on MI for example, some work suggests atypical reactions times during the mental rotation of limbs (Wilson et al., 2004), while others suggest typical reaction times but inaccurate (Williams et al., 2006) and inefficient
profiles (Deconinck et al., 2009). Work on motor adaptation in DCD also reflects this inconsistency with some work showing that children with DCD are unable to adapt their movement over repeated trials following undetectable visual displacement (Kagerer et al. 2006) yet others have failed to demonstrate group differences compared with control children on similar tasks (Cantin et al., 2007) - though in the case of the latter, assessment of individual differences in this study provides some evidence of impaired adaptation. Though each of these studies indicates impaired predictive modeling at some level, there is need for continued work into the IMD hypothesis to better establish the integrity of predictive modeling in children with DCD and its role in impaired motor control and learning. Further, despite performance similarities between children with DCD and PPC and cerebalator patients on a number of paradigms, confirmatory neuro-imaging is required to determine the neural basis of impaired predictive control in DCD.

**Summary of Cognitive Neuroscientific Accounts of DCD.** The above section highlights some of the divergent aetiological accounts of DCD to have emerged using a cognitive neuroscientific framework. Though each theory carries with it a degree of empirical validity, each is also associated with limitations usually related to a lack of independent experimental scrutiny, limited evidence, and/or inconsistent results. As a result a unified account of DCD is yet to emerge. This disparity illustrates the need for further experimental work.

In the coming pages, I will propose that the domain of ROC as measured from double-step reaching performance interpreted using a computational interpretive framework holds great promise in clarifying issue surrounding one of the more promising aetiological accounts of DCD - the IMD hypothesis. This argument begins with a discussion of the evidence on the nature of ROC in clumsy children.
Rapid online motor control in children with Developmental Coordination Disorder (DCD)

One aspect of motor control that has been associated with DCD and that is particularly important to fluid motion is ROC of reaching. In typically developing children, the nature of online control alters with the changing constraints of maturation and experience. Data on online control in DCD, however, has been conflicting. While some recent work using double-step reaching suggests no difficulty in online control (e.g., Plumb et al., 2008), others suggest deficits (Wilmut et al., 2006). The following section will posit that the use of current neuro-computational models as a framework for investigating DCD and a more controlled study of double-step reaching are required to better elucidate the nature of online motor control in children with DCD.

Double-step reaching tasks and online control. One of the mainstays in the investigation of ROC has been target perturbation paradigms, specifically the so-called double-step reaching paradigm (DSRP). Participants are required to reach and touch (or grasp) one of a number of possible targets. For the majority of trials, the target remains the same for the duration of the movement (Figure 1.1 a), while for a small number of trials, the target changes (or ‘jumps’) unexpectedly (Figure 1.1 b); hence, participants must adjust their movement ‘on the fly’. This approach has a rich history in the cognitive neurosciences, informed initially by traditional dual-component models of motor control (see Elliott, Helsen, & Chua, 2001), but more recently by computational models (Castiello, Bennett, Bonfiglioli, Lim, & Peppard, 1999; Desmurget & Grafton, 2003; Gréa et al., 2002; Farnè et al., 2003; Sarlegna, 2006). Traditional dual-component models view motor control as occurring in distinct feedforward (or ballistic) and feedback phases based on the assumption that temporal
constraints prevent sensory feedback from influencing the initial stages of movement (Desmurget & Grafton, 2000; Elliott et al., 2001; Woodworth, 1899). Control of reaching is thought to be initially ballistic in nature, while sensory feedback is used in the latter stages to ‘hone-in’ reaching. As stated above, cognitive neuroscience models now view mature reaching as being controlled by an integrated system (viz. internal modeling); a view supported by a number of studies illustrating that corrections to movement can be made within the order of 100ms (Castiello, Paulignan, & Jeannerod, 1991; Farnè et al., 2003; Paulignan, MacKenzie, Marteniuk, & Jeannerod, 1991), more quickly than (external) feedback-based mechanisms are able to accommodate which can take upwards of 250 ms (Frith et al., 2000).

Figure 1.1. A graphical representation of a ‘non-jump’ (a) and jump trial (b).

**Double-step reaching performance in DCD.** In the case of DCD, it is within the context of dual-component models that the only study to use a double-step reaching paradigm was conceived and results interpreted. Recently, Plumb and
colleagues (2008) compared the performance of children with DCD and controls on a visual perturbation task. The authors split movement into feedforward and feedback stages; time to peak velocity was suggested to represent the point at which reaching ceased to be under feedforward control, with feedback-based mechanisms controlling the remainder of the movement or homing-in phase. The children were required to stand and complete the task using a hand-held stylus. However, the task was modified for children with DCD who had difficulty managing the stylus and performing the task from a standing position. While children with DCD took significantly longer to complete the task (viz MT) and spent more time in the decelerative (feedback based) phase of movement, there was no differential effect of condition (jump vs. no-jump).

The authors suggested that these results were indicative of a generalised deficit in producing movements rather than a specific online control deficit. However, this interpretation is clouded by the fact that the two groups were performing different versions of the same task: children with DCD were seated, unlike controls, and used a much thicker stylus. By the author’s own admission, these modifications simplified the postural and grasp demands of the task for the DCD group. It is a well-documented phenomenon that children with DCD have particular difficulty completing complex movements (i.e. Smyth, 1991; Wilmut et al., 2006). By simplifying the task demands for the DCD group relative to age-matched controls, I argue that any comparison between conditions (‘jump’ and ‘non-jump’) is compromised. Further, the theoretical framework (i.e. the dual-component model of motor control) employed is at odds with recent insights into the nature of ROC, courtesy of neuro-computational modeling. There is a growing consensus that feedback mechanisms are integrated with feedforward mechanisms continuously throughout movement, and not just at the end point (Saunders & Knill, 2003; 2005).
Performance on double-step tasks in DCD: preliminary insight into online control. Two recent studies have investigated the performance of children with DCD on sequential double-step tasks, the first based on a computational framework (Katschmarsky et al., 2001; Wilmut et al., 2006). Double-step performance in DCD was first investigated using an occulomotor paradigm that required sequential saccades—the double-step saccade task (DSST). As stated above, Katschmarsky and colleagues (2001) compared the performance of children with DCD and age-matched controls during the DSST. To re-iterate, the task required that children to track two targets that were presented sequentially. The first was presented for 140 ms, while the second followed immediately for 100 ms requiring participants to generate sequential saccades from fixation on the first to the second target. The second target was extinguished prior to the initiation of the first saccade resulting in disparity between the retinal coordinates of the second target and the required saccadic motor coordinates. In order to produce an accurate second saccade, participants were required to generate a predictive (forward) model of the eye-movement to predict the terminal point of the first eye movement. As predicted, the authors found dysmetria on the second saccade in children with DCD suggesting a decreased capacity to use a forward model to program saccade sequences. However, the DSST is an occulomotor paradigm and one that does not engage mechanisms for correcting movements in-flight. Thus it is difficult to draw inference about ROC in DCD from this evidence.

More recently, Wilmut and colleagues’ (2006) investigated the coordination of hand and eye coupling during a sequential reaching task. Children with DCD and age-matched controls were required to reach for targets under three conditions: a single touch condition during which children reached and touched a single target; a ‘double’
touch’ condition where participants touched two targets presented sequentially; and a ‘double off’ condition, like the ‘double’ condition except that targets were extinguished 250 ms after movement onset. The results showed that while the two groups did not differ on simple aiming, DCD children displayed increased MT and decreased accuracy when making sequential movements. Increased foveation prior to movement in the DCD group suggested a greater reliance on visual feedback control compared with controls. This increased foveation was also suggested to disrupt use of efference copy which must be ‘buffered’ if foveation is prolonged: the longer the foveation, the less accurate the initial efference copy will be as the environment and task change.

Though this pattern of results may explain the slower, more variable reaching seen in children with DCD, the ‘double’ touch condition employed by Wilmut and colleagues (2006) required participants to sequentially touch two targets rather than adapt their movement online because the goal of reaching has changed altogether [as is the case for traditional double-step reaching tasks]. Consequently, rather than using feedback mechanisms to correct an on-going motor command ‘on the fly’ as is required during the ‘jump’ conditions of double-step reaching paradigms, participants would have been able to complete the initial motor command in it’s entirety and then generate a second motor command to complete the task (i.e. reaching from the first to second target). For this reason, while Wilmut and colleagues study informs the nature of eye-hand coupling in DCD, as was their intention, it does not measure ROC per se. Nonetheless, the studies conducted by Wilmut and colleagues and Katschmarsky and colleagues (2001) highlight the important insights that computational models offer our understanding of DCD, and in particular, online motor control. However, the nature of ROC in DCD still remains unclear.
Double-step reaching interpreted using computational models can clarify the nature of ROC in DCD and issue surrounding the IMD hypothesis. Though not yet seen in the DCD (or developmental) literature, at present computational models provide the dominant framework for investigating double-step reaching performance in healthy and patients adults (Castiello et al. 1999; Desmurget & Grafton, 2003; Gréa et al., 2002; Farnè et al., 2003; Sarlegna, 2006). As discussed above, these propose that ROC is only viable to the extent that predictive (forward) and feedback based mechanisms are able to be integrated seamlessly. During non-jump trials, provided that the initial motor command is accurate, the motor command unfolds largely uncorrected since the target remains stationary throughout movement. Thus, though a degree of adjustment is required since the motor command is imperfect (Desmurget & Grafton, 2000), discrepancy between the expected limb trajectory (with respect to the target) and that indicated by actual sensory in-flow is expected to negligible and, hence, demands on online control are minimal. Accordingly, a specific deficit in online control would not be expected to manifest in performance on non-jump trials.

Conversely, in the case of jump trials, a predictive (forward) model of limb position relative to the target is generated and compared with sensory afference which signals the actual target location (in relation to the moving limb). The resulting mismatch is thought to generate an error signal that is used to update limb trajectory. More precisely, computational modeling suggests that rapid online corrections are organized by “superimposing” a dynamic error signal onto the un-folding motor command (Gritsenko et al., 2009). These online adjustments are tuned to the dynamic inertial properties of the moving limb and circumvent the processing delays associated with sensorimotor feedback loops (Flanagan, Vetter, Johansson, &
Wolpert, 2003). Deficits in this process manifest as increased movement time and decreased accuracy select to jump trials. Interestingly, this pattern of deficit is evident in disorders known to impact ROC and forward modeling (e.g. optic ataxia in parietal patients, Gréa et al., 2002).

Not only does the aforementioned theoretical framework provide great scope for clarifying the nature of ROC in children with DCD, but it may also address issue surrounding existing cognitive neuroscientific accounts of DCD; specifically the IMD hypothesis. While there is compelling empirical support for the IMD hypothesis, as I have highlighted above, lines of evidence are conflicting. For example, with respect to evidence demonstrating MI difficulties in DCD, some work has suggested atypical reaction time profiles (Wilson et al., 2004), while others have found typical reaction time profiles but decreased accuracy (Williams et al., 2006) and efficiency (Deconinck et al., 2009). By adding to the existing body of work on predictive modeling in DCD, the area of ROC as measured using the DSRP and interpreted using a computational framework may help to clarify the role of predictive modeling in atypical motor development. If, as predicted by the IMD hypothesis, children with DCD experience difficulties implementing or correcting predictive (internal) models of movement, such impairment would manifest as a deficit in online control- which I have argued depends on the successful integration of predictive and feedback-based mechanisms. This would see slower, less accurate movement select to jump trials of the double-step reaching task.

Conclusion. ROC shows great potential in clarifying issue surrounding the aetiology of DCD- specifically, the IMD hypothesis. However, data on online control in DCD is conflicting with some work suggesting impairment (Wilmut et al., 2006) and others preserved ROC (Plumb et al., 2008); consequently, the exact nature of
online control in children with DCD remains unclear. Two aspects of investigation are required to clarify debate surrounding this issue: firstly, the use of neuro-computational modeling as a framework for interpreting online performance of children with DCD; and secondly, a more controlled investigation of reaching during the double-step task.

**Summary of Chapter 1**

To summarize, DCD is characterized by below age-appropriate motor function that tends to persist into adulthood. These motor difficulties significantly impact the child’s ability to undertake tasks important to everyday living and/or educational achievement. Despite a prevalence rate of roughly 6%, a unified aetiological account of the disorder continues to be elusive. This chapter reviewed the available accounts including the developmental delay and brain impairment theories, followed by information processing and cognitive neuroscientific accounts.

The cognitive neurosciences provide a new and vital framework from which to investigate the aetiological basis of DCD. The theories developed from this framework are numerous and interesting though none has provided a unified account of the disorder. I reviewed three of the most promising accounts to have emerged: impaired movement timing relating to the cerebellum (Lundy-Ekman et al., 1991; Castelnau et al., 2007), deficit in inhibitory control involving fronto-striatal regions (Mandich et al., 2002), and impaired predictive control (viz the IMD hypothesis) possibly involving parietal and, or, cerebellar regions (Williams et al., 2006, 2008; Wilson et al., 2001, 2004). Though promising, I noted disparity in this literature and the need for continued experimental work.

It was argued that the domain of ROC of reaching might help clarify issue
surrounding one of the more promising cognitive neuroscientific accounts, the IMD hypothesis. While ROC is crucial to mature fluid movement and has been implicated in DCD, the available research is conflicting: some recent studies using double-step reaching suggest no impairment in online control (e.g., Plumb et al., 2008), others suggest deficits (during sequential pointing, Wilmut et al., 2006). I argue that two steps are required to clarify this debate: the use of neuro-computational models as a framework for investigating ROC in DCD and a more controlled investigation of double-step reaching.

Accordingly, the first study of this thesis, presented in the following chapter, aimed to address existing controversy about the nature of online control in children with DCD, using a double-step reaching task interpreted from a computational perspective. A chronometric approach was adopted here with deficits expected to manifest as a selective delay in movement time to jump trials (Desmurget & Grafton, 2003).
CHAPTER 2

STUDY 1: DOUBLE-STEP REACHING PERFORMANCE IN CHILDREN WITH DEVELOPMENTAL COORDINATION DISORDER:

CHRONOMETRIC ANALYSIS OF REACHING
Introduction and Overview

Chapter 1 reviewed the more prominent aetiological accounts of DCD to have emerged from a cognitive neuroscientific framework—movement timing impairment related to the cerebellum (Lundy-Ekman et al., 1991; Castelnau et al., 2007), disinhibition of movement related to frontal-striatal regions (Wilmut et al., 2007; Wilson et al., 2001) and impaired predictive control possibly involving the PPC (Wilson et al., 2004; Williams et al., 2006). I argued that while the IMD hypothesis offered one of the more promising accounts to have emerged from this framework, inconsistencies in the available evidence highlighted the need for continued experimental work to clarify the role of predictive modeling in the disorder. Finally, I argued that the domain of ROC as measured by the double-step reaching task interpreted using a computational framework showed great potential in addressing this issue.

As noted in Chapter 1, evidence on the nature of ROC in DCD, however, is mixed. For example, work by Wilmut et al. (2006) demonstrated that children with DCD were equally as efficient when reaching to a single target but were slower and less accurate when reaching sequentially from one target to another; the DCD group spent more time foveating targets presented sequentially before initiating hand movements, which led to an increase in error. This pattern, while suggestive of difficulties in feedforward control, did not examine directly ROC in response to target perturbation. More recently, Plumb and colleagues (2008) suggested preserved ROC during reaching in DCD. They found that the effect of target perturbation on movement time was similar for both DCD and non-DCD groups. However, Plumb
also acknowledged that there were methodological limitations in this study; group comparisons were compromised by the fact that DCD and control groups performed different versions of the same task (i.e. children with DCD were seated and used a large pointing stylus, while control children stood and used a smaller stylus).

The aim of Study 1 was to address the existing controversy about the nature of online control in children with DCD, enlisting chronometric analysis of performance during a double-step reaching task designed and interpreted from a modern neuro-computational perspective. My working assumption here was that online corrections to reaching are viable to extent that the nervous system is able to monitor and detect discrepancy between the expected sensory consequences of movement (according to the forward model) and those indicated by actual sensory in-flow and integrate this information with the on-going motor command. Difficulties doing so manifest as a selective delay in movement time and increased error for jump trials.

**Impaired ROC and Optic Ataxia.** Interestingly, the above pattern of double-step reaching is evident in disorders known to impact online control and forward modeling i.e. optic ataxia seen in posterior parietal patients (Blangero et al., 2008; Gréa et al., 2002; Pisella et al., 2000). For example, Gréa et al. (2002) demonstrated that other than a slight increase in movement time, the reaching characteristics of a patient with bilateral lesions to the PPC were similar to controls for non-jump trials; however, patient movement time was significantly longer for perturbed trials. Further analysis showed that patients tend to reach towards the original goal before generating a second movement towards the new target, rather than making smooth online corrections as per controls.

**Summary.** Study 1 involved chronometric analysis of double-step reaching in children with DCD; performance was interpreted from a neuro-computational
perspective. This analysis was designed to clarify the nature of online control in children with DCD. It was predicted that children with DCD would display a reduced capacity for correcting their movements online shown by slower more error ridden reaching select to jump trials in comparison to age-matched controls.

**Method**

**Participants**

The sample consisted of 17 children (9 girls and 8 boys) between the ages of 7 and 12 years of age who met the research criteria for DCD and 27 age-matched controls (13 girls and 14 boys). Mean age for the DCD group was 9.68 years ($SD=1.7$) and 9.83 years ($SD=1.8$) for the controls. As determined by performance on the McCarron Assessment of Neuromuscular Development (MAND; McCarron, 1997), all children were right-hand dominant with the exception of four children in the DCD group and one control child. The RMIT University Human Research Ethics Committee (HREC) and Research Branch of the Victorian Government Department of Education and Early Childhood Development approved the research project and all parents and children gave informed consent (for a copy of the parental and child plain language statements and consent forms see Appendix B and C respectively).

Children were screened using a two-step procedure (as per Wilson et al., 1997) which was also adopted for the research presented in Chapter 3 and Chapter 4. Principals from two state primary schools in suburban Melbourne were approached and invited to participate in the study. Classroom teachers from these schools identified children whom they considered to show poor movement skill for their age (i.e. they demonstrated significant difficulty conducting everyday tasks such as handwriting, using classroom utensils [i.e. scissors, pencils etc.] and/or physical
education activities; as per DSM-IV diagnostic criteria, Criterion B); this was corroborated by physical education teachers. These children were then assessed using the MAND; those scoring below the 10th percentile were included in the DCD group (see Geuze et al., 2001) (Criterion A). Parents were not asked to indicate whether their child had been referred for or were receiving movement therapy. Exclusion criteria were a past or current diagnosis of ADHD or known learning, neurological or physical disorder (Criteria C). As the children were recruited from mainstream primary schools and had not been diagnosed with a learning disorder, they were assumed to have IQ levels within the normal range (Geuze et al., 2001) (Criteria D). Control children were considered to have age-appropriate levels of motor skill by teachers and a MAND total score above the 20th percentile.

Apparatus

Double-step reaching paradigm (DSRP). The DSRP was used to assess online motor adaptation. The Virtools Software Package (Dassault Systemes, France) was used to develop the task display which was presented on a 17-inch LCD touch-screen (TFT, Nexio). The monitor was mounted at an angle of 15° from horizontal on a tabletop which ensured that targets were freely visible throughout the movement. The (black) surface surrounding the monitor was matched to the color of the monitor boarder and visual display to reduce the contrast within the participant’s visual field. The display consisted of a green circle at the bottom centre of the monitor which acted as a ‘home base’ and three yellow possible target locations (each 2 cm in diameter) presented in a semi-circular formation across the top of the screen. These possible targets were spaced 20° apart at the coordinates of -20°, 0°, and 20° with
respect to the ‘home base’. The distance between the centre of the home base and each target was 30cm.

Participants were seated unrestrained on a height adjustable chair in a dimly lit room. Lighting levels did not permit children to receive visual feedback of their moving limb (Farnè et al., 2003). The index finger of their dominant hand was placed on the ‘home base’ which was positioned approximately 5 cm anterior to the umbilicus at the midline. Their non-dominant hand rested comfortably beside the monitor. An assessor stood behind the participant throughout the task to ensure that they remained attentive and provided further instruction when necessary.

**Procedure**

At the beginning of each trial, the ‘home base’ was illuminated; children were instructed to touch and hold the ‘home base’ with their (dominant) index finger. After a random interval of between 500 and 1500 ms [included to control for anticipatory effects] the home base was extinguished, which coincided with illumination of the central target. Children were required to reach and touch the middle of the target as quickly and accurately as possible, until it disappeared. To ensure that visual attention was initially oriented to the same location for each trial, participants were instructed that the central target location would always be lit first, but that the target may jump to one of two other (peripheral) targets during movement (Castiello et al., 1999; Castiello, Bennett & Chambers, 1998). During these jump trials, children were asked to follow and touch the middle of the lit target.

After each successful trial, children were instructed to return to the ‘home base’ and await the next target. Correct touches resulted in a short auditory tone while no sound was emitted for incorrect touches. Where a trial resulted in error,
participants were instructed to continue touching the screen until they did so within
the target boundaries and (hence) heard the tone.

For 80% of the trials, the central target remained illuminated for the duration
of the movement. For the remaining trials (20%), the central target was initially
illuminated, but at the point of finger lift-off from the ‘home base’, the central target
was turned off but re-appeared immediately at one of the two lateral locations (jump
trials). Participants were instructed to reach and touch the target that was currently lit
until the auditory tone was emitted and the target extinguished.

Prior to the commencement of the task, the assessor modeled the instructions
for a jump and non-jump trial. Participants then completed 8 practice trials (6 non-
jump and 2 jump). If the experimenter deemed it necessary, additional practice trials
were administered until it was clear that the participant understood the instructions.
Participants then completed two blocks of 40 trials that were separated by a one-
minute break. Each block consisted of 32 non-jump trials and 8 jump trials (four to
each side) which were presented in a pseudo-randomized order.

Timed dependent measures were: Reaction Time (RT), measured by the time
between target display onset and finger lift-off from the ‘home base’; and Movement
Time (MT), measured by the interval between lift-off and placement of the finger on
the target location. The difference between the mean MT for jump and non-jump
trials (MT_{diff}) was also calculated. Three types of response error were also recorded:
Touchdown Errors (TDE) were recorded when children touched outside the target
boundary, determined by the centre of pressure; Anticipatory Errors (AE) were
recorded for responses that were initiated (i.e., lift off from the home base) before a
target location was illuminated or for RT’s within 150 ms (see Wilson et al., 1997).
Finally, Centre Touch Errors (CTE) were recorded for finger touches that were made
initially to the central target on jump trials. For a graphical representation of the dependent measures, see Figure 2.1.

![Graphical representation of the double-step reach task parameters.](image)

**Figure.** 2.1. A graphical representation of the double-step reach task parameters. *Note.* Reaction Time (RT), Movement Time (MT), Anticipatory Error (AE) was recorded if participants removed their finger from the home base throughout this period, Touch Down Error (TDE) and Centre Touch Error (CTE). The 150 ms marker denotes the RT window within which response are considered anticipatory.

**Design and Analysis**

For each child, mean reaction time (RT) and movement time (MT) and total errors for each index were calculated. The timed measures were based on legitimate responses only; trials for which an error occurred were removed. For jump trials, timed responses were collapsed over target location (i.e. left and right). Preliminary analysis revealed that condition (i.e. non-jump and jump trials) had no effect on total number of anticipatory errors, and hence they were collapsed over jump and non-
jump trials. A criterion of 50% correct responses on jump trials (or 8 out of 16) was deemed a minimum sampling requirement. Of all correct responses, outliers were defined as responses where RT or MT was > +3 or < -3 SD from the child’s mean for that condition. For the error trials and outliers, combined, an average of 24 (31%) and 13 (16%) trials were removed from the DCD and control groups, respectively. Data from five children were not included in the analysis because they were unable to understand the task rules.

Mean RT, MT and TDE data were compared between groups for jump and non-jump trials using separate 2-way ANOVA (2 [Group] x 2 [Condition]). AE and CTE were compared between groups using independent t-tests. Measures of effect size (partial $\eta^2$) were used to temper the results of significance tests. Finally, I also investigated the effects of age on online control within each group. Here I compared MT$_{\text{diff}}$ scores of younger (7-9 years) and older (10-12 years) children within DCD and Control groups.

Data were tested for violations of normality and homogeneity of variance. For both MT and each index of error, data was transformed using a logarithmic transformation. This transformation did not alter the effects of interest, and in light of ANOVA and $t$-tests being robust to violations of the assumptions of normality and homogeneity of variance (Lindman, 1974; Tabachnik & Fidell, 1996), the data was not transformed for the final data set.

**Results**

**Reaction Time**

Mean RT’s (+/- SE) for each group are displayed in Figure 2.2. The 2-way ANOVA on mean RT revealed no significant interaction between group and
condition, $F(1, 42) = 0.60, p = .44$, partial $\eta^2 = 0.01$. Averaged over condition, the mean RT for children with DCD and controls was 553 and 509 ms, respectively, and was not shown to differ statistically, $F(1,42) = 2.75, p = .10$, partial $\eta^2 = 0.06$. The main effect for condition was also not significant, $F(1,42) = 0.65, p = .43$, partial $\eta^2 = 0.01$; averaged over group, mean RT for jump and non-jump trials was 529 and 534 ms, respectively.

![Figure 2.2](image_url) Mean Reaction Time (RT +/- SE) for DCD and control groups.

**Movement Time**

**Analysis of Group Differences.** Mean MT (+/- SE) for each group is shown in Figure 2.3. The 2-way ANOVA on mean MT revealed a significant interaction between group and condition, Wilk's $\Lambda = 0.75, F(1, 42) = 13.84, p = .001$, and large effect size, partial $\eta^2 = 0.25$. Also significant was the main effect for condition, Wilk's $\Lambda = 0.03, F(1, 42) = 1241.98, p = .001$, partial $\eta^2 = 0.97$. The main effect for group was not significant $F(1, 42) = 3.74, p = .06$, partial $\eta^2 = 0.06$. 

69
0.08; averaged over condition, mean MT for the DCD and control groups was 690 and 634 ms respectively.

![Figure 2.3. Mean Movement Time (MT +/- SE) for DCD and control groups.](image)

**Tests of simple effects** showed no group difference for ‘non-jump trials, $t(42) = 0.75$, $p = .46$, partial $\eta^2 = 0.01$; however, children with DCD were shown to be significantly slower on jump trials, $t(42) = 2.89$, $p = .01$, partial $\eta^2 = 0.17$; mean MT for controls was 778 ms for ‘jump trials’ compared with 867 ms for the DCD group. The difference between the mean MT for jump and non-jump trials ($MT_{dij}$) was also compared between groups. The average $MT_{dij}$ score for the DCD group was significantly higher than controls, $t(42) = 3.72$, $p = .001$, partial $\eta^2 = 0.25$, 353 ms compared to 285 ms.

**Analysis of Individual Differences.** Individual mean $MT_{dij}$ scores within each group are presented in Figure 2.4. There appeared to be a distinction between groups at approximately 300 ms with the majority of children with DCD
exceeding this time and most controls under it. The CI95% for Controls was between 263 and 309 ms, compared with 325 and 382 for the DCD group. The MT$_{diff}$ score for 13 out of 17 (76%) of the DCD group exceeded the upper CI95% limit of the Control group (or 309 ms).

Figure 2.4. Mean MT$_{diff}$ scores for DCD (left) and Control children (right).

*Analysis of MT$_{diff}$ scores within each group (DCD and Control).* Independent $t$-test failed to reveal a significant difference for mean MT$_{diff}$ scores between younger (7 to 9 years of age) and older (10 to 12 years of age) control children, $t\ (25) = 1.23$, $p = .23$, partial $\eta^2$=0.06; means were 303 and 275 ms, respectively. Younger children with DCD produced significantly larger MT$_{diff}$ scores than older children with DCD, $t\ (25) = 3.01, p = .01$, partial $\eta^2$=0.38; means were 397 and 322 ms respectively.
Errors

Mean AE and CTE error scores (+/- SE) for each group are displayed in Figure 2.5. The 2-way ANOVA on mean TDE revealed significant main effects for condition, Wilk's $\Lambda = 0.36$, $F (1, 42) = 76.39$, $p = .001$, partial $\eta^2 = 0.65$, and for group, $F (1, 42) = 11.12$, $p = .002$, partial $\eta^2 = 0.21$. The interaction of group and condition was not significant, $F (1, 42) = 0.53$, $p = .47$, partial $\eta^2 = 0.01$. Independent $t$-tests revealed that the children with DCDcommitted significantly more CTEs, $t (42) = 4.31$, $p = .001$, partial $\eta^2 = 0.31$, while there was no differences between the groups on AE, $t (42) = 1.83$, $p = .07$, partial $\eta^2 = 0.07$.

Figure 2.5. Mean number of errors (+/- SE) for each group. Notes: Anticipatory Error (AE), and Centre Touch Error (CTE).

Summary of results

To summarize, 2-way ANOVA revealed that children with DCD showed a trend for slower movement initiation than controls, while no significant effect for condition or interaction for RT was evident. Children with DCD displayed a
significantly longer MT$_{diff}$ score than controls and 2-way ANOVA showed the interaction effect for group and condition for MT data to be significant. No overall group effect for MT was observed; tests of simple main effects showed that the reaching of children with DCD was significantly slower than that of age-matched controls but only for perturbed trials. Children with DCD were also less accurate than controls, committing significantly more CTEs and TDEs than controls. Children with DCD also committed more AE errors, with this effect approaching significance ($p=0.06$).

**Discussion**

Using a double-step reaching paradigm, Study 1 investigated the ability of children with DCD to make ROCs to reaching. It was predicted that children with DCD would show an impaired capacity for making online adjustments to reaching as manifest by significantly delayed movement time and increased errors on jump trials. As predicted, the movement times of these children were significantly prolonged relative to controls, when the location of targets was shifted at the point of lift off. As well, the DCD group committed more movement errors. Children with DCD also displayed a trend for slower movement initiation than controls. The pattern of results suggests that children with DCD show impairment in the ability to correct movement online using a forward (predictive) model of limb trajectory. This view is discussed in detail below, starting with the RT results.
Reaction Time

The absence of any effect for condition (i.e. non-jump vs. jump trials) on RT found in Study 1 was expected since the target perturbation did not occur until after movement lift-off (post RT). Hence task requirements up to the point of lift-off were identical for both conditions; rather, by definition, online control would be exerted after lift-off and maximally in response to target perturbation.

There was a trend for children with DCD to be slower to initiate movement compared to controls ($p= .10$). This finding is in agreement with other work that shows consistently that children with DCD are generally slower to respond to external cues (Henderson, Rose, & Henderson, 1992; Wilson & McKenzie, 1998). Reaction time is one measure used to infer the speed of neural transmission which reflects the integrity of the central nervous system. Others has also made the point that in the case of movement, RT may reflect the time necessary to plan (and initiate) the impending motor command (Desmurget et al., 2004). In DCD, it is possible that the general effect on RT reflects both low transmission times and a deficit in planning. However, this study was not designed to tease this apart, but rather to posit control issues that bear on MT data. This is now discussed.

Movement Time

Like earlier studies using the DSRP (Castiello et al., 1999; Farnè et al., 2003; Plumb et al., 2008), movement time increased significantly for jump trials. This is attributed to two key factors: firstly, the increase in movement amplitude required to complete the task; and secondly, the increase in task complexity related to online correction of movement. Assuming that the initial motor command is accurate, non-jump trials allow reaching to un-fold largely unchanged. While some online
modulation is expected for simple reaching since the motor command is imperfect (Desmurget & Grafton, 2000), demands on this system are minimal. Conversely, in response to target perturbation, discrepancies between the predicted location of the hand (in relation to the target) and its actual location based on the flow of visual sensory information must first be detected and the resultant error signals integrated with the on-going motor command to alter the movement path.

As predicted, the movement times of children with DCD were roughly equivalent to those of controls on trials where target location remained fixed. This finding accords with Wilmut and colleagues (2006) who showed the MT of children with DCD to be almost identical to controls for simple aiming movements. However, Study 1 showed that children with DCD were differentially affected by target jumps, relative to non-jump trials as indicated by the larger $\text{MT}_{\text{Diff}}$ score compared with control children and the presence of a strong interaction effect. Inspection of Figure 2.3 illustrates that the two groups performed similarly on the non-perturbed trials (mean difference = 22 ms) whereas the DCD group showed impaired performance when completing jump trials (mean difference = 90 ms); this pattern was confirmed by an analysis of simple effects. Further, examination of individual differences showed that over 70% of the DCD group were disadvantaged by the jump condition relative to controls. Taken together, these results suggest that although children with DCD were able to plan and perform simple aiming movements as efficiently as their typically developing peers, they were less efficient at correcting their movements online in response to target perturbation. This pattern of results rules out the possibility that the impaired performance exhibited in DCD children may reflect a general motor deficit as such impairment would be expected to affect reaching performance irrespective of trial condition (c.f. Plumb et al., 2008).
The presence of a significant interaction effect on MT stands in contrast to the results of Plumb et al (2008) who also compared children with DCD and controls on a DSRP. Importantly, in Plumb’s study the two groups performed somewhat different versions of this task: control children conducted the task while standing whereas children with DCD were seated and used a larger stylus. In the present study both groups were seated and all other task parameters were identical (i.e. both groups were required to point and touch the target using their dominant index finger). Task modifications of the type used by Plumb and colleagues’ would result in a comparative increase in task complexity for controls by virtue of the added degrees of freedom that must be accommodated when planning a movement from a standing position, with all its postural constraints. Given that the magnitude of condition effects are in the order of milliseconds, any change in task complexity, no matter how small, would likely impact the pattern of results (esp. interactions with group) whether the dependent measure is time or accuracy based. Thus, it is possible that the failure to observe an interaction in Plumb’s study may be due to differences in task complexity between the two groups which obscures an underlying issue with online motor control in DCD.

Again, rapid and efficient online correction in response to target jumps requires children to integrate error signals that are generated when the predicted location of the hand (in relation to the target) fails to match its actual location based on the flow of visual sensory information—an internal feedback loop that enables fast online corrections. The prolonged movement time observed in DCD on jump trials may reflect impairment in the ability to either generate this error signal following target perturbation, or integrate it seamlessly with the on-going motor command. As discussed in Chapter 1, this notion that children with DCD have an impaired ability to
utilise internal models of movement has been canvassed previously—ala the IMD hypothesis (Wilson et al., 1997; Wilson et al., 2001; Williams et al., 2006). While for simple (straight-line) movements in peripersonal space, the motor deficits seen in DCD may not be as apparent, particularly when initial fixation and target location are aligned (as per Wilmut et al., 2006). For targets that are displaced laterally, however, trajectory planning might present more difficulty for these children.

Analysis of age differences within each motor group. There is little data on the development of ROC per se, however goal-directed reaching undergoes significant maturation between the ages of 7 and 12 years. Over this period, the development of reaching follows a non-monotonic trajectory, characterised by an increase in proficiency with a temporary re-organisation of control at around 8 years of age (Hay, 1979; Pellizzer & Hauert, 1996). Indeed, at this age, performance efficiency has been noted to decline (e.g., Pellizzer & Hauert, 1996) and then follow an improving trend after about 9 years of age. Accordingly, I undertook additional analysis to investigate the development of ROC by comparing younger (i.e. 7 to 9 years of age) and older (10 to 12 years of age) children in the control and DCD groups respectively on MT\text{Diff} score. Interestingly, for children with DCD ROC was less developed in younger children compared to older. This suggests that while impairment continues throughout development for children with DCD, they may ‘catch-up’ to typically developing children, at least to a degree, throughout development. For typically developing children, there was no significant change with age on the present measure of online control (c.f. Pellizzer & Hauert). Thus, even younger control children possess a degree of competence with respect to ROC. The lack of difference between younger and older control children on the present measure of online control is at odds with the general observation that movement efficiency and
control improves from the ages of 7 to 12 years. However, as noted above, work on
goal-directed reaching suggests an increase in proficiency at around 9 years of age as
feedback and feedforward mechanisms become better integrated. Inclusion of 9-year-
olds in the “younger group” might therefore dilute the between-group comparison
here as their performance compensates for some of the more immature response
patterns seen in 7-year-olds, for example. Moreover, the slightly greater variability in
the younger control group is consistent with the view that 9-year olds are using a
more sophisticated mode of control than 7 to 8 year-olds, though these age
comparisons are limited by small sample size. These interesting developmental trends
are investigated in Study 3.

Summary. Children with DCD showed a select deficit in making
correction to their reaching in response to unexpected target perturbation which may
reflect difficulty correcting the on-going motor command on the basis of the error
signal which arises following a mismatch between the expected trajectory of the limb
(according to the forward model) and that indicated by sensory in-flow. In short, this
performance profile is consistent a deficit in predictive modeling.

Movement Errors

Touch Down Errors. The number of TDEs committed by both groups
increased significantly for the jump trials. Since task complexity increases during
jump trials, this effect was expected. Interestingly, although the DCD group produced
significantly more TDE than controls overall, no interaction effect was observed. The
general inaccuracy of the DCD group is in line with earlier research (Wilson et al.,
1997). However it was also reasonable to expect that they would be more
disadvantaged than controls on jump trials as online control demands increased; but
this was not the case. Taken together, a reduced ability to implement ROC (as shown in MT data) may bias children with DCD toward a speed-accuracy trade-off when dealing with target shifts: rather than making additional errors, they may compensate for impaired online control by slowing their movements to maintain some semblance of accuracy during jump trials. Interestingly, this type of pattern is also seen in older adults (Shumway-Cook & Woollacott, 2007, pg. 487).

**Centre Touch Errors.** The DCD group also made more CTEs than controls, perhaps suggesting a problem of response inhibition. This type of error implies a failure to suspend or alter trajectory in response to the shift in target location, before a preplanned reach has completely unfolded. In the main, the number of CTEs we see in healthy adult populations and typically developing (older) children are very small (in the order of < 1% of trials). This makes the current result, with moderate effect size, noteworthy. There are two possible explanations for the current group difference. First, since successful correction of movement is dependent on the efficient integration of forward estimates of limb endpoint (relative to target) with the actual sensory estimates, the CTE data could be interpreted as a disruption of predictive control in DCD. However, the average number of CTEs was in order of 2-3 out of 16 jump trials—perhaps too few to draw strong inferences about online control. The alternative explanation is to attribute the higher number of CTEs to a reduced ability to inhibit or modulate responses to what was a highly salient stimulus (the centrally cued location).

Interestingly, as canvassed in chapter 1, difficulties inhibiting shifts of visual attention to compelling cues has been reported previously in DCD (Mandich et al., 2003; Wilmut et al., 2007; Wilson & Maruff, 1999; Wilson et al. 1997). Most of this data is drawn from the COVAT, or variant thereof. The work of Wilson and
colleagues, in particular, showed that children with DCD manifest deficits in the ability to disengage voluntary attention from invalidly cued locations. Others have detected similar patterns of performance but interpreted the results somewhat differently, implicating inhibitory control per se (e.g., Mandich and colleagues). The co-occurrence of inhibitory and movement skill problems has been well documented in both younger (Livesey et al., 2006) and older (Piek et al., 2007) children. And the overlap is even greater in children with co-morbid DCD and ADHD (Sergeant, Piek, & Oosterlaan, 2006). It is likely that the ability to modulate action planning by inhibitory control might be reduced in both groups, placing limits on motor learning (see also Barkley, 1997). Developmentally, we simply need more longitudinal data to unravel the dynamic relationship between inhibition and movement control in children.

**Anticipatory Errors.** Interestingly, children with DCD also tended to make more anticipatory errors (although this difference just failed to reach significance, $p = .07$). This type of error represents the ability to maintain sustained attention and to prevent responses before locations are cued, rather than the ability to actively disengage attention from invalid cues, as per the COVAT. However, I return to the possibility that inhibitory control may be compromised to some extent in DCD, at least to the extent that it concerns organization of a movement response to spatial targets. In other work, Piek and colleagues (2007a) have found that children with DCD were poorer on a range of executive tasks that measured response inhibition, working memory and set-shifting—they were slower and more variable. It remains to be seen whether deficits of this type also impact more strategic aspects of executive control (e.g., error monitoring). Taken together, results for measures of error highlight the need for further investigation of the role of inhibition in DCD.
symptomatology, as well as the particular role, if any, it plays in impaired online control.

**General Discussion**

Study 1 has shown that children with DCD display a select impairment in making rapid online corrections to reaching as manifest by delayed MT and increased error during double-step reaching. From a computational perspective, this pattern of performance may reflect impairment in generating the error signal which arises on jump trials when the expected sensory consequences of movement (according to the predictive model) differ from those actually experienced, or integrating this signal seamlessly with the on-going motor command.

Intriguingly, there are similarities between the pattern of double-step reaching demonstrated by children with DCD in Study 1 and adults with optic ataxia, a disorder associated with damage to the PPC that results in difficulties making rapid online adjustments to reaching (Glover, 2003; Pisella et al., 2000). It has been suggested that this difficulty might reflect a decreased ability to integrate sensory feedback with the efference copy (as per predictive modeling) (see Glover, 2003). Similarly to children with DCD in the present study, patients with PPC lesions display increased movement time following unexpected target perturbation yet relatively preserved simple reaching (Grèa et al., 2002). The issue of the possible neural basis of impaired ROC in children with DCD is elaborated on in the coming chapters, particularly chapter 5.
Limitations

As I highlighted in the introduction of this chapter, Study 1 was designed to assess rapid online motor control in DCD as a probe to the mechanisms subserving impaired motor control in the disorder. Hence, I acknowledge the limitations of using chronometric data in isolation of kinematic data to examine mechanisms of motor control. Although global measures of performance like MT and errors can provide useful information about the integrity of control mechanisms, they provide little information about the subtle changes in control that are shown at different points in the movement cycle. Specifically, from chronometric data alone it is not possible to determine whether poor double-step reaching in children with DCD results from control issues early in the reaching trajectory (where demands on online control are greatest since large-scale trajectory corrections are implemented here) or later (where demands on online control are comparatively low since the target remains stationary after trajectory corrections have been implemented). That said, the large effect size observed for the interaction between group and condition on MT, together with the absence of any group effect on RT, does support the suggestion that the rapid online adjustments necessary to maintain speed and efficiency of movement are compromised in DCD. Nonetheless, these results pave the way for exploring the kinematics of ROC in DCD.

Summary

Study 1 showed that children with DCD display a reduced ability to adapt their movement online in response to target perturbation, manifest as delayed movement time and increased response error. This pattern of performance was consistent with the view that the impaired ROC found in DCD may, at least partly, be
due to an impaired ability to correct the ongoing motor command using predictive (forward) models of movement (Desmurget & Grafton, 2003). Notwithstanding some of the limitations of chronometric data, results presented here clarify some inconsistencies in the DCD literature surrounding the nature of ROC in DCD and lay the foundation for more rigorous kinematic analysis which is presented next, in Study 2.
CHAPTER 3

STUDY 2: DOUBLE-STEP REACHING PERFORMANCE IN CHILDREN WITH DEVELOPMENTAL COORDINATION DISORDER: KINEMATIC ANALYSIS OF REACHING
Introduction and Overview

In Chapter 2, chronometric analysis of double-step reaching showed that children with DCD display a reduced capacity for making rapid online correction to reaching, as demonstrated by increased movement time and decreased accuracy during jump trials. I argued that this pattern of performance may reflect an impaired ability to use predictive models of limb trajectory to correct the unfolding movement—ala predictive (internal) modeling.

While chronometric analysis provides important information about the global properties of control mechanisms, one major limitation of chronometric analysis alone is that it does not allow one to dissect the subtle transitions in motor control that occur at different time points in the movement cycle—i.e. the question of whether control parameters are affected early or late in the movement trajectory. On jump trials, due to time delays associated with processing non-visual and visual sensory feedback, reaching is thought to rely heavily on predictive control during the early phase of movement, up to the point when early kinematic markers are expressed (i.e. time to peak acceleration and velocity: tPA and tPV). These early markers together with the first detectable change in movement trajectory are thought to reflect the integration of real-time sensory feedback with the ongoing motor command. Though online control is exerted over the entire movement cycle, demands on this system are maximal during the early phase of double-step reaching when the larger scale changes in trajectory are implemented in response to target perturbation, and reduced during the later (post-correction) phase of reaching which serves mainly to brake the limb as it captures the new target location (see Wolpert & Flanagan, 2001). During the latter
phase, there would be minimal error between the expected and actual sensory consequences of movement because the target simply does not move from its second location. Chronometric analysis (i.e. movement time) does not allow one to dissect the type of control exerted during early and later stages of movement.

Other work using the double-step task has revealed distinct patterns of deficit based on early kinematic makers. In patients with optic ataxia, for instance, corrections to the reach trajectory after target perturbation occur significantly later than in healthy adults suggesting difficulties using internal feedback control to update the ongoing motor command (Gréa et al., 2002). Desmurget and Grafton argue that the PPC and its reciprocal connections to the cerebellum may support these early corrections. It is thought that predictive models for limb position may be generated and/or monitored at the level of PPC (see Desmurget & Sirigu, 2009). These forward estimates enable the system to respond rapidly if self-to-target relations change during the course of a movement, as when targets shift their location. Indeed, the PPC is thought to be the site where comparison between the expected location of the limb (with respect to the target) and that indicated by actual sensory inflow occurs, and the error signal generated (Desmurget et al., 1999).

From a neuro-computational perspective (e.g., Desmurget & Grafton, 2003; Desmurget & Sirigu, 2009; Wolpert, Ghahramani, & Flanagan, 2001) rapid online correction is thought to be dependent on two processes: the first concerns the capacity of the motor system to monitor the presence of any error between the predicted limb trajectory (according to a forward model) and actual limb position based on sensory inflow; and second, the integration of a resultant error signal with the ongoing motor command to alter trajectories in-flight and with minimal time lag. Importantly, these control processes can be inferred from kinematic landmarks in reaching. Adult data
for double-step reaching show that early kinematic markers (i.e. tPA and tPV) occur earlier for jump compared with non-jump trials (Castiello et al., 1991; Farnè et al., 2003; Paulignan et al., 1991) suggesting very rapid online adjustments [i.e. at around 100 ms, too fast for sensory feedback control alone which can take upwards of 250 ms (Frith, et al., 2000)]. Earlier braking is then correlated with the subsequent change in trajectory (i.e. Farnè et al., 2003) which occurs some 150-200 ms after these initial landmarks. Thus, tPA and tPV may mark a period during which discrepancy between the expected and actual sensory consequences of movement is initially detected (i.e. Farnè et al., 2003; Paulignan et al. 1991). Fast visual channels associated with dorsal-dorsal routes are likely to support this (see Pisella, Binkofski, Lasek, Toni, & Rossetti, 2006). This information must then be used to re-calibrate the movement and adjust the ongoing motor command; the earliest detectable change in trajectory toward the new target is thought to represent this point (Dubrowski et al., 2002; van Braeckel, Butcher, Geuze, Stremmelaar, & Bouma, 2007). Dissociation between the error detection and trajectory correction processes is further highlighted by evidence from optic ataxic patients who experience difficulty correcting ongoing movement following target perturbation (Gréa et al., 2002) despite a preserved capacity to detect error (Pisella et al., 2000).

Summary. Thus, the broad aim of Study 2 was to investigate ROC in children with DCD using both kinematic and chronometric analysis of double-step reaching. More specifically, I hoped to break down the kinematic analysis into earlier and later phases of processing in order to isolate the different control parameters that might explain the slower and less accurate double-step reaching performance in DCD. Given the fundamental impairments that have observed in DCD on other aspects of predictive control (e.g., double-step saccades, reported in Katschmarksy et al., 2001),
I predicted that performance deficits would manifest on early markers of double-step reaching where demands on this system are maximal.

**Method**

**Participants**

The sample consisted of 13 children (9 girls and 4 boys) between the ages of 8 and 12 years of age who met the research criteria for DCD and 13 age-matched controls (6 girls and 7 boys) who were not part of the Study 1 sample. Mean age for the DCD group was 10.5 years ($SD= 1.7$) and 10.3 years ($SD= 1.4$) for the controls. All children were right-hand dominant with the exception of 4 children in the DCD group. Children were screened using the same procedure adopted in Study 1 (see Chapter 2) that was approved by the RMIT HREC and Research Branch of the Victorian Government Department of Education and Early Childhood Development.

**Apparatus**

**Double-step reaching paradigm (DSRP).** The apparatus display was the same as that adopted for Study 1 (see Chapter 2). Participants wore a thin polyester glove on their reaching (i.e. dominant) hand. An electromagnetic sensor was attached to the position of the fingernail of the index finger of the glove using Velcro; the underside of the gloves index finger was removed to maximize tactile feedback. Kinematic data for reaching was recorded using the Flock of Bird (FOB) motion tracking system (Ascension, VT, USA) sampled at 100Hz. Raw data was converted into three dimensional coordinates ($x,y,z$), with the $y$-axis representing the distance component of movement, $x$-axis the direction component, and $z$-axis the depth component. Acceleration, velocity and reaching trajectory profiles for each trial were
recorded from this data. See Figure 3.1 for typical non-jump and jump reaching plots for children with DCD and age-matched controls.

![Diagram of reaching trajectories](image)

*Figure 3.1.* A typical reaching trajectory plot from a control (left) and DCD (right) child for non-jump (dashed line) and jump trials (solid line).

**Procedure**

The procedure was the same as that adopted for Study 1, as were the chronometric measures (see Chapter 2).

Four kinematic measures were recorded: time to Peak Acceleration (tPA); time to Peak Velocity (tPV), Time to Correction of Movement Trajectory (TC), and Post-correction Time (PCT). TC was measured as the time of movement correction away from the initial (central) target towards the correct target during ‘jump’ trials. This was determined manually using a 2D (x by y) representation of each reaching trajectory for jump trials to identify the time at which the hand deviated away from its
(virtually) straight-line path and toward the cued peripheral target (as per Pisella et al., 2000; van Braeckel et al., 2007). Negligible pronation and supination of the lower arm occurs during the course of such movement; hence, there was no need to adjust these measures. PCT was determined for ‘jump’ trials as the time elapsed from TC to the completion of movement.

**Design and Analysis**

Kinematic data were filtered off-line using a dual-pass Butterworth second order filter with a cut-off of 10 Hz. For jump trials, timed responses were collapsed over target location (i.e. left and right). For each child, mean values were recorded for each dependent measure; trials on which errors occurred were counted but not included in the analysis of chronometric and kinematic data. Preliminary analysis revealed that condition had no effect on total numbers of AE committed and hence they were collapsed over ‘jump’ and ‘non-jump’ trials. For inclusion in parametric analysis, all children met a minimum of 50% correct responses on jump trials (or 8 out of 16). For each dependent measure, outliers were defined as responses > +3 or < -3 SD from the child’s mean for that condition. An average of 24 (30%) and 17 (21%) trials were removed from the DCD and control groups, respectively.

Separate 2-way ANOVA (2 [Group] x 2 [Condition]) were conducted on mean values for RT, MT, tPA, tPV and TDE. Independent t-tests were used to compare groups on AE, CTE, MTdiff. TC and PCT. Measures of effect size (partial $\eta^2$) were used to temper the results of significance tests.
Results

Chronometric Analysis

Reaction Time. Mean RTs (+/- SE) for each group are displayed in Figure 3.2. The 2-way ANOVA on mean RT revealed no significant interaction effect between group and condition, $F(1, 24) = 0.19, p = .66$, partial $\eta^2 = 0.01$. Averaged over condition, the mean RT for the DCD group (572 ms) was significantly longer than the control group (494 ms), $F(1, 24) = 9.87, p = .004$, partial $\eta^2 = 0.29$. The main effect for condition was not significant, $F(1, 24) = 0.01, p = .93$, partial $\eta^2 = 0.000$; averaged over group, mean RT for ‘jump’ and ‘non-jump’ trials was 533 and 532 ms, respectively.

![Figure 3.2. Mean Reaction Time (RT +/- SE) for DCD and control groups.](image)

Movement Time. Mean MT’s (+/- SE) for each group are shown in Figure 3.3. The 2-way ANOVA on mean MT revealed a significant interaction between group and condition, Wilk’s $\Lambda = 0.69, F(1,24) = 10.52, p = .003$; and large effect size, partial $\eta^2 = 0.31$. Also significant was the main effect for condition, Wilk's $\Lambda = 0.04,$
\[ F (1, 24) = 619.35, p = .001, \text{ partial } \eta^2 = 0.96, \] while that for group was not, \( F = 1.23, p = .28, \text{ partial } \eta^2 = 0.05. \)

Tests of simple main effects failed to show a significant group difference on non-jump trials, \( t (24) = .42, p = .68, \text{ partial } \eta^2 = 0.01, \) but children with DCD (885 ms) were significantly slower on ‘jump’ trials than control children (816 ms), \( t (24) = 2.10, p = .05, \text{ partial, } \eta^2 = 0.15. \) The mean MT difference score for the DCD group (338 ms) was significantly higher than controls (260 ms), \( t (24) = 3.24, p = .003, \text{ partial } \eta^2 = 0.31. \)

**Analysis of Individual Differences.** Individual differences on MT difference scores for each group are shown in Figure 3.4. There appeared to be a distinction between groups at approximately 300 ms: most children with DCD exceeded this time while most controls did not. The CI\(_{95}\%\) for the DCD group was between 302 and 373 ms compared with 225 and 295 ms for controls. Nine of the 13 children with DCD (or 69\%) exceeded the upper CI\(_{95}\%\) of the Control group (i.e. 295
ms) while 3 of the 13 control children (or 23%) exceeded the lower CI$_{95\%}$ of the DCD group (i.e. 302 ms).

![Figure 3.4. Mean MT$_{diff}$ scores for DCD and Control children.](image)

**Kinematic Analysis**

**Time to Peak Acceleration.** The 2-way ANOVA on mean tPA revealed no significant interaction effect between group and condition, $F (1, 24) = 0.14$, $p = .71$, partial $\eta^2 = 0.01$. Averaged over condition, the mean tPA for DCD children and controls (176 and 160 ms, respectively) did not differ statistically, $F = 1.50$, $p = .23$, partial $\eta^2 = 0.06$. The main effect for condition was also not significant, $F = 0.75$, $p = .34$, partial $\eta^2 = 0.03$; averaged over group, mean tPA for ‘jump’ and ‘non-jump’ trials was 171 and 166 ms, respectively.

**Time to Peak Velocity.** The 2-way ANOVA on mean tPV revealed no significant interaction between group and condition, $F = .79$, $p = .38$, partial $\eta^2 = 0.03$. Averaged over condition, the mean tPV for DCD children and controls (190 and 178 ms, respectively) did not differ statistically, $F = 0.55$, $p = .46$, partial $\eta^2 = 0.02$. The
main effect for condition was also not significant, $F = 1.29, p = .27$, partial $\eta^2 = 0.05$; averaged over group, mean RT for ‘jump’ and ‘non-jump’ trials was 182 and 186 ms, respectively.

**Time to Correction (TC).** Mean TC (+/− SE) for each group is displayed in Figure 3.5. Independent $t$-test revealed that children with DCD took significantly longer to initiate movement correction on ‘jump’ trials than control children, $t (24) = 3.49, p < .01$, partial $\eta^2 = 0.34$.

![Figure 3.5. Mean Time to Correction of Movement Trajectory for DCD and Control children.](image)

**Post-Correction Time (PCT).** Independent $t$-test showed no significant difference in time spent in the declarative phase of ‘jump’ trials for children in the DCD and Control groups, respectively, $t (25) = 1.60, p > .05$, partial $\eta^2 = 0.08$. 
Errors

Averaged across condition, children with DCD (mean = 15.8) committed significantly more TDEs than controls (10.2), $F(1, 24) = 4.76$, $p = .04$, partial $\eta^2 = 0.17$. The 2-way ANOVA on TDE failed to reveal a significant effect for condition, $F(1, 24) = 1.24$, $p = .28$, partial $\eta^2 = 0.05$; nor was the condition by group interaction significant, $F(1, 24) = 0.11$, $p = .74$, partial $\eta^2 = 0.01$. Finally, there were no significant group differences on AE, $t(24) = 0.19$, $p = 0.85$, partial $\eta^2 = 0.002$ (group mean for DCD = 1.69; control = 1.84), or CTE, $t(24) = 1.90$, $p = 0.07$, partial $\eta^2 = 0.13$ (group mean for DCD = 1.77; control = .61).

Summary of results.

To summarize, 2-way ANOVA showed that children with DCD were significantly slower to initiate movement than age-matched controls; no effect for condition or interaction was shown. While children with DCD completed non-jump reaching as efficiently as control children they were significantly slower to account for unexpected target perturbations; shown by a significant interaction effect on MT and significantly larger $MT_{\text{Diff}}$ scores. 2-way ANOVA for kinematic variables failed to show group differences for tPA or tPA nor were condition or interaction effects on these metrics found to differ. Children with DCD were significantly slower to correct the trajectory of their reaching away from the initial target on jump trials (shown by significantly slower TC; no differences were observed for PCT. 2-way ANOVA showed that trial condition had no effect on number of TDEs committed; averaged over condition, children with DCD made significantly more TDEs than controls. While no group differences were observed for mean AEs committed, children with DCD showed a non-significant trend for increased CTEs ($p = .07$).
Discussion

In Study 1, chronometric analysis of double-step reaching in children with DCD showed that these children suffer from a reduced ability to make rapid online corrections, demonstrated by slower and less accurate reaching on jump trials. However, it was not possible to determine whether the motor control issue was expressed early or late in the reach trajectory. The kinematic study presented in Study 2 was designed to isolate early markers of online control that indicate use of forward estimates of limb position during double-step reaching.

As predicted, chronometric data showed that children with DCD were generally slower to initiate movements compared with controls, an attribute of movement that has been reported repeatedly (e.g., Henderson et al., 1992; Wilson & McKenzie, 1998). Importantly, their reaching movements were significantly slower and less accurate on trials when the target unexpectedly jumped at reach onset. Kinematic data presented here clarifies the underlying nature of this impairment. On jump trials, reaching is thought to rely heavily on predictive modeling during the early phase of movement, which is expressed as reduced tPA and tPV compared with non-jump trials. Interestingly, no group differences were observed on tPA or tPV. However, children with DCD were significantly slower to correct the trajectory of their reach away from the initial target location on jump trials (given by TC). The TC metric indicates the point at which fast internal feedback signals are integrated with the ongoing motor command (i.e. Dubrowski et al., 2002; van Braeckel et al., 2007). This process of rapid modification in limb trajectory occurs within the order of 250-350 ms in adults and can be considered a crucial outcome of motor prediction (Desmurget & Grafton, 2000). By comparison, no difference between groups was observed on PCT where demands on online control are reduced because of the
elapsed time since target displacement. Taken together, this pattern of results suggests impairment in DCD of the ability to implement ROC in response to target perturbation. Next, this hypothesis is discussed in detail.

**Chronometric Analysis**

**Reaction Time.** Consistent with the results of Study 1, no effect for condition (i.e. non-jump vs. jump) on RT was observed. Again, this result was expected since target perturbation occurred after movement initiation: RT was recorded prior to this point and thus the task requirements were equivalent for both trial types. For similar reasons I did not expect any interaction between group and condition on RT.

The presence of a significant group effect for RT was expected and replicates Study 1 and previous research demonstrating slower initiation of movement towards external stimuli in DCD (e.g., Henderson, et al., 1992; Wilson & McKenzie, 1998). Slower RT to external cues suggests either slower processing speed, inefficient preparation of movement, or both (Desmurget et al., 2004). Though neither Study 1 or 2 were designed to address this broad issue, there is some reason to believe that the general visuomotor processing issue in DCD is due to immaturity in the development of motor networks, both afferent and efferent (see also Sigmundsson, 2003; Gilger & Kaplan, 2001).

**Movement Time.** Like Study 1, when averaged across group movement time (MT) increased significantly for jump trials relative to non-jump trials. As discussed in Study 1, this is largely attributable to the increase in movement amplitude and task complexity associated with double-step reaching compared to non-jump trials - see Chapter 2 for a discussion.
As I predicted here and demonstrated in Study 1, there was no difference in movement time between groups for non-jump trials or simple reaching (see also Wilmut et al., 2006). While simple reaching to a stationary target requires a degree of online control, demands on movement predictive modeling are assumed to be minimal for actions of this type. By comparison, children with DCD showed specific impairment when adjusting their reaching following unexpected target perturbation (see Figure 3.3); this was shown by the strong interaction effect between group and condition on MT and the significantly higher $MT_{\text{Diff}}$ scores in the DCD group. Further, an analysis of individual differences showed that around 70% of the DCD group were unduly constrained by target perturbation- i.e. their mean $MT_{\text{Diff}}$ scores were above the 95% CI of controls. In short, this pattern of results is entirely consistent with the results of Study 1 and that of others who show that children with DCD experience difficulties controlling movements that are perturbed. For example, Volman & Geuze (1998a) showed that children with DCD had difficulty re-establishing a pattern of synchronous tapping upon mechanical perturbation to the finger. This behavioural pattern is thought to reflect difficulty organising dynamic control of movement as a consequence of less stable coordination patterns. Though the authors did not attribute this difficulty to a neural substrate, motor timing issues are the hallmark of cerebellar impairment (Ivry & Keele, 1989). The cerebellum is also thought to play a crucial role in online control of reaching, monitoring in real time the somatic consequences of action and any discrepancy between the expected dynamics of the limb and those indicated by sensory inflow (Miall & King, 2008). Thus, there is evidence that the cerebellum might play a role in the difficulties that children with DCD (or a sub-group thereof) show in adjusting action upon external perturbation (this issue is discussed in detail below). To summarise, on the basis of
chronometric data alone, here there is tentative support for the general conclusion that DCD is linked to problems using predictive models as a means of implementing rapid online adjustments. This is also consistent with data from Wilmut and colleagues who show that these children foveate sequential targets longer prior to initiating hand movements, suggesting difficulties with predictive control. However, it is to the kinematic data that I now turn in order to shore up this hypothesis.

**Kinematic Analysis**

**Early control parameters.** Unlike performance from healthy adults, there was no significant difference between jump and non-jump reaching for either group on tPA or tPV, and no overall group difference (see also Plumb and colleagues, 2008). In adults, tPA and tPV on jump trials (at around 100 ms) occurs earlier than that on non-jump trials (Castiello et al., 1991; Farnè et al., 2003; Paulignan et al., 1991) and precedes the actual redirection of the limb by about 150-200 ms. Reduced tPA, for example, implies some motor consequence of the target displacement—more specifically, the process of error detection per se (Farnè et al., 2003; Paulignan et al., 1991). However, additional time is then needed to integrate any error signal with the ongoing motor command which ultimately results in a change in limb trajectory, which is inferred from TC (Dubrowski et al., 2002; van Braeckel et al., 2007).

The pattern of performance observed in Study 2 suggests that the powerful internal feedback loops which support very early error detection in adults may still be unfolding in primary-school-aged children. This hypothesis is supported by the only other developmental study of online control using a DSRP which also failed to show a condition effect on tPA or tPV in 7-10 year olds (van Braeckel et al., 2007). However, on jump trials in this earlier study, the target disappeared 100 ms after movement
onset and re-appeared in the new location 120 ms later. Hence, the task used by van Braeckel may not fully capture the very early and rapid mechanisms Study 2 is tapping into.

Slower TC in children with DCD may suggest a reduced ability to integrate internal feedback signals with the ongoing motor command (van Braeckel et al., 2007). Current consensus suggests that the capacity to use this control system in an adult-like fashion emerges between the ages of 8 and 12 years in typically developing children (Hay, 1979; Hay & Redon, 1999; Pellizzer & Hauert, 1996). Van Braeckel showed that 63% of typically developing children aged between 7 and 10 years met predefined accuracy demands by completing double-step reaching in a single movement (i.e. without touching the initial target). Temporal values for TC in this study cannot be compared directly to ours or to the adult data due to variations in the task (described above). Though no work has specifically compared the double-step reaching of healthy primary school-aged children with adults, the mean TC score of control children in Study 2 (M = 328 ms) is at the lower end of the distribution of values for healthy adults. For target jumps that are coincident with finger/hand lift-off, some of the range of values reported for adults include the following: 280-330 ms (Dubrowski et al., 2001); 255-295 ms (Paulignan et al., 1991), 238-264 ms (Rossit & Harvey, 2008), and 339 ms (Sarlegna, 2006); It should be noted that participants in the Sarlegna study were instructed to reach at a ‘comfortable’ speed (rather than as quickly and accurately as possible), which may account for slower TC. Thus, while a degree of proficiency is evident in the rapid online corrections of the present control group, the system appears to undergo further refinement over adolescence and early adulthood. Whether the performance pattern of children with
DCD reflects deviance from the typical developmental trajectory or an immaturity of sorts is unclear from this data.

Recent neurophysiological data from studies of human and non-human primates has clarified the specific neural networks that support predictive control, particularly those involved in saccade planning and for rapid adjustments of limb trajectory (Blakemore & Sirigu, 2003). The role of PPC in predictive control (particularly in state estimation) has been well documented for goal-directed movements to visually defined targets. It has been shown, for example, that receptive fields in frontal eye field and PPC are updated in anticipation of a saccade (Rizzolatti, Riggio, & Sheliga, 1994). This forward estimate of saccade direction appears to provide a spatial (or egocentric) frame for planning limb movements (Ariff, Donchin, Nanyakhara, & Shadmehr, 2002). For target-directed reaching, the parietal cortex contributes to state estimation by integrating dynamic visual inputs that signal changes in the environment with forward estimates of the state of the limb and visual environment (Archambault, Caminiti, & Battaglia-Mayer, 2009; Shadmehr & Krakauer, 2008). In other words, when a visual target jumps, the unexpected sensory information must be integrated with the output of the forward model, otherwise the reach will continue along its original (but now redundant) path. The upshot is that output signals from PPC provide crucial error information that is then used by motor cortex to alter motor commands to the moving limbs. My data suggest that the process of integration may be impaired in DCD.

Intriguingly, the kinematic pattern of results observed in children with DCD is very similar to that seen in optic ataxia, a neuropsychological condition known to impact ROC (Gréa et al., 2002) (see also chapter 2). Here tPA and tPV during double-step reaching was similar to healthy controls yet patients were far slower to correct
the trajectory of their reaching following target perturbation suggesting difficulties integrating error information into a corrective online response. This account of impaired ROC in optic ataxia is supported by evidence showing that patients are able to terminate movement in response to unexpected target perturbation as quickly as healthy controls (Pisella et al., 2000) - suggesting preserved error detection - yet cannot amend movement trajectories to a displaced target. This work supports a growing body of evidence that demonstrates similar performance profiles between children with DCD and patients with PPC damage on measures of predictive modeling [i.e., motor imagery ability, assessed using mental limb rotation (Williams et al., 2006; Williams et al., 2008), the visually guided pointing task (Wilson et al., 2001) and double-step saccade task (DSST) (Katschmarsky et al., 2001).

While there is some converging evidence that “delay” in maturation of the PPC may explain the impairments in predictive control that we see in DCD, other work suggests that the cerebellum may also be implicated. For example, Hill and Wing (1999) infer deficits in predictive control based on evidence of impaired grip force modulation in DCD. When lifting an object vertically, a child with DCD was shown to increase grip force earlier than controls. But also, in response to load perturbation, grip-force changes occurred later in DCD. Similar results have been observed by Pereira and colleagues (2001), with pronounced deficits in grip-force modulation seen in a sub-group of children with DCD. The pattern of deficit here is similar to that seen in cerebellar patients (see Wolpert, Miall, & Kawato, 1998). Finally, and more recently, as noted in chapter 1, Kagerer and colleagues (2006) have shown impaired visual-motor adaptation in DCD using prism displacement. Using a centre-out pointing task involving a 60-deg rotation of visual feedback, typically developing showed strong adaptation effects after prolonged training regardless of
whether an abrupt or gradual change in visual feedback was used. Children with DCD, however, showed no after-effects when exposed to gradual changes in feedback. This suggests a reduced ability to detect and correct movement errors over repeated trials—in other words, a problem updating an internal model for the pointing movement. Again, the pattern of performance resembles that seen in cerebellar dysfunction (Miall & King, 2008). The neural basis of predictive modeling concerns in DCD is elaborated on in Chapter 5.

**Late control parameters: PCT.** The lack of group difference on PCT indicates that temporal control during the latter stages of reaching is relatively preserved in DCD. This is based on the assumption that once an initial adjustment to the movement trajectory is made in response to the target jump, demands on ROC are minimal for the remainder of the movement (see Wolpert & Flanagan, 2001). This argument is supported by observation that the accuracy of double-step reaching by adults is not affected by target perturbation (Castiello et al., 1991; Paulignan et al., 1991; Sarlegna, 2006); similarly, there was no effect of condition on TDE in the present study. Further, visual inspection of reaching trajectory plots for each of the groups in the present study showed that once the initial corrections to trajectory were implemented in response to the target jump, movement followed a (virtually) straight-line path, like that seen on non-jump trials (where demands on ROC are also minimal). Reach trajectories of a similar shape on jump trials have been observed for healthy younger (Castiello et al., 1991; Farnè et al., 2003; Sarlegna, 2006) and older adults (Sarlegna, 2006; Sarlegna, Ziviani, Watter, Ozanne, Woodyatt, & Springfield, 2006), and right brain-damaged patients (Farnè et al., 2003). Taken together, data suggest that although children with DCD take longer to implement changes in trajectory (in response to perturbation), they are able to then capture targets with
reasonable efficiency. Both the post-correction phase of jump trials and simple reaching under no-jump conditions are characterised by relatively low demands on ROC. On balance there is support for the notion that simple reaching is age-appropriate in DCD under stable environmental conditions.

**Summary of kinematic findings.** Taken together, data is consistent with the broad hypothesis that ROC - which relies on predictive estimates of limb position - is impaired in children with DCD. Specifically, slower correction of reaching trajectory away from the initial target on jump trials may reflect difficulty integrating information about the target perturbation with the ongoing motor command. By comparison, control of the latter stages of movement (where predictive modeling demands are minimal) appears to be preserved.

**Errors**

**Anticipatory errors.** No significant group difference was observed on AE suggesting that children with DCD were able to inhibit inappropriate movement and/or maintain finger contact with the home base as well as control children (see also Study 1). It is noteworthy, however, that others have reported inhibition problems in DCD using tasks of executive function (e.g., Piek et al., 2007a). The particular parameters under which these problems might manifest is a topic of further investigation.

**Touch Down Errors.** As predicted, averaged across condition, children with DCD committed significantly more TDEs when compared to controls. This result is in line with previous research showing decreased accuracy in DCD (Smits-Engelsman et al., 2003) as well as Study 1 of this thesis.
Centre Touch Errors. Children with DCD showed a trend for committing significantly more CTEs, though the effect was not significant ($p = .07$); this finding again accords with Study 1. CTEs reflect an individuals’ capacity to correct an initiated motor response in following environmental change in a timely fashion. Accordingly, these results suggest that children with DCD experience difficulty correcting or suspending movement in response to target perturbation prior to the initial motor command unfolding completely. To re-iterate, I argued in Study 1 that there were two potential explanations for this pattern of performance: firstly, since efficient correction of reaching in response to a target jump requires the seamless integration of predictive models of movement with feedback based mechanisms, the increase in CTEs seen in children with DCD may reflect a deficit in integrating predictive and feedback based mechanisms (ala the IMD hypothesis). However, the average number of CTEs committed was roughly 2 out of 16 per child with DCD, limiting the capacity for drawing inferences about online control. The second, and more likely explanation for the increased CTEs seen in children with DCD, is that they may suffer from an impaired ability to inhibit responses to compelling (though inappropriate) stimuli (see Chapter 2 for a detailed analysis).

Limitations

There is now compelling evidence for impaired ROC in children with DCD. Establishing whether this deficit reflects a developmental immaturity or deviance from the typical developmental trajectory is important to our understanding of the role of impaired ROC in poor motor skill and the future development of remedial efforts. For example, in the case of developmental immaturity, it is assumed that children with DCD have the capacity to ‘catch-up’ to their same-age peers and hence remedial
effort should focus on assisting children with DCD attain age-appropriate levels of motor function (van Braeckel, Butcher, Geuze, Bos, & Bourma, 2010). Conversely, in the case of deviance, it is assumed that impairment will persist (McConnell, 1998; van Braeckel et al., 2010) and thus remediation would be better directed at the development of compensatory strategies so that impairment is minimised (Miller & Bachrach, 2006). Deviance versus immaturity can be established using well-designed cross-sectional studies comparing the motor performance of children with DCD and younger controls. If the performance of children with DCD is of a similar profile and level to that of younger (neurologically immature) controls, then developmental immaturity is inferred. Alternatively, if the pattern does not fit along a normal developmental trajectory, then deviance is inferred. This method, for example, has been used to establish developmental immaturity as the likely explanation for impaired postural control in DCD (Wann, Mon-Williams, & Rushton, 1998), praxis skills (Hill, Bishop, & Nimmo-Smith, 1998) and COVAT (Wilmut et al., 2007).

Further, as discussed, similarities between the abnormal performance of DCD children and PPC patients during ROC were noted. However, where children with DCD appear to demonstrate less accurate and efficient online control compared to control children, PPC damage results in far slower TCs (i.e. mean TC = 516 ms vs. 378 ms) and often an inability to correct movement online at all (Gréa et al., 2002). Thus, despite performance similarities between children with DCD and PPC patients on the double-step task, the deficits displayed by the latter are clearly more severe. Hence, I do not propose that the two disorders share exactly the same performance profile or that their performance is explained by the same neuro-cognitive mechanism—caution is needed when comparing the two populations. However, there is some evidence provided here from performance similarities between children with
DCD and PPC patients during the double-step task that the PPC, or the parieto-cerebellar axis more generally, may be involved (at some level) in impaired ROC (and hence predictive modeling) in DCD.

Finally, given variability in the presentation of DCD, it cannot be asserted that a deficit in predictive control explains all cases of DCD. Indeed, a small sub-group (i.e. 3/13) of children with DCD in the present sample demonstrated preserved double-step reaching suggesting functional predictive modeling, at least to an age-appropriate standard. What can be said is that there is good evidence to suggest that most children with DCD have difficulties implementing forwards models, and that this affects their ability to alter movements in-flight. Whether these difficulties reflect a developmental delay is addressed in Study 3 which is presented in the next chapter.

**Summary of Study 2.**

Study 2 replicated the earlier chronometric analysis of double-step reaching in DCD presented in Study 1: impaired ROC was indicated by increased movement time on jump trials. Kinematic data allowed me to further isolate the locus of impairment in DCD. Early kinematic markers (tPA and tPV) failed to show any group differences. However, children with DCD were significantly slower to correct the trajectory of their reaching away from the initial target on jump trials. No abnormalities were shown for the post-correction phase of jump trials: no group differences were observed on PCT and TDEs. Taken together, there is evidence that children with DCD show some impaired control during the early stages of double-step reaching (when demands on ROC are greatest). Importantly, ROC is predicated on the ability to generate and monitor forward estimates of limb position in relation to changes in the relative position of the hand and target. A reduced ability to integrate
the resulting error signals with the ongoing motor command on jump trials may explain the longer TC in children with DCD, and hence their atypical pattern of performance. Intriguingly, the kinematics of double-jump reaching in DCD showed similarities to that seen in parietal patients; however, I urge caution when drawing strong inferences about their neuro-cognitive bases. In sum, the strong effect sizes observed in this and the previous study provide compelling evidence for impaired ROC in children with DCD. Whether this difficulty reflects a developmental immaturity or deviance from the typical developmental continuum is the subject of investigation in the third, and final, study presented in the next chapter.
CHAPTER 4

DISRUPTIONS TO ONLINE CONTROL IN CHILDREN WITH DCD:
DEVELOPMENTAL DELAY OR DEVIANCE?
Introduction and Overview

In Study 1 and 2, children with DCD demonstrated reduced competence correcting movement online during a double-step reaching task. I suggested that this deficit might reflect a decreased capacity for correcting movement in-flight using a predictive (forward) model of limb trajectory; specifically, in Chapter 3 I argued that children with DCD may experience difficulties using the error signal that arises when the predicted (according to the forward model) and actual sensory consequences differ to correct the on-going motor command.

Essential to understanding the role of predictive modeling in motor control and skill acquisition is establishing whether impaired double-step reaching (suggesting atypical predictive modeling) seen in children with DCD in Study 1 and 2 reflects immaturity of the motor system or a deviation from the normal developmental trajectory. This goal is equally important to the development of effective intervention since as discussed in chapter 3, a child’s prognosis is expected to differ depending on whether motor impairment results from immaturity or developmental deviance. In the case of immaturity, it is assumed that children have the potential to acquire age-appropriate motor skills, Conversely, should impairment reflect a developmental deviance children might not be expected to acquire age-appropriate levels of motor competence. As noted in the previous chapter, from an experimental perspective, the immaturity/deviance issue can be explored using carefully designed cross-sectional studies comparing the performance of children with DCD to younger (neurologically immature) control children. Immaturity is inferred when the profile of deficit displayed by children with DCD on a motor task mirrors that of younger control
children (Hill et al., 1998; Wann et al., 1998; Wilmut et al., 2007); if their profile does not fit on the normal developmental continuum, then deviance is inferred.

To date, evidence has been equivocal as to whether motor difficulties in DCD reflect developmental immaturity or deviance. While support for the developmental immaturity model exists in the growing number of studies that have shown similarities between the performance of children with DCD and younger typically developing children during movement [i.e. praxis skill (Hill et al., 1998), postural control (Wann et al., 1998) and COVAT (Wilmut et al., 2007)], there is also a body of evidence that has highlighted similarities between the abnormal motor performance of children with DCD and adult lesion patients supporting the developmental deviance model of DCD (Katschmarsky et al., 2001; Wilson et al., 2001; Wilson et al., 2004). For example, as discussed in Chapter 1, a number of studies investigating MI in children with DCD have observed the same atypical performance patterns during the mental rotation of limbs (compared to age-matched controls and normal adults) as is seen in PPC patients (Williams et al., 2006; Wilson et al., 2004).

With respect to ROC, in the preceding chapters I have highlighted parallels in the double-step reaching profile of children with DCD and PPC patients. In particular, I have shown that both groups experience difficulties correcting the trajectory of their reaching following unexpected target perturbation, possibly suggesting difficulties correcting movement in-flight using a predictive model of movement. From this, it might reasonably be inferred that impaired predictive modeling in DCD reflects developmental deviance since performance of PPC patients reflects output of a neurologically deviant motor system. However, as was the case for Wilson and colleagues work on MI in DCD, no group of younger controls was included for comparison in Study 1 or 2 of this thesis. Should the early profile of double-step
reaching (where, as I argued in the previous chapter, demands on predictive modeling are greatest) of children with DCD mirror that of younger control children, then the developmental immaturity model of impaired predictive control remains a plausible account of atypical ROC (and predictive modeling) in DCD. Next I will discuss evidence on how predictive modeling might develop in typically developing individuals.

**Predictive modeling and typical motor development.** As highlighted in Chapter 2 and 3, there is very little data on the development of ROC per se in typically developing children. Still, there is a large body of evidence demonstrating that goal-directed reaching undergoes considerable maturation between the ages of 5 and 12 years (Ferrel, Bard, & Fleury, 2001; Hay, 1978, 1979; Pellizer & Hauert, 1996; Smyth, Peacock, & Katamba, 2004). This period is characterised by a gradual (though non-monotonic) increase in reaching proficiency concomitant to reorganisation of the motor system at around 8 years of age. The more proficient reaching observed throughout this period (and into early adulthood) appears to reflect an increased capacity for integrating predictive (forward) and feedback-based control mechanisms. Evidence from open and closed-loop reaching consistently shows that children up to the age of 7 years tend to rely on predictive strategies to guide reaching. By the age of (≈) 8 years they have begun to develop the mechanisms necessary to integrate feedback-based information with predictive strategies more efficiently; this stage is characterised by a temporary decrease in reaching accuracy (Bard, Hay, & Fleury, 1990; Hay, 1979; Pellizer & Hauert, 1996). By the age of 9 to 12 years, children have begun to develop an integrated system similar to that seen in adults, though calibration of this system continues into adulthood (Ferrel et al. 2001).
This suggested developmental progression is, in part, supported by the only study to specifically investigate ROC in typically developing children (van Braeckel et al., 2007). Here, the authors compared the performance of children between the ages of 7 to 10 years on a modified double-step perturbation paradigm on jump trials, the initial target was extinguished 100ms after movement initiation and re-appeared in a peripheral location after a 120 ms delay. van Braeckel and colleagues showed that 63% of children met predetermined accuracy demands by completing at least two thirds of jump trials in a single movement without touching the initial target location. Based on the assumption that online corrections of this type require the successful integration of predictive (feedforward) and feedback mechanisms, these results suggest that by roughly 7 years of age children have developed (or at least begun to) the ability to correct the ongoing movement using a predictive estimate of the sensory consequences of movement with a degree of proficiency. However, as noted in chapter 3, task differences prevent reliable comparison of performance measures in van Braeckel and colleagues’ study with the majority of double-step reaching experiments (including the present). Most importantly, the delay in target perturbation (as well as re-appearance) adopted by van Braeckel et al. differs from traditional double-step designs where target perturbation coincides with moment onset; accordingly, it is not possible to reliably compare key performance variables such as tPA, tPV, TC and PCT.

Research on the development of predictive control using non-reaching paradigms. Evidence from performance of typically developing children during isometric force control (Smits-Engelsman et al., 2003), visuomotor adaptation (Ferrel-Chapus, Hay, Olivier, Bard, & Fleury, 2002), postural control (Hay & Redon, 1999) and force adaptation (Konczak, Jansen-Osmann, & Kalveram, 2003) also points
towards a shift in control strategies between the ages of 5 and 12 years characterised
by more efficient integration of predictive and feedback based mechanisms. For
example, Konczak and colleagues showed that 4 to 7 year-olds took longer to adapt
goal-directed forearm flexion movements following sudden externally driven force-
field changes (viz damping) than 8 to 11 year olds who, in turn, took longer to do so
than adults. Force-field changes of this kind result in discrepancy between the
expected limb trajectory (according to the forward model) and that indicated by
sensory in-flow, resulting in the generation of an error signal. Successful motor
correction is dependent on this signal being used to correct the ongoing motor
command. Faster movement correction in older children (8 to 11 year olds) suggests a
greater capacity for using error information to correct the motor command compared
to younger children (4 to 7 year olds). Improved efficiency in the adult group in turn
suggests that while children older than 8 years have developed a reasonable degree of
competence using this hybrid strategy, it would seem that continued refinement of
predictive modeling mechanisms occurs into adulthood (see also Ferrel et al., 2001).

Additionally, Hay and Redon (1999) showed that the amplitude of postural
disturbance resulting from self-induced perturbation was similar in typically
developing children between 9 and 10 years of age and adults yet decreased in 6 to 8
year olds. Based on the assumption that lower postural disturbance following self-
induced perturbation reflects greater reliance on anticipatory control strategies to re-
establish postural stability, these results suggest a greater reliance on predictive
strategies to re-gain stability in children aged 6 to 8 years compared to older children
and adults who used a more integrated approach.

Taken together, there is strong evidence that the ability to integrate feedback
mechanism efficiently with predictive modes of control begins to develop at, or
around, 8 years of age and becomes adult-like between the ages of 9 and 12 years (Hay & Redon, 1999)- though fine-tuning continues into early adulthood (Ferrel et al., 2001; Konczak et al., 2003).

**Summary.** The aim of Study 3 was to investigate whether a decreased capacity for correcting movement online (viz. predictive modeling) in children with DCD reflects a developmental immaturity or a deviance. This was achieved by comparing the kinematic and chronometric performance of children with DCD (8-12 years of age) with a younger group of control children (5 to 7 years of age) as well as age-matched controls on the double-step reaching task. A group of young adults was included (20 to 28 years) to provide a reference point for mature movement and hence better elucidate the nature of predictive control across typical development. In doing so, I hoped to gain a clearer understanding of where, if at all, predictive modelling in children with DCD fit along the normal developmental continuum.

Based on earlier work that suggests a transition in motor control strategies at around 8 years of age that sees an increased to capacity for integrating predictive and feedback-based strategies (which I have argued is crucial to efficient online control), I expected typically developing children younger than 8 years to demonstrate a decreased capacity for making rapid online corrections during double-step reaching than children aged 8 to 12 years; manifest as slower, less accurate double-step reaching. Further, since the mechanisms supporting predictive modeling are expected to continue to refine into adulthood (i.e. Ferrel et al., 2001), I predicted improved double-step reaching in young adults compared to older children. With respect to children with DCD, again I predicted a decreased capacity for making online corrections to reaching. Since I suggested that difficulties using predictive modeling strategies might explain poor ROC in children with DCD in Study 1 and 2 and that
this system might also be immature in younger control children, I expected the performance of children with DCD to parallel that of younger controls during double-step reaching thus supporting the developmental immaturity model of impaired predictive modeling in DCD.

Method

Participants

The sample consisted of 5 children between the ages of 8 and 12 years who met research criteria for DCD (a per study 1 and 2); preliminary inspection showed that this additional sample did not differ substantially from the DCD sample of Study 2 on key measures of ROC (i.e. MT\textsubscript{Diff}, interaction on MT, TC). Consequently, data from the two groups was collapsed for analysis in the present study. Accordingly, an additional 5 age-matched control children were included with the control sample collected in Study 2. Again, preliminary analysis did not show significant differences on key measures of ROC between the two samples. In total, the DCD (11 girls and 7 boys) and age-matched control groups (10 girls and 8 boys) for Study 3 consisted of 18 participants respectively. The mean ages for the DCD group and age-matched controls were 10.47 (SD= 1.60) years and 10.42 (SD= 1.22) years respectively.

The sample also included a group of 12 ‘younger’ control children aged between 5 and 7 years (7 girls and 5 boys) and 14 young adults aged between 20 and 28 years (5 females, 9 males). The mean ages for the younger control and young adult groups were 6.60 years and 24.70 years respectively. All participants were right-hand dominant with the exception of 4 children in the DCD group and 1 in the age-matched control group. All children were screened using the same procedure adopted in Study 1 and Study 2 that was approved by the RMIT HREC and the Victorian Government
Department of Education and Early Childhood Development (See Chapter 2). Children younger than 7 years of age completed a modified consent form (See Appendix D). Young adults were undergraduate psychology students at RMIT University; all gave informed consent (for a copy of the young adult participant plain language statement and informed consent form see Appendix E). Young adults were considered to have age-appropriate levels of motor skill and a MAND total score above the 20th percentile.

**Apparatus**

Apparatus were identical to that employed in Study 2.

**Procedure**

The procedure was identical to that employed in Study 2.

**Design and Analysis**

As for Study 2, kinematic data were filtered off-line using a dual pass Butterworth second order filter with a cut-off of 10 Hz. Dependent variables were the also identical to Study 2. Preliminary analysis revealed that condition had no effect on total number of AEs or TDEs committed so trials were collapsed over ‘jump’ and ‘non-jump’ trials for these indices. As for the previous two studies, disregarding all types of error, a criterion of 50% correct responses on jump trials (or 8 out of 16) was deemed a minimum sampling requirement; all participants tested met this criterion. Of all correct responses, outliers were defined as responses where RT, MT, MT\textsubscript{diff}, tPA, tPV, TC or PCT was > +3 or < -3 SD from the participants mean for that condition. Trials on which an error occurred or trial performance was deemed an outlier were excluded from the analysis; an average of 24 (30%), 18 (23%), 23 (29%)
and 10 (12%) trials were removed from the DCD, age-matched control, younger control and young adult groups respectively.

Separate 2-way ANOVA (4 [Group] x 2 [Condition]) were conducted on mean RT, MT, tPA and tPV data; appropriate post-hoc analysis were conducted to investigate significant findings. Since investigation of interaction effects for MT between separate pairs of participant groups were of particular importance to my analysis, following appropriate post-hoc analysis of the omnibus 2-way ANOVA, separate 2-way ANOVA were then conducted to compare MT of paired groups of interest separately. Accordingly, (typical) developmental trends were first investigated by conducting separate 2-way ANOVA comparing the MT of younger and age-matched controls followed by comparison of age-matched controls and young adults. The performance of children with and without DCD were then analysed; separate 2-way ANOVAs comparing firstly, the performance of children with DCD and age-matched controls was undertaken followed by children with DCD compared to younger control children. 1-way ANOVAs were conducted on MT$_{diff}$, TC, PCT, AE, CTE and TDE. Appropriate post-hoc tests were conducted to investigate significant findings.

Since correcting the family-wise error rate unduly increases the likelihood of committing a Type II error (see O’Keefe, 2003; Saville, 1990), no adjustments were made to the standard alpha level of $p < .05$. Measures of effect size were used to temper the results of significance tests; Partial-eta squared (partial $\eta^2$) was used for this purpose.

In terms of the presentation of the analysis which is presented next, for all dependent variables where post-hoc analysis took place, they were presented in the following order. First, developmental trends are presented. These were investigated
by comparing the performance of younger and older control children, then older children and adults. Next, data on children with and without DCD is presented. Here I compared the performance of children with DCD with age-matched controls and then the DCD group with younger controls.

Results

Chronometric Analysis

Reaction Time. Mean RTs (+/- SE) for each group are displayed in Figure 4.1. The 2-way ANOVA comparing all groups on mean RT revealed no significant interaction effect between group and condition $F= 0.35$, $p = .79$, partial $\eta^2 = 0.02$. The main effect for condition was not significant, $F = 1.02$, $p = .32$, partial $\eta^2 = 0.02$. A significant main effect was found for group on RT, $F (3, 58)= 13.08$, $p= .001$, partial $\eta^2 = 0.40$.

![Figure 4.1. Mean group reaction times (RT, +/- SE). NOTE: younger control, YC; age-matched control, AMC; young adult, A; developmental coordination disorder, DCD.](image)
Averaged over condition the RT of younger control children (group mean = 679 ms) was significantly longer than age-matched controls (522 ms) ($t(28) = 4.50$, $p = .001$, partial $\eta^2 = 0.42$); no significant difference was observed between age-matched controls and young adults (481 ms) ($t(30) = 1.49$, $p = .15$, partial $\eta^2 = 0.07$).

Averaged across conditions, no significant difference was observed on RT between children with DCD and age-matched controls ($t(28) = 1.46$, $p = .15$, partial $\eta^2 = 0.06$); children with DCD (564 ms) were significantly faster to initiate reaching compared to younger controls ($t(28) = 3.19$, $p = .01$, partial $\eta^2 = 0.28$).

**Movement Time.**  Mean MT’s (+/- SE) for each group are shown in Figure 4.2. The 2-way ANOVA comparing all groups on mean MT revealed a significant interaction between group and condition, Wilk’s $\Lambda = 0.68$, $F(3,58) = 8.15$, $p = .001$; and large effect size, partial $\eta^2 = 0.32$. Also significant were the main effects for condition, Wilk’s $\Lambda = 0.06$, $F(1, 58) = 977.26$, $p = .001$, partial $\eta^2 = 0.94$; and group, $F(3,58) = 23.01$, $p = .001$, partial $\eta^2 = 0.54$.

![Figure 4.2. Mean group movement time (MT, +/- SE).](image-url)
Analysis of simple main effects showed that the reaching of age-matched control children (557 and 833 ms; non-jump and jump) was significantly faster than younger control children (651 and 1039 ms) during both non-jump \( (t (28)= 3.60, p= .001, \text{partial } \eta^2 = 0.31) \) and jump trials \( (t (28)= 4.29, p= .001, \text{partial } \eta^2 = 0.40) \). In turn, age-matched controls were slower to complete non-jump (young adult= 442 ms) \( (t (30)= 4.03, p= .001, \text{partial } \eta^2 = 0.35) \) and jump trials (young adults= 691 ms) \( (t (30)= 4.54, p= .001, \text{partial } \eta^2 = 0.41) \) compared to young adults.

No difference for MT was found between DCD and age-matched controls during non-jump reaching (DCD= 557 ms) \( (t (34)= 0.03, p= .97, \text{partial } \eta^2 = 0.000) \), however, children with DCD (901 ms) were significantly slower than age-matched control children during jump trials \( t (34)= 2.20, p= .03, \text{partial } \eta^2 = 0.13 \). Children with DCD were significantly faster to complete non-jump \( (t (28)= 3.65, p= .001, \text{partial } \eta^2 = 0.32) \) and jump trial reaching \( (t (28)= 2.83, p= .01, \text{partial } \eta^2 = 0.22) \) than younger controls.

**Developmental trends.** A 2-Way ANOVA showed a significant interaction for group and condition on MT comparing younger control and age-matched controls, Wilk's \( \Lambda = 0.75, F (1, 28) = 9.53, p= .005, \text{partial } \eta^2 = 0.25 \). The main effects for condition (Wilk's \( \Lambda = 0.79, F (1, 28) = 328.18, p= .001, \text{partial } \eta^2 = 0.92 \)) and group \( (F (1, 28) = 19.40, p= .001, \text{partial } \eta^2 = 0.40) \) were also significant.

When comparing age-matched control children with young adults on MT, the 2-Way ANOVA failed to reveal a significant interaction effect for group by condition, \( F= 1.96, p= .17, \text{partial } \eta^2 = 0.06 \). The main effects for condition (Wilk's \( \Lambda = 0.36, F (1, 30) = 805.28, p= .001, \text{partial } \eta^2 = 0.96 \)) and group \( (F (1, 30) = 20.21, p= .001, \text{partial } \eta^2 = 0.40) \) were both significant.
MT\textsubscript{Diff} scores across typical development. Individual mean MT\textsubscript{Diff} scores for all groups are presented in Figure 4.3. MT\textsubscript{Diff} scores were significantly lower for age-matched (276 ms) compared to younger controls (388 ms), \( t(28)=3.09, p=.005, \) partial \( \eta^2 = 0.25. \) No significant difference was observed for young adults (250 ms) and age-matched control children (276 ms), \( t(30)=1.40, p=.17, \) partial \( \eta^2 = 0.06. \) The CI\textsubscript{95\%} for the age-matched control group was between 242 and 309 ms compared with 329 and 412 ms for younger controls and 212 and 288 ms for young adults. Eight of the 12 younger control children (or 67\%) exceeded the upper CI\textsubscript{95\%} of the age-matched control group (i.e. 309 ms), while 3 of the 18 age-matched controls (or 17\%) exceeded the lower CI\textsubscript{95\%} of the younger control group (i.e. 329 ms). Eight of the 18 age-matched control children (or 44\%) exceeded the upper CI\textsubscript{95\%} of the young adult group (i.e. 288 ms), while 9 of the 14 young adults (or 64\%) exceeded the lower CI\textsubscript{95\%} of the age-matched control group (i.e. 242 ms).

![Figure 4.3](image-url)

\textit{Figure 4.3.} Individual mean MT\textsubscript{Diff} scores for each group.
**Children with and without DCD.** A 2-way ANOVA on MT revealed a significant interaction effect for condition by group when comparing children with DCD and age-matched controls, Wilk's $\Lambda = 0.76$, $F (1, 34) = 10.66$, $p = .003$, partial $\eta^2 = 0.24$; while the condition effect was also found to be significant, Wilk's $\Lambda = 0.04$, $F (1, 34) = 881.10$, $p = .001$, partial $\eta^2 = 0.96$. No effect for group was observed, $F = 1.85$, $p = .18$, partial $\eta^2 = 0.05$.

A 2-way ANOVA failed to show a significant interaction effect for group and condition on MT when comparing children with DCD and younger controls, $F = 1.51$, $p = .23$, partial $\eta^2 = 0.05$. The main effects for condition (Wilk's $\Lambda = 0.07$, $F (1, 28) = 397.40$, $p = .001$, partial $\eta^2 = 0.93$) and group ($F (1, 28) = 11.36$, $p = .002$, partial $\eta^2 = 0.29$) were both significant.

**Comparison of MT_{Diff} scores for children with and without DCD.** Analysis of MT_{Diff} scores revealed that children with DCD (group mean= 344 ms) scored significantly higher than age-matched control children (275 ms), $t (34) = 3.26$, $p = .003$, partial $\eta^2 = 0.24$; no difference was observed between children with DCD and younger controls (388 ms), $t (28) = 1.23$, $p = .23$, partial $\eta^2 = 0.05$. Analysis of individual differences revealed that the CI_{95%} for the DCD group was between 310 and 378 ms. Thirteen of the 18 children with DCD (or 72%) exceeded the upper CI_{95%} of the age-matched control group (i.e. 309 ms) while 5 of the 18 age-matched controls (or 28%) exceeded the lower CI_{95%} of the DCD group (i.e. 310 ms).

**Kinematic Analysis**

**Early control parameters.**

**tPA and tPV.** The 2-way ANOVA on mean tPA comparing all groups showed the main effect for group to be significant, $F (3,58) = 24.48$, $p = .001$, partial
\( \eta^2 = 0.56 \). The analysis failed to reveal a significant interaction effect for group and condition \( (F= 0.38, p= .77, \text{ partial } \eta^2 = 0.02) \); nor was the main effect for condition significant \( (F= 0.84, p= .36, \text{ partial } \eta^2 = 0.01) \).

The 2-way ANOVA on mean tPV for all groups showed the main effect for group not to be significant, \( F= 2.67, p= .06, \text{ partial } \eta^2 = 0.12 \). The analysis also failed to reveal a significant interaction effect for group and condition \( (F= 0.40, p= .75, \text{ partial } \eta^2 = 0.02) \). The main effect for condition was also not found to be significant \( (F= 1.67, p= .21, \text{ partial } \eta^2 = 0.03) \).

**Developmental trends.** Averaged across condition, analysis failed to reveal a significant difference in tPA \( (t (28)= .97, p= .34, \text{ partial } \eta^2 = 0.03) \) or tPV \( (t (28)= .88, p= .39, \text{ partial } \eta^2 = 0.03) \) between control children and younger control children. Adults reached tPA \( (t (30)= 8.17, p= .001, \text{ partial } \eta^2 = 0.69) \) significantly earlier than age-matched controls; no significant difference was observed on tPV \( (t (30)= 1.60, p= .12, \text{ partial } \eta^2 = 0.08) \).

For younger controls, neither tPA \( (t (11)= .46, p= .70, \text{ partial } \eta^2 = 0.001) \) nor tPV \( (t (11)= .06, p= .95, \text{ partial } \eta^2 = 0.001) \) differed significantly across conditions; this was also the case of age-matches controls \( [tPA, \ t (17)= .48, p= .63, \text{ partial } \eta^2 = 0.003, \text{ and } tPV, \ t (17)= .48, p= .64, \text{ partial } \eta^2 = 0.001] \). For young adults, there was no significant difference for tPA across conditions \( (t (13)= 1.16, p= .27, \text{ partial } \eta^2 = 0.001) \), while tPV \( (t (13)= 3.85, p= .01, \text{ partial } \eta^2 = 0.01) \) was significantly faster for jump trials (group mean= 144 ms) compared to non-jump trials (152 ms).

**Children with and without DCD.** For children with DCD, neither tPA \( (t (17)= 1.55, p= .14, \text{ partial } \eta^2 = 0.01) \) nor tPV \( (t (17)= .44, p= .66, \text{ partial } \eta^2 = 0.001) \) were significantly affected by condition.
Averaged across condition, analysis failed to show a significant difference between either tPA ($t\ (34)= 1.98, \ p = .06, \ \text{partial } \eta^2 = 0.10$) or tPV ($t\ (34)= 1.47, \ p = .15, \ \text{partial } \eta^2 = 0.10$) for children with DCD and age-matched control children; this was also the case when comparing children with DCD and younger controls [tPA, ($t\ (28)= .28, \ p = .78, \ \text{partial } \eta^2 = 0.003$); tPV, ($t\ (28)= .11, \ p = .91, \ \text{partial } \eta^2 = 0.001$)].

**Time to Correction (TC).** Mean TC’s are presented in Figure 4.4. A 1-Way ANOVA showed a significant group effect for TC, $F\ (3,58) = 22.35, \ p = .001$, partial $\eta^2 = 0.54$. Independent $t$-tests showed that younger control children were slower than age-matched controls to correct the trajectory of their reaching on jump trials, ($t\ (28) = 4.45, \ p = .001, \ \text{partial } \eta^2 = 0.51$); while age-matched controls were significantly slower than young adults ($t\ (30) = 3.85, \ p = .001, \ \text{partial } \eta^2 = 0.33$). Analysis of individual differences revealed that the CI$_{95\%}$ for the age-matched control group was between 314 and 352 ms compared to 368 and 412 ms for younger control children and 268 and 309 ms for young adults. Ten of the 12 younger control children (or 83%) exceeded the upper CI$_{95\%}$ of the age-matched control group (i.e. 352 ms), while 1 of the 18 age-matched controls (or 6%) exceeded the lower CI$_{95\%}$ of the younger control group (i.e. 368 ms). Sixteen of the 18 age-matched control children (or 89%) exceeded the upper CI$_{95\%}$ of the young adult group (i.e. 309 ms), while 4 of the 14 young adults (or 29%) exceeded the lower CI$_{95\%}$ of the age-matched control group (i.e. 314 ms).
Independent \( t \)-tests also showed that children with DCD were slower to correct the trajectory of their reaching compared to age-matched controls (\( t (34) = 4.42, p = .001 \), partial \( \eta^2 = 0.37 \)); no difference was observed when comparing DCD children to younger controls (\( t (28) = .62, p = .54 \), partial \( \eta^2 = 0.01 \)). Analysis of individual differences showed that the CI\(_{95\%}\) for the DCD group was between 365 and 400ms. Sixteen of the 18 children with DCD (or 89\%) exceeded the upper CI\(_{95\%}\) of the age-matched control group (i.e. 352ms), while 2 of the 18 age-matched controls (or 11\%) exceeded the lower CI\(_{95\%}\) of the DCD group (i.e. 365 ms).

**Late control parameters (Post-Correction Time- PCT).**

A 1-Way ANOVA showed a significant group effect for PCT across groups, \( F (3,58) = 16.12, p = .001 \), partial \( \eta^2 = 0.45 \). Independent \( t \)-tests showed that younger control children spent significantly longer reaching post trajectory correction on jump trials compared to age-matched controls (\( t (28) = 3.83, p = .001 \), partial \( \eta^2 = 0.34 \)). PCT values were significantly longer for age-matched controls compared to young adults (\( t (30) = 3.48, p = .002 \), partial \( \eta^2 = 0.29 \)).
No group differences on PCT were shown between children with DCD and age-matched controls \((t (34) = .91, p=.37,\) partial \(\eta^2 = 0.02)\), while children with DCD spent significantly less time reaching post-correction of movement trajectory compared to younger controls \((t (28) = 3.31, p=.003,\) partial \(\eta^2 = 0.28)\).

**Error**

**Anticipatory errors.** Mean group AEs are presented in Figure 4.5. The 1-Way ANOVA did not show a significant group effect for AE, \(F (3, 58) = 1.57, p=.21,\) partial \(\eta^2= 0.08\). Independent \(t\)-tests failed to reveal a difference on AE when comparing younger and age-matched controls \((t (28) = .61, p=.55,\) partial \(\eta^2 = 0.01)\); no significant difference was observed when comparing age-matched controls and young adults \((t (30) = 1.83, p=.08,\) partial \(\eta^2 = 0.10)\).

No difference on AEs was shown between children with DCD and age-matched controls \((t (34) = .13, p=.90,\) partial \(\eta^2 = 0.000)\) or younger controls \((t (28) = .71, p=.49,\) partial \(\eta^2 = 0.17)\).

![Figure 4.5. Mean group AE and CTE (+/- SE).](image)
**Centre touch errors.** Mean group CTEs are presented in Figure 4.4. The 1-way ANOVA on mean CTEs revealed significant group differences, $F(3, 58) = 3.01, p = .04$, partial $\eta^2 = 0.14$. Independent $t$-tests failed to reveal a difference on CTE between younger and age-matched controls ($t(28) = 1.93, p = .06$, partial $\eta^2 = 0.12$); no difference was observed when comparing age-matched control and young adults ($t(30) = .56, p = .58$, partial $\eta^2 = 0.01$).

Children with DCD committed significantly more CTEs compared to age-matched controls ($t(34) = 1.96, p = .05$, partial $\eta^2 = 0.10$) while no difference was shown between children with DCD and younger controls ($t(28) = .28, p = .78$, partial $\eta^2 = 0.003$).

**Touch down errors.** The 1-way ANOVA on mean TDEs showed a significant group effect, $F(3, 58) = 8.60, p = .001$, partial $\eta^2 = 0.31$. Analysis revealed that younger controls committed significantly more TDEs than age-matched controls ($t(28) = 2.41, p = .02$, partial $\eta^2 = 0.17$), while no significant difference was observed between age-matched controls and young adults ($t(30) = 1.88, p = .07$, partial $\eta^2 = 0.11$).

Children with DCD committed significantly more TDEs than age-matched controls ($t(34) = 2.72, p = .01$, partial $\eta^2 = 0.18$); no difference was observed between children with DCD and younger controls ($t(28) = .16, p = .88$, partial $\eta^2 = 0.001$).

**Summary of results**

In summary, there were a number of significant findings with respect to group comparison of typically developing participants. Analysis showed that RT decreased significantly as a function of age, shown by a significant group effect on the 2-way ANOVA and post-hoc analysis: this was also the case for MT. Comparing younger
and age-matched controls, 2-way ANOVA on MT revealed a significant interaction effect. The same comparison of age-matched controls and young adults failed to reveal an interaction effect. MT\textsubscript{Diff} was significantly lower in age-matched compared to younger controls; there was a non-significant trend for lower MT\textsubscript{Diff} in adults compared to age-matched controls.

2-way ANOVA failed to reveal a condition effect for either tPA or tPV. Averaged over condition, no differences were observed between younger and age-matched controls for tPA or tPV. Young adults reached both parameters faster than age-matched controls- the difference for tPV indicated a non-significant trend. Also, young adults were the only group to show a condition dependent decrease in tPV, though no such effect was observed on tPA. 1-way ANOVA revealed a significant group effect for both TC and PCT, with post-hoc analysis showing significant decline with age on these indices.

In terms of errors, no difference was observed between younger and age-matched control children for AE, while young adults showed a non-significant trend for decreased AEs compared to age-matched controls. Analysis revealed a non-significant trend for decreased CTEs by age-matched controls compared to younger controls; no difference was observed between young adults and age-matched controls. Finally, age-matched controls committed significantly less TDEs compared to younger controls and showed a non-significant trend for increased TDEs compared to young adults.

A number of significant findings were also detected when comparing children with and without DCD. Averaged across condition, children with DCD showed a non-significant trend for slower RT’s than age-matched controls though they were significantly faster on this metric than younger controls. The 2-way ANOVA showed
a significant interaction effect on MT when comparing the DCD and age-matched control groups, though no overall group effect was detected; children with DCD also showed significantly higher $MT_{\text{Diff}}$ scores. The 2-way ANOVA comparing the MT of children with DCD and younger controls showed a significant group main effect though no interaction for group by condition was observed; also, $MT_{\text{Diff}}$ scores did not differ.

No differences for tPA or tPV were observed between children with DCD or either control group. Independent $t$-tests showed that TC was significantly slower in the DCD group compared to the age-matched controls while no difference was shown compared to younger controls. No difference between DCD and age-matched control children was shown for PCT while younger control children were shown to be significantly slower than the DCD children on this metric.

Mean AEs did not differ between the DCD or controls groups. Children with DCD committed significantly more CTEs than age-matched controls; no difference was shown in comparison to younger control children. Averaged over condition, children with DCD committed significantly more TDEs than age-matched controls; no group differences were observed in comparison to younger controls.

**Discussion**

The aim of Study 3 was to explore whether the abnormal pattern of online control in children with DCD (Study 1 and 2) reflects immaturity of the motor system or deviance from a normal developmental trajectory. The performance of children with DCD (8 to 12 years) was compared to younger control children (5 to 7 years) and age-matched controls on the double-step reaching task, using both kinematic and chronometric variables. The performance of a group of young adults (20 to 28 years)
provided a benchmark for mature control. I predicted incremental improvements in performance between younger controls, older (i.e. age-matched) controls, and young adults, manifest as faster and more accurate double-step reaching with age. As was the case for Study 1 and 2, I predicted that children with DCD would demonstrate a reduced capacity for ROC, similar in nature to the pattern seen in younger controls.

As predicted, younger control children were slower to adjust for target perturbation at movement onset than age-matched controls. In turn, more efficient performance was seen in young adults when compared with age-matched controls. Kinematic analysis showed that improved efficiency of movement throughout development was reflected both in the early stages of reaching (i.e. decreasing TC with age) where demands on predictive modeling are thought to be high, as well as later in the movement cycle (i.e. faster PCT with age) where rapid corrections are no longer required. Furthermore, adults demonstrated very early adjustments to reaching on jump trials not demonstrated by any child group (shown by a significant decrease in tPV on jump trials). I argue that this pattern of results indicates that improved online control throughout childhood and into early adulthood is, at least in part, supported by an increased capacity to integrate feedback-based mechanisms with forward estimates of future limb position (i.e. predictive models). Data suggests that while this system is quite functional between the ages of 8 and 12 years, continued fine-tuning occurs through adolescence and into adulthood.

Finally, the double-step reaching of children with DCD and younger controls was equally disadvantaged by target jumps, as shown by similar MT Diff and TC scores. Based on the assumption that TC reflects the stage in reaching that error information has been successfully integrated with the unfolding motor command, these results suggest that both children with DCD and younger controls share a
reduced capacity for correcting movements in-flight, based on forward estimates of limb position. I argue that this finding supports the view that atypical predictive modeling may reflect a developmental immaturity. These suggestions are discussed in detail below.

**Chronometric Analysis – Reaction Time**

*Condition effect.* As expected, no effect for condition was found, like Study 1 and 2. This result was discussed in Chapter 2.

*Developmental trends.* As expected, younger control children were significantly slower to initiate movement compared to age-matched controls who, in turn, showed a non-significant trend for slower movement initiation compared to young adults ($p=.15$). This overall age-dependent decrease in RT may reflect increased efficiency in information processing and/or motor planning (see Desmurget et al., 2004) and supports a compelling body of evidence demonstrating a dramatic increase in the speed of information processing from childhood and then into adolescence where performance tends to plateau (see Luna, Garver, Urban, Lazar, & Sweeney, 2004). From a neuromaturational perspective, this progression is thought to be subserved by a shift from diffuse to focal neural processing throughout childhood and into adolescence (i.e. increased activation of task related neural regions and deactivation of unrelated areas) (Casey, 2006; Durston et al., 2006)- this process is the emergent property of a combination of synaptic pruning, increased neuronal myelination and stronger connections within and between task relevant brain networks (Bunge & Wright, 2007).

*Children with and without DCD.* Children with DCD demonstrated a non-significant ($p=.15$) trend for slower movement initiation compared to age-matched
controls suggesting slower processing speed and/or organisation of movement in DCD. This result accords with Study 1 and 2 and is consistent with the bulk of available evidence demonstrating slower movement initiation towards external stimuli in DCD (Wilson & McKenzie, 1998).

Children with DCD were also significantly faster to initiate reaching compared to younger controls. Hence, it would appear that the increase in information processing and/or motor planning efficiency shown here and elsewhere throughout typical development (see Luna et al., 2004) also occurs in children with DCD though perhaps to a lesser degree (since their RT was still slower than age-matched controls). This finding is important as it demonstrates that the similar profiles of impaired ROC observed between children with DCD and younger controls (discussed below) cannot be explained in terms of shared inefficiencies in neural processing.

**Chronometric Analysis – Movement Time**

*Condition effect.* In accordance with Study 1 and 2, movement time increased significantly from non-jump to jump trials. As discussed in chapters 2 and 3, this is largely attributed to the increase in movement amplitude and task complexity associated with jump compared to non-jump trials (See Chapter 2).

*Developmental trends.* As expected, overall reaching speed increased with age (shown by a significant group main effect on MT and respective post-hoc analysis). This supports the body of evidence showing generally faster movement times over childhood (particularly throughout the primary school years) and into adolescence (Shumway-Cook & Woollacott, 2007). In comparison to younger typically developing children, age-matched controls were significantly faster to adjust for target perturbation shown by lower MT\textsubscript{Diff} scores. This finding was further highlighted by, analysis of individual differences that showed that 67% of younger
controls were unduly constrained by target displacement with respect to older controls- that is, their mean MT\textsubscript{Diff} score fell above the 95\% CI for age-matched control children. This type of online correction is thought to be dependent on the capacity of the nervous system to use error information which arises following discrepancy between the expected and actual person-target relationship with the unfolding motor command. Clearly, older children showed a greater capacity for implementing this control system compared with younger children aged 5 to 7 years, a finding further supported by . This finding also accords with evidence from the developmental literature suggesting that the motor system undergoes re-organization at around 8 years of age concomitant to an increased capacity for predictive modeling (Hay, 1979; Konczak et al., 2003; Pellizer & Hauert, 1996; van Braeckel et al., 2007).

Adults were somewhat less disadvantaged by jump trials than older children, shown by a moderate effect size on MT\textsubscript{Diff} scores. Indeed, only roughly half (56\%) of the older control children had mean MT\textsubscript{Diff} scores that fell within the CI 95\% of the young adult group. Thus, it seems that while the hybrid control system which supports ROC undergoes large scale maturation between the ages of 8 and 12 years, fine-tuning continues into adulthood, consistent with earlier work (Ferrel et al. 2001; Konczak et al., 2003). This is discussed in detail below in the context of kinematic data.

**Children with and without DCD.** In accordance with Study 1 and 2 of this thesis, children with DCD were able to complete reaching as efficiently as their same-age peers when the target remained stationary throughout movement (non-jump) (see also Wlimut et al., 2006). However, the reaching of children with DCD was impacted by target perturbation to a far greater extent than age-matched controls demonstrated by increased MT\textsubscript{Diff} scores and a significant interaction effect on MT. As was found
to be case in Study 1 and 2, analysis of individual difference showed that some 72% of children with DCD were unduly constrained by target perturbation- i.e. their mean MT\text{Diff} score fell above the 95% CI for age-matched controls. These results are consistent with the hypothesis that ROC is compromised in DCD. Interestingly, children with DCD and younger controls were equally affected by unexpected target perturbation, reflected in equivalent MT\text{Diff} scores and a lack of interaction effect on MT providing preliminary evidence that impaired predictive modeling may reflect a developmental immaturity.

Since simple (non-jump) reaching was preserved in children with DCD compared to age-matched controls, these results re-affirm the notion that the aspects of the motor system responsible for simple reaching may be preserved in children with DCD (see also Wilmut et al., 2006).

**Summary.** Younger control children were slower to adjust to target perturbation than age-matched controls as shown by higher MT\text{Diff} scores. These results suggest an increased capacity for integrating predictive and feedback-based mechanisms in 8 to 12 year olds compared to 5 to 7 year olds. Similarly, young adults were faster to correct their movement in response to target perturbation compared to age-matched controls indicating that maturation of this system continues into adolescence and early adulthood.

Again, children with DCD were slower to correct their reaching following unexpected target perturbation compared to age-matched controls. Similarities in performance to younger controls supports the notion that impaired predictive modeling in DCD may reflect a developmental immaturity. A discussion of the kinematic data to follow will clarify this hypothesis.
Kinematic Analysis

Early control parameters

$tPA$ and $tPV$. With respect to age effects, no differences were observed for either $tPA$ or $tPV$ between any of the child groups. Temporal constraints prevent both non-visual and visual-feedback systems from affecting movement to these early markers and hence reaching is thought to be chiefly under the control of the initial motor command. These results therefore suggest an equal reliance on the initial motor command to guide the preliminary stages of reaching in typically and atypically developing children aged 5 to 12 years. As expected, adults tended to be were faster to reach both $tPA$ and $tPV$ compared to age-matched control children, providing a greater window for exerting online adjustments before the target is captured by the hand.

Unlike healthy adults, (Castiello et al., 1991; Farné et al., 2003; Paulignan et al., 1991), no group of children (including DCD) showed earlier $tPA$ or $tPV$ on jump trials relative to non-jump. Again, these early adjustments are a consequence of target displacement, specifically the stage in reaching when error between the expected sensory consequences of movement (according to the predictive model) and those indicated by actual sensory inflow has been detected (Farnè et al., 2003; Paulignan et al., 1991). The point in movement when reaching trajectory deviates towards the new cued target is thought to indicate the successful integration of the resultant error signal with the on-going motor command (Dubrowski et al., 2002; van Braeckel et al., 2007). As mentioned in Chapter 3, the lack of effect for task condition on $tPA$ or $tPV$ (see also van Braeckel et al., 2007) may reflect a developmental immaturity; that is, the fast internal feedback loops that support the early detection of error in adults are
not fully developed in children aged 5 to 12 years (see Chapter 3 for a more detailed discussion).

Consistent with earlier work (i.e. Castiello et al., 1991; Farnè et al., 2003; Paulignan et al., 1991), young adults showed a shorter tPV on jump compared to non-jump trials. However, this effect was not shown for tPA. Interestingly, tPA occurred relatively quickly for adults in the present study compared to previous research when target perturbation (more or less) coincided with movement initiation [mean= 73ms here Vs. 99-110 (Castiello et al., 1991), 97-108ms (Farnè et al., 2003), 98-104ms (Paulignan et al., 1991)]. Still, the value for tPA presented here is not without precedence- i.e. 63ms (Prablanc & Martin, 1992). In fact, while tPA values below 100ms are quite common on jump trials, to my knowledge condition dependent decreases have not been recorded earlier than 97 ms post target perturbation. Thus, it may be that the fast internal feedback loops that support this very early braking in the reaching cycle require at least (≈) 100ms to implement. Still, given that on average adults reached tPV within 150 ms, the decrease in time to this marker from non-jump to jump trials still likely reflects interruption of the reaching trajectory using powerful internal feedback loops. These circumvent delays associated with processing sensory feedback by comparing a forward estimate of the unfolding limb trajectory to actual sensory feedback. Any discrepancy results in the generation of an error signal which is used to correct the unfolding motor command with minimal time-lag. By comparison, sensory feedback loops alone can take upwards of 250 ms to process incoming visual information (see Frith et al., 2000).

**Time to correction of movement trajectory**

**Developmental trends.** Younger control children were significantly slower to correct the trajectory of their reaching away from the initial target than age-
matched controls. Indeed, analysis of individual differences showed that the TC of 83% of younger controls exceeded the upper 95% CI of age-matched controls. These findings suggest more efficient integration of error information with the on-going motor command in children aged 8 to 12 years compared to 5 to 7 years-olds. Again, this finding is consistent with earlier research on the typical development of predictive modeling suggesting a transition in the nature of online control at around 8 years of age; at this point, children develop an increased capacity to use predictive models as a basis for making rapid online corrections (Hay, 1979; Pellizer & Hauert, 1996). This is discussed in detail in the next and final chapter.

As expected, young adults were significantly faster to correct the trajectory of their reaching compared to age-matched controls. In fact, only 11% of the mean TC values for older children fell within the 95% CI of young adults. Mean TC values presented here for adults (288ms) are consistent with earlier double-step studies where target perturbation occurs at movement onset [i.e. 280-330ms (Dubrowski et al., 2001), 255-295 ms (Paulignan et al., 1991) 238- 264ms, (Rossit & Harvey, 2008)]. Also, the TC values (mean= 333ms) for age-matched controls were largely replicated in Study 2 (328ms). This evidence is consistent with the view that young adults are able to update the ongoing motor command by way of sensory feedback faster than children aged 8 to 12 years. Based on the finding of a condition dependent decrease in tPV, it may be that adults are able to use error information to re-calibrate the un-folding motor command earlier in the reaching cycle. Again, this finding supports earlier work indicating continued development of predictive modeling systems into adolescence and early adulthood (see Ferrel et al., 2001; Konczak et al., 2003).
Children with and without DCD. Similarly to Study 2, children with DCD were significantly slower to correct the trajectory of their reaching in response to target perturbation compared to age-matched controls, with the mean TC scores of 83% of children with DCD exceeding the upper 95% CI of age-matched controls. Interestingly, the magnitude of slower TC shown by the DCD group was similar to that seen in younger controls (manifest as equivalent TC scores). Hence, it appears that children with DCD and younger controls may share a similar degree of difficulty integrating information about target perturbation (i.e. the error signal) with the ongoing motor command. Together with the finding of similar tPA and tPV between these two groups, the parallel kinematic profile of the early stages of double-step reaching (where demands on predictive modeling are greatest), provides additional support for the immaturity model of atypical predictive modeling in DCD. Importantly, these parallels cannot be explained by a shared general motor deficit since children with DCD are able to perform simple (non-jump) reaching at age-appropriate levels, nor can they be explained in terms of shared slowing in information processing since RT was faster in children with DCD than younger controls.

Late control parameters

Post-Correction Reaching Time.

Developmental trends. PCT was significantly longer for younger compared to age-matched controls. As I discussed in the previous chapter, demands on ROC are considered minimal throughout this stage of the movement cycle since the target remains stationary. Assuming that the recalibrated motor command is accurate, sensory inflow should match the forward models predictions about the limb-target relationships throughout the remainder of the movement. Accordingly, these
results suggest more efficient reaching in age-matched controls even when demands on predictive modeling are minimal. This result accords with the finding of slower non-jump reaching in younger controls where, again, demands on ROC are minimal. Taken together, slower PCT in younger children probably reflects a general level of immaturity in the motor system (Rival, Olivier, & Ceyte, 2003).

Not only is the internal feedback system more refined in adults compared with older children, faster PCT suggests a higher level of general maturation of the motor system which affords significant functional advantage for simple reaching and actions that are not subject to perturbation. In adults, these movement types are characterized by highly stereotyped kinematics- i.e. relatively smooth bell-shaped velocity profiles (Coluccini, Maini, Martelloni, Sgandurra, & Cioni, 2007) and virtually straight-line reaching trajectories (Farnè et al., 2003).

**Children with and without DCD.** Like Study 2, children with DCD were able to complete the post-correction phase of movement as efficiently as age-matched peers, and were faster than younger controls. Similarly, the DCD group were faster than younger controls when reaching for stationary targets. Taken together, the ability to conduct simple reaching when demands on rapid online control are minimal appears to be well developed in children with DCD. Interestingly this result indicates that immaturity of the motor system in DCD, at least with respect to reaching, may be isolated to those mechanisms responsible for the ROC (i.e. predictive modeling).

**Summary of kinematic analysis.** With respect to typical development, kinematic analysis verifies that efficient double-step reaching across development may, at least in part, to be subserved by an increasing capacity to implement predictive modeling strategies; in particular, TC values decreased incrementally between younger controls, age-matched controls and younger adults. Also, only
young adults showed evidence of a mature fast internal feedback system (shown by a decrease in tPV for jump trials) suggesting that while predictive modeling systems see considerable maturation by the ages of 8 to 12 years, continued refinement continues into adolescence and early adulthood.

While some aspects of the motor system appear to have developed at age-appropriate levels in children with DCD (i.e. those necessary for simple reaching), the similar atypical profile demonstrated by children with DCD and younger controls in the early stages of double-step reaching (in particular on TC) suggest shared difficulty correcting the motor command using predictive models of movement in-flight and supports the developmental immaturity model of atypical predictive modeling in DCD.

**Error.**

**Anticipatory Errors.** Interestingly, no difference on AEs were shown between younger and age-matched control children; however, the effect size was moderate. As discussed in earlier chapters, this type of error reflects either difficulty inhibiting the initiation of inappropriate movement or difficulty maintaining finger position on the home base. The moderate effect shown supports the bulk of evidence suggesting a watershed in executive functioning (including inhibition) at around 8 years of age concomitant to unfolding of the frontal cortices and its upstream connections (Huizinga, Dolan, & van der Molen, 2006; Korkman, Kemp, & Kirk, 2001). That group differences did not reach significance likely reflects a mixture of the modest sample size of both groups as well as a floor effect on AEs (since even in young controls, AEs occurred on ≈3% of trials). Accordingly, it may be that the group mean differences on AEs were not large enough to be detected using the current design, a notion supported by the moderate effect size.
Age-matched controls tended to show more anticipatory errors than young adults \((p = .08; \text{partial } \eta^2 = 0.10)\), suggesting a reduced capacity for inhibiting unwanted movements. This finding accords with neuro-developmental models of executive functioning which show continued unfolding of response inhibition over adolescence and into early adulthood (Davidson, Amso, Anderson, & Diamond, 2006; Huizinga et al.; Korkman et al., 2001).

No differences in AEs were observed between DCD and control groups suggesting an equal capacity to maintaining finger position on the home-base or inhibit the initiation of inappropriate movement. While this result accords with the findings of study 2, in light of available evidence suggesting impaired inhibition in DCD [for example, on measures of executive functioning (Piek et al., 2007a), and during the COVAT (Mandich et al., 2003; Wilmut et al., 2007) and ‘Simon’ task (Mandich et al., 2002)], as noted in the previous chapter, future research would benefit from investigating the particular parameters under which difficulties with inhibition impact motor performance in DCD.

**Centre touch error.** Younger control children showed a non-significant trend \((p = .06)\) for increased CTEs compared to age-matched controls. As noted in Study 1 and 2, this type of error may reflect difficulties inhibiting on-going (yet inappropriate) movements in younger controls. An increased capacity for cognitive control (i.e. inhibition) in 8 to 12 years olds compared to 5 to 7 years olds is consistent with neuro-developmental models of motor and cognitive development (Hauizinga et al., 2006; Korkman et al., 2001).

Interestingly, with respect to CTEs, age-matched controls were as accurate as adults. These results suggest an equal capacity for inhibiting an on-going movement in adults and children between the ages of 8 and 12 years. Intriguingly, this finding is
at odds with general consensus that inhibitory control continues to mature from childhood into adolescence and adulthood (Hauizinga et al.; Korkman et al., 2001). Again, I argue that the lack of difference here may be reflective of the modest sample size employed as well as a floor effect since the mean number of errors committed by both groups was relatively low (i.e. on average < 1). Accordingly, group differences may not have been large enough on this particular indicator of inhibitory control to be detected using the current design.

Finally, as was found in Study 1 and Study 2, children with DCD experienced significant difficulty correcting their reaching on jump trials prior to touching the initial (central) target compared to controls (no difference was observed in comparison to younger controls). As discussed in Chapter 2, the suggestion of inhibitory difficulties in children with DCD has been canvassed previously (i.e. Mandich et al., 2002; Wilmut et al., 2007) (see Chapter 1, for a detailed discussion).

**Touch down error.** As expected, younger controls committed significantly more TDEs than age-matched controls; this trend for increased movement accuracy between 5 and 12 years is commonly reflected in the developmental literature (Favilla, 2006). Age-matched control children showed a non-significant ($p = .07$) trend for increased TDE compared to young adults. Thus, as might be expected of a mature motor system, the faster reaching demonstrated by adults was also more accurate. Again, this increase in motor proficiency from childhood and into adolescence and early adulthood is reflected in the motor literature (Rival et al., 2003).

Finally, children with DCD committed significantly more TDEs than age-matched control children yet were equally as accurate as younger controls. This result mirrors those found in Study 1 and Study 2 and supports a significant body of
research showing less accurate goal-directed reaching in children with DCD (Wilson et al., 1997).

**General discussion**

**Atypical ROC (viz predictive modeling) in DCD reflects a developmental immaturity.** Similarities in the kinematic performance profiles of children with motor difficulties and younger typically developing children supports the hypothesis that developmental immaturity explains the poor ROC (i.e. predictive control) of children with DCD. Developmental immaturity has been proposed as an explanation for a number of motor difficulties commonly associated with DCD [such as poor postural control (Wann et al. 1998), praxis skills (Hill and colleagues) and COVAT (Wilmut et al., 2007)]. For example, Hill (1998) showed that children with DCD aged 7 to 13 years and younger controls (5 to 7 years) experienced similar difficulty repeating familiar hand gestures when compared to age-matched controls. Additionally, Hill and colleagues (1998) demonstrated that children with DCD aged between 7 and 13 years and younger control children (aged 5 to 6 years) made significantly more errors than age-matched controls during the production of gestures completed to verbal command, both transitive (involving functional use of an object e.g. teeth brushing) and intransitive (i.e. not involving functional use of an object, e.g. a wave to someone). In short, the evidence presented here supports an increasing body of work suggesting that atypical motor performance in children with DCD may reflect developmental immaturity.

Interestingly, children with DCD and younger controls both demonstrate a pattern of difficulty correcting their reaching on jump trials similar to that seen in PPC patients. All groups show a decreased capacity to correct their reaching
trajectory in response to unexpected target perturbation (Gréa et al., 2002, see chapter 3). In some adult neurological patients, the impaired system can generate movement that is similar (though not entirely identical) to a healthy, yet neurologically immature system (see Chen, 1995). However, impairment in PPC patients is clearly far more severe since these patients often present with an inability to correct reaching in-flight at all, rather than decreased efficiency (see Chapter 3). Accordingly, I do not propose that the PPC is damaged in either children with DCD or younger controls but rather that inefficient ROC (and predictive modeling) shown by the two groups may involve immaturity at the level of the PPC.

Limitations

It is acknowledged that there is some overlap between the samples comprising the DCD and age-matched control groups presented in Study 2 and 3 of this thesis (i.e., 13 of the 18 children comprising the DCD and control groups carried over from the corresponding samples from Study 2). However, the results of these additional participants mirrored that of the Study 2 sample on the key markers of predictive control. Furthermore, the primary goal of Study 3 was to clarify the immaturity/deviance issue of atypical predictive control in children with DCD. Accordingly, it was the comparison of DCD with younger controls that was of principle interest to the present study, and fitting performance along a developmental trajectory from immature to mature (viz healthy adults) movement.

Furthermore, consistent with Study 1 and 2, analysis of individual differences suggested impaired online control in the majority of children with DCD. However, double-step reaching was preserved in roughly 20% of this group- i.e., their mean $MT_{Diff}$ and TC scores fell within the 95% CI of age-matched controls, suggesting age-
appropiate predictive modeling. Thus, while there is compelling evidence to suggest that most children with DCD may experience difficulties employing predictive modeling strategies, the deficit is clearly not universal. The implications of these findings for the development of a unified account of DCD are discussed in detail in the next and final chapter.

**Summary**

In Study 3, I showed that typically developing children (8 to 12 years) were able to account for unexpected target perturbation far more efficiently than younger children (5 to 7 years); this trend continued into early adulthood. Kinematic analyses of the early stages of reaching revealed that more efficient double-step performance throughout childhood and then into early adulthood was subserved, at least in part, by an increased capacity to compare forward estimates of limb position with sensory inflow and integrate subsequent error information with the ongoing motor command (shown by faster TC scores with typical development). Interestingly, young adults demonstrated the capacity for very early error detection (illustrated by a condition dependent decrease in tPV) not demonstrated by any child group. Thus, while the predictive control system that supports the rapid correction of reaching online achieves a degree of proficiency between the ages of 8 and 12 years, continued development occurs into adulthood.

Finally, with respect to children with DCD, the early kinematic profile of double-step reaching shown by this group paralleled that seen in younger control children (i.e. both demonstrated a similar decreased capacity to correct the trajectory of their reaching during jump trials manifest by similar TC scores). I argued that these similarities suggest a shared difficulty in using predictive estimates of limb position to
update motor commands in-flight (i.e. predictive modeling), which supports the developmental immaturity model of DCD. Clinical implications are discussed in the next chapter (Chapter 5).
CHAPTER 5

GENERAL DISCUSSION
Chapter 5  General Discussion

General Discussion

Summary

The aim of this thesis was to clarify the nature of rapid online control in children with DCD using a neuro-computational framework. To this end, a detailed investigation of double-step reaching was conducted as a way of exploring one of the more prominent aetiological accounts of DCD—the IMD hypothesis. This approach is based on the assumption that ROC utilizes a predictive estimate of limb position to correct movements in-flight. In this concluding chapter, I first provide a brief summary of the three studies presented earlier; here I highlight the consistent finding across studies of impaired ROC in DCD. Further, the results of Study 3 support the hypothesis that this impairment may reflect a developmental immaturity.

I will also highlight the important contribution that this thesis makes in clarifying the nature of ROC in children with DCD and, more broadly, the relationship between ROC and skill acquisition. I then discuss the implications of this thesis for the validation of the IMD hypothesis and the development of a unified account of DCD. The putative neural basis of impaired predictive control in DCD is then discussed followed by clinical implications of the findings of this thesis and directions for future research.

Summary of Studies 1, 2 and 3

Study 1 – Chronometric evaluation of double-step reaching in DCD. Rapid online control was investigated in children with DCD and age-matched controls using a tightly controlled double-step reaching paradigm. Children with DCD were disadvantaged by unexpected target perturbation at movement onset (manifest by slower, less accurate reaching). Based on the assumption that ROC is only viable to
the extent that predictive (feedfoward) and feedback based mechanisms can be integrated seamlessly, I suggested that these results reflected difficulty using predictive models of movement.

Because of the limitations of chronometric analysis, I was unable to determine the phase in reaching where control mechanisms were impacted; specifically, whether impairment occurred early in the reach trajectory on jump trials (where online control demands are greatest) or later in the cycle (i.e. post reach correction) when rapid adjustments are minimal. This issue was addressed in Study 2.

**Study 2 – Combined chronometric and kinematic analysis of double-step reaching in DCD.** This study used the same stimulus display and response requirements as Study 1, but incorporated kinematic markers of performance. The goal of kinematic analysis was to determine whether double-step reaching was impacted early or later in the reaching trajectory in children with DCD. The logic behind this design was that demands on ROC (and predictive modeling) are greatest early in the reach cycle where large-scale corrections to hand trajectory are required. Hence, impairments in predictive control would manifest during this phase of the movement. The pattern of movement time seen in Study 1 was replicated: children with DCD experienced difficulty correcting their movement in response to unexpected target perturbation. Kinematic analysis showed no group differences on tPA and tPV, but children with DCD were significantly slower to correct reach trajectory away from the initial target on jump trials. This pattern of performance suggested some impairment in the integration of error information (generated as a result of discrepancy between the expected sensory effects of the movement as specified in the forward model and the actual sensory consequences of reaching) into the unfolding motor command. By comparison, the later (post-correction) stages of
reaches to jump targets were not impaired in DCD. Consistent with Study 1, these results suggest that children with DCD have a specific problem in making early, rapid online adjustments, based on forward estimates of limb position. What remained unclear, however, was whether the atypical pattern of performance in DCD reflects deviance from the typical developmental trajectory or a delay/immaturity.

**Study 3 – Atypical ROC in DCD: developmental delay or deviance?** To examine the issue of developmental deviance versus delay, the performance of children with DCD (8 to 12 years) was compared to an age-matched control group, a group of younger control children (5 to 7 years), and a group of young adults (20 to 28 years). The profile of double-step reaching in DCD was shown to be similar to younger controls. Chronometric data showed that both groups were slower and less accurate on jump trials compared with age-matched controls. Kinematic analysis replicated the pattern seen in Study 2 which was also mirrored in younger controls: time to correction in DCD and younger children was delayed relative to age-matched controls. Age comparisons showed clear developmental progression, with young adults performing more optimally than all groups of children. By comparison, the post-correction phase showed no differences between the DCD group and age-matched controls. Taken together, parallels between the reaching of DCD and younger control children support the hypothesis that a developmental immaturity may best explain impaired ROC in DCD.

**Summary.** To summarize the above studies, it appears that children with DCD experience a reduced capacity for implementing rapid online corrections in response to unexpected visual perturbation. From a neuro-computational framework, delay in maturation of the parietal cortex or parieto-cerebellar axis may explain the difficulties these children have in making the early and rapid adjustments that are
thought to rely on forward estimates of limb position (i.e. predictive modeling). The specific control issue appears to be one of integrating error information with the on-going motor command. In the next section, I discuss these findings in relation to earlier research on motor control in DCD, as well as the broader implications of my data for theory.

Implications for understanding ROC

Results from this thesis have important implications for understanding the development of ROC in children generally, and the nature of ROC deficits in DCD, specifically. Of the former, my set of studies is one of the few to map clear developmental trends in ROC using a visual perturbation paradigm. With respect to DCD, the work can be readily interpreted from the perspective of the IMD framework of Wilson and colleagues (1997, 1999, 2001, 2004; Williams et al., 2006, 2008). These implications for normative development and DCD are discussed in turn.

ROC across typical development. Study 3 is one of very few studies to provide evidence on the development of online control during reaching across childhood (5 to 12 years), as well as mature performance in young adults. When interpreted from a computational perspective, this work supports the developmental progression of the motor system suggested in earlier work on simple reaching (Chicoine, Lassonde, & Proteau, 1992; Ferrel et al., 2001; Hay, 1979; Pellizzer & Hauert, 1996). Children between the ages of 5 and 7 years were significantly constrained by unexpected target perturbation compared to 8 to 12 year olds, while adults were somewhat more efficient than the oldest group of children. In keeping with previous research, this pattern of results suggests that use of predictive modeling is refined significantly between the ages of 8 and 12 years (see Chicoine et al. 1992;
Hay, 1979; Hay & Redon, 1999; Pellizzer & Hauert, 1996), but still undergoes further refinement between later childhood and early adulthood (see Ferrel et al., 2001; Konczak et al., 2003). What we know about the maturation of motor centres (in particular parieto-cerebellar networks) supports this pattern of development. While the premotor and primary motor cortices reach maturity in the early years of life, key association cortices including the parietal and frontal regions follow a more protracted development, reaching maturity at around 10 years and late adolescences, respectively (Casey, Tottenham, Liston, & Durston, 2005). Dorsal-dorsal routes and links between the parietal cortex and cerebellum are thought to subserve the fast internal feedback loops that support early error detection. The gradual unfolding of these networks is supported by data showing that only in young adults are significant reductions in tPV observed on visual perturbation trials, relative to unperturbed (See Chapter 4), suggesting early error detection (Castiello et al., 1991; Farnè et al., 2003; Paulignan et al., 1991).

**Impaired ROC in DCD: Implications for the IMD hypothesis.**

In Chapter 1, I argued that the theoretical and experimental framework adopted by the current thesis held great promise in clarifying the aetiology of DCD, specifically the IMD hypothesis. Briefly, the IMD hypothesis holds that impaired motor control and learning in children with DCD may result from difficulties either generating and/or monitoring internal (predictive) models of action. As noted, this hypothesis is based on converging methods including measures of MI (Williams et al., 2006; Wilson et al., 2004), DSST (Katschmarsky et al., 2001), VGPT (Wilson et al., 2001), and an intervention study (Wilson et al., 2002). However, there are some conflicting lines of evidence bearing on the IMD hypothesis. For example, some
work on MI suggests atypical timing of imagined limb rotation (Wilson et al., 2004) while others suggest relatively preserved timing but less accurate (Williams et al., 2006), or slower performance (Deconinck et al., 2009). I argued that a neuro-computational investigation of double-step reaching might clarify the nature of predictive control in DCD. Next I provide a detailed explanation of the implications of the results of this thesis for the IMD hypothesis.

Prior to the initiation of goal-directed reaching visual and proprioceptive signals are used to estimate the initial state of the limb while visual coordinates estimate the prospective target location (Desmurget, Pélisson, Rossetti, & Prablanc, 1998). The nervous system uses this information to generate an accurate motor command to achieve the desired goal-state. At movement onset, a corollary discharge encodes a copy of this command (via an efference copy) which is used by the predictive (forward) model to anticipate how the movement will unfold in relation to the coded target location and specifically, its expected sensory consequences. From a neural perspective, a functional loop between the parietal lobe and cerebellum is thought to be involved in monitoring and comparing these forward estimates of limb position with the real-time sensory consequences of movement (Blakemore & Sirigu 2003; Shadmehr & Krakauer, 2008)- the neural basis of predictive modeling is discussed in greater detail below. In the case of mismatch, an error signal is generated and used to correct the on-going motor command. Specifically, online corrections to movement are implemented by “superimposing” a dynamic error signal onto the ongoing feedforward motor command (Gritsenko et al., 2009). This form of motor control circumvents time delays associated with the processing of sensory feedback which can take upwards of 250 ms (Frith et al., 2000). This control system is crucial to maintaining the stability of the unfolding movement since the position of the
moving limb may have changed considerably by the time sensory signals have been encoded and can be used to correct the ongoing motor command. This model fits well with recent evidence showing that sensory feedback can contribute to online control throughout the movement cycle (Saunders & Knill, 2003; 2005), rather than simply towards the end of movement (ala the old dual-component view).

In the case of double-step reaching, a forward (predictive) model of the limb-target relationship is compared to the actual sensory consequences of reaching throughout the reaching cycle. The unexpected target perturbation causes incongruence between the expected (according to the forward model) and actual consequences of movement. Successful correction of reaching trajectory towards the new target is dependent on the resultant dynamic error signal being integrated with the unfolding feedforward motor command. Impairment in this process manifests as slower, less accurate double-step reaching and specifically, inefficient correction of reaching trajectory towards the new cued target. Conversely, since the target remains stationary for the remainder of the movement cycle (i.e. post reaching trajectory correction) provided that the re-calibrated motor command is accurate, reaching can unfold largely on the basis of the motor plan with minimal adjustments and, thus, demands on predictive modeling are comparatively low. Hence, a specific deficit in predictive modeling would not be expected to impact these latter stages of the reaching cycle.

Children with DCD experienced difficulties correcting the trajectory of their reaching towards the new target, yet were able to complete reaching as efficiently as controls once these corrections had been implemented. Accordingly, this consistent double-step reaching profile of children with DCD observed across the three studies of this thesis provides strong evidence that children with DCD experience difficulties
using predictive estimates of limb position to correct their reaching trajectory (ala the IMD hypothesis).

As noted earlier in this thesis, however, computational models of motor control have been a relatively recent theoretical advancement. Traditional models of motor control, such as the dual-component model for example, instead propose that the control of goal-directed reaching occurs in two distinct stages: first, a ballistic, feedforward stage, under guidance from the initial motor command, followed by a honing-in phase under feedback control (see Elliott et al., 1999). This is based on the assumption that time constraints prevent sensory feedback from influencing the initial stages of reaching, i.e. prior to tPV. Applying this framework to the findings of the present thesis, since there were no differences between DCD and age-matched control groups for tPV in any study, the present data would suggest that children with DCD are able to control those aspects of reaching that are principally under the guidance of the initial motor command as well as their control peers. It also follows that those with DCD are able to generate and carry-out a simple motor command at age-appropriate levels. Conversely, since movement time post tPV was longer in DCD, it would appear that they spent significantly longer in the ‘feedback’ based stage of reaching. This result would suggest difficulties using sensory feedback mechanisms to guide reaching in the latter phase of the movement (as per Plumb and Colleagues, 2008). A deficit using feedback-based mechanisms would also explain the longer time to movement trajectory correction in DCD since this kinematic marker occurs post tPV (and hence during the feedback-based stage of movement) and is dependent on the successful registry and implementation of sensory feedback. However, un-like recent computational models proposed (such as the predictive modelling framework discussed above), traditional dual-component models are unable to account for recent
evidence that sensory feedback can contribute to online control throughout the movement cycle (see Saunders & Knill, 2003; 2005), rather than simply towards the end of movement. It is for this reason that I argue that the predictive (internal) modelling framework offers the more reliable framework for interpreting the double-step reaching profile of children with DCD.

As I discussed in the introductory chapter of this thesis, a number of studies outside Wilson and colleagues laboratory have either directly, or indirectly, implicated impaired predictive modeling in motor control issues associated with DCD. These studies highlight the role that the impaired predictive might play in a variety of control issues characteristic of DCD— for example, difficulties controlling posture (Jover et al., 2010; Przysucha et al., 2007), grip-force (Pereria et al., 2001) and drawing (Smits-Engelsman et al., 2003). The validity of any aetiological account of DCD is judged on the weight and type of research that supports it. While a number of questions remain unanswered regarding the exact role of predictive modeling in skill acquisition (these are discussed below) and the reciprocal role of neural development (also discussed below), taken in the context of the larger body of evidence on the IMD hypothesis, the consistent finding of impaired ROC across all three studies which comprise this thesis provides compelling evidence for the notion that predictive modeling may be compromised in DCD. That is not to say that impaired predictive modeling is universal to all children with DCD. Indeed, online control was preserved in a sub-group of roughly 20% of children in each DCD sample presented here (demonstrated by similar MT\_DIFF and TC scores to age-matched controls) suggesting age-appropriate predictive modeling. Given the highly heterogenous nature of DCD symptom expression, this finding is not unexpected. Indeed, it would be unrealistic to expect any neuro-cognitive account to capture the
motor difficulties experienced by all children with DCD. Nonetheless, this body of evidence does provide strong support for the suggestion that predictive modeling is likely to be compromised in the majority of children with DCD. Next I, I draw on current neuro-computational theory to consider possible neural loci for this difficulty.

**Toward a unified account of DCD: the neural basis of impaired predictive modeling.** Converging measures have highlighted the role of the posterior parietal cortex (PPC) in online control and forward modeling. It has been suggested that the PPC (and its downstream connections) may be the principle network for processing predictive models of action (for a good review, see Desmurget & Sirigu, 2009), and is strongly implicated in the organization and execution of goal-directed reaching (Andersen & Buneo, 2000; Buneo & Andersen, 2006). More specifically, the PPC is thought to subserve integration of forward estimates of limb endpoint with optic flow about the dynamic properties of the limb trajectory, and generation of the error signals by which the unfolding movement command can be modulated, online (Diedrichsen et al., 2005; Mulliken, Musallam, & Andersen, 2008; Shadmehr & Krakauer, 2008). This notion is supported both by neuroimaging work on healthy adults during double-step reaching (Grafton, Schmitt, van Horn, & Diedrichsen, 2008) as well evidence showing that online corrections to reaching are impaired when the PPC is interrupted via TMS (Desmurget et al., 1999) or lesioning (Blangero et al., 2008; Gréa et al., 2002). For example, Desmurget and colleagues showed that in healthy adults, TMS of the left PPC at movement onset disrupted right arm trajectory corrections on jump trials, yet did not effect the accuracy of simple non-jump trials.

Importantly, the deficits in target-directed reaching in patients with PPC lesions (specifically, optic ataxia) are prominent for tasks involving target perturbation and other responses that rely heavily on predictive modeling (Glover,
2003; Gréa et al., 2002; Pisella et al., 2000). It is argued that this problem reflects difficulty integrating feedback-based information with the efference copy (viz internal modeling) (see Glover, 2003). Like the DCD children I tested, impaired online control in patients with PPC lesions is inferred from elevated movement time in response to target jumps (Gréa et al., 2002). By comparison, responses are relatively preserved for simple reaching in patients with bilateral PPC lesions suggesting that, at least with regards to motor control, the PPC may be specifically involved in comparing the estimated limb trajectory (according to the predictive model) with optic in-flow (i.e. online control) during visually guided movements and may not be as important for actions that can unfold largely unchanged on the basis of the initial motor command. As well, kinematic data has revealed that these patients have a propensity to reach towards the original goal and then generate a second movement towards the new target location, compared to the smooth corrections observed in healthy adults. This evidence provides further support for the notion that the PPC may be involved in comparing feedforward limb estimates with actual sensory signals and in the case of mismatch, generating the error signal which is then used to correct the ongoing motor command. Clearly, the pattern I observed in DCD was not so severe as to prevent trajectory changes altogether, as is often the case in PPC patients (i.e. Gréa et al., 2002, see Chapter 3). Furthermore, the comparison between the double-step reaching performance of children with DCD and PPC patients offered here is correlational and therefore can not be argued as grounds to suggest the same (neurological) causal basis. Accordingly, I do not propose that performance similarities between children with DCD and PPC patients are indicative of parietal lesioning in clumsy children. Instead I argue that they support the notion of possible
neurodevelopmental delay at the level of the PPC (Desmurget & Grafton, 2003; Desmurget & Sirigu, 2009).

Interestingly, the pattern of performance seen in DCD on other tasks measuring predictive control is also reminiscent of that observed among patients with PPC lesions, notably people with ideomotor apraxia, a disorder associated with left parietal lobe damage (Buxbaum, Johnson-Frey, & Bartlett-Williams, 2005). These studies include motor imagery (Wilson et al., 2004; Williams et al., 2006), oculomotor control (Katschmarsky et al., 2001), and orienting of visuospatial attention (Wilson et al., 2001). In the case of MI, for example, patients with parietal damage show atypical reaction times similar to those seen in children with DCD during imagined finger movements (Sirigu et al., 1996; Wilson et al., 2001). Specifically, while actual movement during the VGPT appears to conform to the speed-accuracy trade-off described by Fitt’s Law in both parietal patients and DCD, imagined movement does not [c.f. healthy adults (Sirigu et al., 1996) and children (Wilson et al., 2001); see Chapter 1]. In the absence of confirmatory neuroimaging, however, whether the PPC is a site of developmental deviance or delay in DCD remains a question for continued focus.

**Cerebellar contributions to predictive control.** Predictive control is subserved by a distributed neural network. In particular, like the parietal lobe, the cerebellum has also been implicated in the generation and monitoring of internal models of movement (see Blakemore & Sirigu, 2003). Indeed, cerebellar impairment has been proposed by earlier work as a potential neural basis for movement problems in DCD. This is based on research highlighting symptom similarities between cerebellar patients and children with DCD. Specifically, impaired movement timing, atypical postural control and difficulties alternating movement have all been
associated with DCD and linked to cerebellar dysfunction (for a good review see Zwicker, Missiuna & Boyd, 2009).

*Cerebellar contributions to control and learning.* In Chapter 1, I clarified the important distinction between the role of predictive modeling in motor control and motor adaptation (or learning)- the cerebellum is crucial for both. For example, in real-time response to mechanical perturbations to the moving limb (e.g., Shadmehr et al., 2010) and in adjusting to displaced visual information over repeated learning trials under conditions of visual adaptation (Redding et al., 2005).

The distinction between the role of predictive modeling in motor control and adaptation is also supported by neuroimaging and neuropsychological evidence which suggest distinct, yet overlapping, neural substrates. With respect to motor control, ascending cerebellar pathways are thought to monitor the somatic consequences of movement, detecting discrepancy between the predicted dynamic properties of the limb according to the unfolding motor command with actual behavior and has been referred to as a ‘somatic detector’ (Miall & King, 2008). Miall and King recently asked healthy adults to reach steadily towards their right with limb and target vision occluded at movement onset. After a random interval of 500-1500ms, participants were informed via an auditory cue to either return their hand back to the initial position or upwards towards a virtual target. Coincidental TMS of the lateral cerebellum resulted in increased positional error and trajectory deviations. Interestingly, similar effects have been observed in cerebellar patients (for a good review see Molinari, Restucci, & Leggio, 2009). Thus, in conjunction with the PPC which serves as a visuo-spatial comparator of the predicted (according to the forward model) and actual limb trajectory, the cerebellum may serve to predict and monitor
the somatic consequences of movement as part of a wider parietal-cerebellar functional loop to support the online control of goal-directed reaching.

With respect to motor adaptation, evidence that patients with cerebellar damage display reduced or non-existent adaptation to movement when vision is undetectably displaced using prisms or cursor rotation suggests that the cerebellum may play a role in the adaptation of predictive models (Martin et al. 1996; Tseng, Diedrichsen, Krakauer, Shadmehr, & Bastian, 2007)- this notion is further supported by neuroimaging evidence (Diedrichsen et al., 2005). Interestingly, as highlighted in Chapter 1 children with DCD display a similar decreased capacity for adapting their movement following prismatic (Cantin et al., 2007) and cursor rotation (Kagerer et al., 2006) as cerebellar patients providing additional evidence that this area may be impacted in children with DCD.

Interestingly, while patients with lesioning to the posterior inferior cerebellar artery demonstrate impaired motor adaptation following prismatic visual displacement yet preserved motor control (Martin et al., 1996), damage to the superior cerebellar artery, cerebellar thalamus (Martin et al., 1996) and PPC (Pisella et al., 2004) appears to result in impaired online control (ataxia) yet preserved adaptation. Importantly, this double-dissociation supports the view that the processes of motor control and adaptation may be supported by distinct neural networks.

*The neurophysiology of predictive control in DCD.* Only a limited number of neurophysiological studies of DCD have been conducted that bear on the IMD hypothesis. In particular, two recent neuroimaging studies provide evidence of disruption at the level of parietal and motor regions. For example, Kashiwagi and colleagues (2009) recently conducted fMRI of visually-guided pointing performance in 12 boys with DCD and 12 age-matched controls. They demonstrated that a slower
and less accurate performance profile shown by the DCD group compared to age-matched controls was associated with less activation of the left PPC and post central gyrus. Zwicker et al., (2010) also showed that children with DCD activate different neural regions to controls during simple fine motor control (i.e. tracing an outline of a flower). Specifically, it appeared that children with DCD rely on visuo-spatial processing networks while control children activate more anterior regions associated with motor initiation and control (see table 5.1). These results were suggested to possibly reflect greater reliance on visual processing to control movement rather than the predictive model in children with DCD compared to controls. Furthermore, while no behavioural differences existed between the groups, children with DCD activated almost twice as many neural regions as controls suggesting greater effort was required to complete the task.
Table 5.1. Neural regions more actively employed by DCD and control children respectively during a simple drawing task (Zwicker et al., 2010).

<table>
<thead>
<tr>
<th>DCD</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left inferior parietal lobule</td>
<td>Left precuneus</td>
</tr>
<tr>
<td>Right middle frontal gyrus</td>
<td>Left superior frontal gyrus</td>
</tr>
<tr>
<td>Right supramarginal gyrus</td>
<td>Right superior temporal gyrus, insula</td>
</tr>
<tr>
<td>Right lingual gyrus</td>
<td>Left inferior frontal gyrus</td>
</tr>
<tr>
<td>Right parahippocampal gyrus</td>
<td>Left postcentral gyrus</td>
</tr>
<tr>
<td>Right posterior cingulate gyrus</td>
<td></td>
</tr>
<tr>
<td>Right precentral gyrus, R medial frontal gyrus</td>
<td></td>
</tr>
<tr>
<td>Right cerebellar lobule VI</td>
<td></td>
</tr>
<tr>
<td>Right superior temporal gyrus</td>
<td></td>
</tr>
</tbody>
</table>

Interestingly, while functional neuroimaging of DCD performance during tasks requiring motor adaptation (or any other motor task which predominantly employs the cerebellum) is lacking, recent structural MRI of a 19 year-old female with DCD showed structural abnormalities in the cerebellum supporting the notion that the cerebellum may play a role in DCD (Mariën et al. 2010). Specifically, the patient showed atypical fissuration of the anterior vermis which is consistent with rostral vermis dysplasia. It should be noted though that the clinical significance of this form of dysplasia is not well understood (Mariën et al.).

Finally, a recent EEG study by Lust and colleagues (2006) failed to show differences in sensitivity between children with DCD and controls during the mental rotation of limbs. It should be noted that as opposed to Wilson et al., (2006), Williams
et al. (2006) and Deconinck et al., (2009) behavioural differences between groups were not observed which the authors suggested may have resulted, in part, from the small sample size (DCD= 10, controls= 7) and may also reflect the heterogeneous nature of the DCD population. Whether neural sensitivity differences exist between those children with DCD who display atypical MI and controls is yet to be determined.

Clearly, more neurophysiological data is needed to clarify the neural underpinnings of DCD; adult models are often the basis for drawing inferences about motor control and learning in children. Since the cortical regions of most interest in the present group of studies, the parietal and frontal cortices, follow a more protracted developmental trajectory than other brain regions— the current consensus being that the parietal lobe approaches maturity between the ages of 10 and 12 years while the frontal lobe continues to mature well into the late teens (Casey et al. 2005; Lenroot & Giedd, 2006) – these regions are still unfolding in the children I tested. The upshot is that it is important to temper any conclusions drawn about the integrity of these emerging neural networks in DCD, particularly when performance trends are interpreted with reference to mature adults on similar tasks. Neuro-imaging data is required to better understand how these networks change with age in both typical and atypical development.

**How might a deficit in predictive modeling manifest for children with DCD?**

Successful completion of many everyday motor tasks depends on the motor systems capacity to rapidly correct an on-going movement according to changing environmental or task parameters—e.g., the ability to correct grasp when a cup is heavier than anticipated, to check a soccer kick because a team mate unexpectedly
changes position or to correct our step should an aspect of the ground differ from initial expectation (i.e. the depth of a step, texture of a surface). All such scenarios are dependent on the nervous system’s ability to detect error between the expected and actual sensory consequences of movement and integrate this information with the ongoing motor command. Accordingly, problems of motor prediction in DCD shown in this thesis and elsewhere (e.g. Williams et al., 2006; Wilson et al., 2001) may account, at least in part, for the difficulties these children have interacting with their physical environment (Missiuna et al., 2008) and adapting to different task constraints (Astill, 2007; Utley & Astill, 2007).

Importantly, the ramifications of impaired predictive modeling occur over longer timescales and extend to motor learning itself. As noted, the error signal that results from any discrepancy between the forward estimate of limb position and that indicated by actual sensory feedback is used to not only recalibrate the on-going movement, but also to refine, or adapt, the predictive model over repeated trials, thus increasing the accuracy (and repertoire) of future predictions (Assaiante, Chabeauti, & Sveistrup, 2010; Wolpert, 1997). Changes in prediction can then also be used to inform and refine the inverse model (Flanagan et al., 2003). This process of refining predictive models provides a cogent framework for explaining differences in skill acquisition (see Wolpert et al., 2001), including some of the difficulties children with DCD have in refining movement despite repeated practice (Marchiori et al., 1987; Utley & Astill, 2007). Put another way, part of this resistance to practice/exposure might be a reduced ability to update predictive models over trials. This would manifest as a repeated pattern of poorly executed (goal-directed) movement with hypo- or hypermetric qualities. For example, Marchiori and colleagues (1987) showed that even after 1,200 practice trials over a six-week period, a group of boys
with DCD displayed minimal improvement in hockey shooting. Conversely, control
children demonstrated proficient shooting without training. This idea is supported by
a growing body of work showing similarities in the performance profile of children
with DCD and younger controls on motor tasks like hockey shooting, (Marchiori et
al., 1987), praxis performance (Hill et al., 1998), and even postural control (Wann et
al., 1998).

Clinical implications of this research

The notion that developmental immaturity may best explain poor ROC (i.e.
predictive control) in children with DCD (see Chapter 4) has several implications for
therapy; these are discussed in the following section. Most importantly, it suggests
that these children possess the capacity to develop age-appropriate levels of motor
competence, though intervention remains key to achieving this end. MI training is
discussed as a promising framework for facilitating development of age-appropriate
predictive modeling in DCD and improving motor skill more broadly. Implementation
issues and recommendations are discussed.

Treating the immature system. Based on the suggestion that impaired
ROC in DCD reflects a developmental immaturity, it is assumed that these children
possess the capacity to ‘catch-up’ to their same-age peers (van Braeckel et al., 2010).
In contrast, were impaired ROC thought to reflect a deviance from the normal
developmental trajectory, a pattern of continued impairment might be expected
(McConnell, 1998). Thus, broadly speaking, the goal of any intervention designed to
address impaired ROC (or predictive modeling more generally) in DCD should focus
on assisting children develop age appropriate levels of motor competence rather than,
for example, promoting the use of compensatory strategies to minimize the impact of impairment on everyday functioning (van Braeckel et al., 2010).

We should, however, be mindful that while the capacity for an immature neural system to ‘catch-up’ to age-appropriate levels might exist, it is by no means assured. There is a growing awareness of the importance of experiential learning in neural plasticity and development (Galvan, 2010; Johnson, 2011; Mundkur, 2005). Indeed, even in adults where neural plasticity is thought to decline, there is evidence that regular physical activity increases neural plasticity and improves motor learning and rehabilitation (Cirillo, Lavender, Ridding, & Semmler, 2009). Taken with evidence that children with DCD tend to adopt sedentary lifestyles (Missiuna et al., 2008b; Poulsen et al., 2007), it may be that the lifestyles which these children stereotypically adopt might limit their capacity to gain the requisite motor experience needed to facilitate neural development and motor rehabilitation. This notion is supported by longitudinal evidence showing that without intervention motor impairment (and secondary psycho-social difficulties) in DCD generally persists or accentuates through to early adulthood (Cantell et al., 1994, 2003; Losse et al., 1991; Missiuna et al., 2007). Thus, there is a clear need for intervention to remediate impaired motor control in DCD.

**Can we train predictive control?** As discussed in Chapter 1, efforts to develop effective intervention in DCD have thus far been largely unsuccessful. With respect to impaired predictive modeling in DCD however, preliminary evidence has been somewhat more promising. A recent MI training intervention delivered through interactive CD-ROM was shown to be as effective at improving motor skills in children with DCD as perceptuo-motor training, possibly the most commonly adopted intervention strategy at present (Wilson et al., 2002). However, as the goal of their
study was to investigate wider implementation of the MI intervention, the criteria for motor impairment in the Wilson and colleagues study was motor function below the 50\textsuperscript{th} percentile as measured using the MABC. Thus, only 11 of 54 children performed below the 15\textsuperscript{th} percentile, the minimum quantitative cut-off for identification of motor difficulties using a standardized measure of motor competence (see Sugden, 2006). Given recent findings that children with more severe DCD benefit less from specific verbal MI instructions (Williams et al., 2008), additional work is necessary to determine the efficacy of MI training in improving more severe motor impairment. It may be, for example, that there is/are sub-group/s of children with DCD who are less responsive to verbal MI instructions (e.g. those with more severe DCD \textit{ala} Williams et al.) and that these individuals might benefit from an alternative medium of cueing/instruction (for example, visual), a plausible suggestion given the highly heterogeneous nature of the disorder. This notion is supported by evidence from the adult literature indicating that the efficacy of MI instructions may depend on the medium of cueing strategy (i.e. verbal or visual) and that there are inter- and intra-population differences surrounding which strategy is most effective. Recently, Hovington and Brouwer (2010) measured motor evoked potentials (MEP) during physical performance of finger abduction as well as imagined performance of this movement following either verbal, visual or combined cueing in a group of younger (20-35 years) and older (over 55 years) healthy adults and 10 adults with chronic stroke. Verbal MI instructions resulted in greater MEPs in older adults (and stroke patients) than either visual or combined cueing. Alternatively, MEPs were larger following visual cueing in younger adults. It is difficult to draw reliable inferences from this data since it is one of very few studies of its kind. Also, it is not entirely clear why certain cueing strategies resulted in greater MEPs in some groups and not
others (see Hovington and Brouwer for a discussion). Nonetheless, the upshot from this research is that additional work is required to determine firstly, whether verbal or visual cueing strategies (or a combination) best facilitate MI training in DCD; and secondly, whether this might be affected from the severity of motor impairment.

Implementing MI training: issues and recommendations. There are a number of broader clinical issues surrounding the use of MI as an intervention strategy; these are discussed in the coming paragraphs (for a good review see Malouin & Richards, 2010). A caveat to this discussion, however, is the importance of a child-centered approach to intervention. That is, in-keeping with current best practice principles, any intervention should aim to address the specific difficulties of the child in the context of their own strengths, weaknesses and desired treatment outcomes.

Firstly, though MI training alone often results in improved motor skill in adults, these improvements are typically enhanced when a combination of physical and imagined practice are implemented (Dickstein & Deutsch, 2007; Malouin & Richards, 2010). Accordingly, MI training should be used as an adjunct to, rather than replacement of, physical therapy.

In terms of imagery content, there is a growing body of evidence that task familiarity is important to the successful use of MI training (Dickstein & Deutsch). For example, Mulder, Ziljstra, Ziljstra, and Hochstenbach (2004) demonstrated that MI training only improved performance of a novel motor task (abduction of the big toe) in individuals who had obtained a degree of mastery of the task prior to MI training. Accordingly, MI training should, at least initially, involve mental rehearsal of tasks that are familiar to the child. In-keeping with a child-centered approach to intervention, these tasks should also be tailored so as to be of ecological relevance to the child and address goals specific to their needs (Dickstein & Deutsch).
Just as with actual movement, it has been suggested that imagery be taught progressively, beginning with simple and then extending to more complex movements (Short, Afremow, & Overby, 2001). This is particularly important in the case of DCD since more difficult movement is associated with increased imagery complexity (Dickstein & Deutsch, 2007) and there is strong evidence that children with DCD already experience difficulty employing MI without instruction/training (Williams et al., 2008). It is also suggested that imagery sessions be short to minimize fatigue and maintain motivation (recommendations range from 5-20 minute blocks, Dickstein & Deutsch, 2007; Short et al., 2001), and that in-keeping with best practice principles for physical therapy, sessions are structured and regular (Short et al.). How sessions might be structured and scheduled is considered in the coming paragraphs.

Research shows that children with motor difficulties benefit from increased practice trial numbers during physical intervention. For example, children with CP, a severe disorder of movement that in its milder form is often mistaken for severe DCD (for a good discussion on this issue see Pearsall-Jones, Piek, & Levy, 2010), require more practice trials for motor learning to occur than age-matched controls and rapid degradation of skill is observed without continued practice (see Garvey, Giannetti, Alter, & Lum, 2007 for a good review). How trial numbers might be tailored to increase the efficacy of MI training for children with DCD remains to be established. However, given evidence that children with DCD experience difficulties refining skills over repeated practice (Marchiori et al., 1987; Utley & Astill, 2007), it would seem reasonable to presume that greater trial numbers would be beneficial, though not the extent that children become fatigued or disinterested.

Other treatment factors may be of equal, if not greater, importance to treatment efficacy than trial numbers. For example, when multiple actions are being
practiced/learnt, whether or not sessions are blocked (i.e. practice continues for a
given movement until a level of proficiency is achieved, then a new movement is
practiced) or randomized (i.e. practice trials alternate between tasks intermittently)
may affect outcomes (Thomson, 2005). Though blocked trials show better short-term
benefits than randomized sessions, when retention is measured days or hours later,
randomized trials illicit improved retention than blocked designs (Schmidt, 1991).
These findings have led a number of clinicians and authors to suggest that given the
short-term efficacy of blocked-trial sessions, this form of training should be employed
when individuals are learning new skills. Alternatively, if a degree of proficiency
exists, sessions should be randomized to maximize long-term retention (see Thomson,
2005).

Furthermore, the amount of rest between practice sessions is a crucial factor in
treatment efficacy (Thomson, 2005; Trempe & Proteau, 2010). Research shows that
when therapy session length is equivalent, longer rest periods between sessions
facilitates performance and learning (Thomson, 2005). Thus, it is important that
children receive adequate rest between intervention sessions. Recent examples of
intervention programs for DCD that have elicited positive outcomes have scheduled
sessions on a weekly basis (Männistö, Cantell, Huovinen, Kooistra, & Larkin, 2006;
Wilson at al., 2002), though as symptom severity increases and the necessity for more
frequent therapy sessions also increases, it may not be feasible for intervals to be so
long.

Accordingly, while preliminary evidence supports the use of MI training as a
remedial strategy for improving predictive modeling (and motor skill more generally)
in children with DCD, continued investigation is clearly required to determine how
MI training might be administered in order to maximize treatment outcomes (i.e. are
visual, verbal or a combination of the two cueing strategies more effective), as well as whether symptom severity might influence such clinical decision making. Also, given the heterogeneity of the disorder, it is likely that some children with DCD who experience predictive modeling difficulties may not benefit from MI training, irrespective of whether a verbal or visual cueing strategy is used. For this reason, there is need to explore alternative intervention avenues which target predictive control difficulties in DCD.

Is impaired predictive control the cause or a symptom of movement problems? The current thesis raises the important issue as to whether impaired predictive movement is the cause of motor difficulties in children with DCD or whether it is a symptom of it. Research suggests that children with DCD tend to adopt sedentary lifestyles (Missiuna et al. 2008b; Poulsen et al., 2007). While the gradual proliferation of motor related associative cortices throughout childhood and into adolescence plays a crucial role in motor development, neural development is in turn shaped by the interaction between the developing child and their environment (Blakemore & Choudhury, 2006; Casey et al., 2005). The upshot here is that acquisition and refinement of motor skill is as dependent on exposure to a range of physical activities, environments and scenarios as it is on the integrity of cortical development. Hence, it can be argued that immature predictive modeling in DCD may reflect limited exposure to physical activity; in other words, the “immaturity” may be a consequence of the motor impairment and not its cause. I will argue in the next section that this hypothesis is unlikely.

If decreased physical activity were responsible for immature predictive modeling in children with DCD, we would expect the integrity of this system to be similar to that of individuals who have had a similar, but limited exposure to such
activities (i.e. younger control children). As the results of Study 3 of this thesis attest, there are indeed similarities in the ability of children with DCD and younger control children to implement predictive modeling strategies. However, if decreased physical activity were the cause of these problems in DCD, then it would also be reasonable to expect that other areas of motor control would be equally impacted. Thus, the general profile of motor performance, and not just those reliant on predictive modeling, would also be expected to show immaturity. As shown in Study 3, children with DCD were able to complete simple reaching at age-appropriate levels and were generally faster than younger controls. That is, the immature motor profile demonstrated by children with DCD was selective, confined to those aspects of movement reliant on predictive modeling. This same pattern of selective impairment has been demonstrated in previous studies of online control in children with DCD. For example, Wilmut and colleagues (2006) showed that children with DCD demonstrate age-appropriate simple reaching to a single target yet increased movement time and decreased accuracy when required to reach to sequential targets. Here, the authors reported increased foveation time prior to reaching onset. This would likely disrupt use of the predictive model since it would need to be ‘buffered’ until movement onset. Accordingly, as foveation time increases, the predictive model is rendered less accurate as task and environment change. Thus, it would seem unlikely that the atypical double-step reaching of children with DCD (from which I inferred a deficit in predictive modeling) is a consequence of decreased physical activity.

Furthermore, I have noted the important role that predictive modeling is thought to play in motor learning. Briefly, if movement inaccuracies continue over repeated trials, the error signal which arises when the expected (according to the predictive model) and actual sensory consequences of movement do not match can be
used to update the predictive and inverse models iteratively (Shadmehr & Krakauer, 2008). Accordingly, over repeated practice trials in healthy individuals we would expect to see incremental improvements in movement predictions and more accurate motor commands - i.e. more proficient actions. As I noted earlier, even when children with DCD are exposed to repeated trials of physical activity such as hockey shooting (Marchiori et al., 1987) or ball catching (Utley & Astill, 2007) they continue to experience difficulty organizing their movements. This finding would therefore suggest difficulties employing predictive modeling strategies. This notion is further supported by evidence that skill acquisition improves in children with DCD following MI training which specifically aims to improve predictive modeling abilities (Wilson et al., 2002).

**Directions for future research**

A major issue for future investigation is to map how individual differences in predictive modeling might predict skill acquisition and motor competence in children. This involves investigating both the relationship between the integrity of predictive control and the level of motor skill in children with DCD, as well as differences in control as a function of maturation or age (i.e. typical motor development). Next, I propose that better understanding the impact of frequently co-occurring developmental disorders on predictive modeling in children with DCD is important to achieving this end and may have implications for the diagnosis and treatment of DCD. I also highlight the limitations associated with using traditional double-step reaching tasks to isolate the contribution of predictive modeling mechanisms to online control. I then discuss the need for continued investigation into remedial efforts as well as validation of alternative neuro-cognitive accounts of DCD.
Establishing the relationship between the severity of impaired predictive modeling and motor difficulties. An interesting area that is important to clarifying the relationship between predictive modeling and motor skill level relates to the investigation of the relationship between the severity of impaired predictive modeling in children with DCD and motor competence. Recent evidence from imagined movement in DCD has demonstrated that the magnitude of MI impairment might be related to the severity of motor impairment. Here, investigation of MI in children with mild and severe DCD showed increased MI deficit in children with severe DCD compared to mild (Williams et al., 2008). Interestingly, children with severe DCD also demonstrated a decreased benefit from MI instruction compared to mild DCD. This work highlights firstly, that since, from MI performance we can reliably infer the integrity of internal (predictive) modeling (see Chapter 1), the capacity for predictive modeling is not uniform across DCD samples; secondly, that there may be a relationship between the severity of impaired predictive control and motor impairment; and finally, that symptom severity may mediate intervention efficacy. This latter point has particular significance for treatment planning since some presentation types may be more resistant to remedial efforts than others.

Longitudinal investigation will better elucidate the relationship between predictive modeling and motor competence. While the cross-sectional, between-groups design adopted in this thesis provides important insight into motor development, carefully designed longitudinal research is required to better investigate the relationships between the development of predictive modeling and motor competence in both typically and atypically developing children. Statistical methods such as growth curve modeling (GCM), for example, allow investigators to track linear and non-linear developmental trends across multiple observation points.
Importantly, in keeping with contemporary developmental theory, researchers can map changes at both a between-person and within-person level while investigating the impact of covariates. In the context of the development of predictive modeling in DCD, this latter point is particularly important since the effects of frequently co-morbid issues such as ADHD and LD on motor competence can be investigated. While a detailed analysis of GCM is beyond the scope of this thesis, briefly, they offer a number of advantages over traditional longitudinal statistical methods (i.e. mixed-model and repeated measures ANOVA) by permitting analysis of change at the individual level, flexibility in testing schedules (i.e. assessment can occur at different times points rather than analysis assuming all participants were assessed at the same time \( \text{viz ANOVA} \)), the ability to use data from participants who have missing data, and, finally, the capacity to generalize modeling to non-normal data (DeLucia & Pitts, 2006).

**The impact of co-occurring disorders.** Motor impairment often presents co-morbidly with developmental disorders such as ADHD and learning disabilities (LD) [i.e. Specific language disorder (SLD), Reading Disability (RD)]. As I discussed in Chapter 1, there has been considerable debate as to the aetiological basis of this overlap with a number of theories questioning the usefulness of conceptualizing DCD, ADHD and LD as unitary disorders, instead suggesting that these respective syndromes may reflect manifestations of a single disorder taking the form of a generalized non-specific brain impairment (i.e. *Atypical Brain Development Theory*, Kaplan and colleagues, 1998; *Deficits in Attention, Motor Control and Perception (DAMP) Theory*, Gillberg and colleagues; *Automatization Deficit Hypothesis*, Visser, 2003). Unfortunately, these theories typically offer little scope for isolating causal mechanisms for targeted intervention and for this reason have largely been
unsuccessful in producing effective remediation programs (see Chapter 1 for a more detailed analysis). As a result, each syndrome is currently conceptualized as a unitary disorder.

Despite consensus that co-morbidity is the rule rather than the exception (Kaplan et al., 1998), the effect of co-morbidity on DCD symptom expression (and intervention efficacy) has largely been ignored in the literature. Recent evidence suggests that DCD symptom expression exacerbates in the presence of co-occurring ADHD and LD (Jongmans, Smits-Engelsman, & Schoemaker, 2003; Jucaite, Fernell, Forssberg, & Hadders-Algra, 2003) and that symptom severity is further mediated by the number of co occurring disorders (Crawford & Dewey, 2008). This highlights the need for continued work into the impact of co-morbidity on perceptuo-motor difficulties in children with DCD (including predictive modeling). For example, Jongmans and colleagues (2003) demonstrated that children with co-morbid DCD and LD showed significantly greater perceptuo-motor difficulties than children with DCD; these children had specific difficulty performing manual dexterity and balancing tasks. More recently, Crawford and Dewey found that while children with DCD displayed preserved visual-perceptual functioning, when DCD presented co-morbidly with either ADHD or reading disability (RD), children showed impaired visual perceptual performance. Interestingly, the severity of this deficit increased with the number of co-occurring disorders (i.e. DCD+ADHD+RD).

Given high co-morbidity levels, another important issue is whether the constellation of neuro-cognitive impairment in ‘pure’ cases of DCD might differ from that seen in children with co-morbid conditions. Though the motor impairments in pure DCD and co-morbid (for example DCD/ADHD) may be qualitatively similar, it is nonetheless possible that they are subserved by separable neuro-cognitive issues.
For example, atypical predictive modeling may explain impairment in some pure cases of DCD yet the attentional and inhibitory difficulties that are often associated with ADHD might contribute to poor movement in co-morbid cases (Lewis, Vance, Maruff, Wilson, & Cairney, 2008). This notion is supported by a recent study by Lewis and colleagues that demonstrated that the atypical response times that are characteristic of imagined movement in DCD (from which impaired predictive modeling is inferred) were evident in children with pure DCD but not co-morbid DCD/ADHD suggesting that while predictive control issues may contribute to motor difficulties in pure cases of DCD, motor impairment in co-morbid DCD/ADHD may emanate from a distinct neuro-cognitive difficulty.

Thus, despite preliminary evidence indicating that co-morbid developmental disorders may exacerbate DCD symptomatology and that pure and co-morbid cases of DCD may be characterized by distinct neuro-cognitive deficit, additional work is required to explore these possibilities (specifically with respect to the integrity/ role of predictive modeling in pure and co-morbid cases of DCD). Given the frequent co-occurrence of these disorders with DCD, whether co-morbidity is subserved by separable aetiology to pure DCD and/or impacts symptom severity and expression has important implications for motor screening and treatment planning.

**Limitations of traditional double-step reaching tasks.** As I have argued throughout this thesis, the well-validated traditional double-step reaching task offers great insight into the integrity of online control. I have also argued that current neuro-computational modeling provides the most valid interpretive framework for double-step reaching analysis. In accordance, I have proposed that the slower double-step reaching and inefficient trajectory corrections shown by children with DCD may reflect a decreased capacity for implementing predictive modeling mechanisms. It
should be noted, however, that this profile of double-step reaching can also be explained using alternative interpretive frameworks. Most importantly, in Chapter 1 I discussed evidence suggesting that children with DCD may experience difficulties inhibiting shifts of visual attention from the location of invalid cues and re-directing it towards valid target locations (for example, during the COVAT, Mandich et al., 2003; Wilson & Maruff, 1999; Wilson et al. 1997; See Chapter 1). Accordingly, it is plausible that the slower time to correction and slower jump trial reaching observed in children with DCD may reflect difficulties inhibiting/disengaging visual attention from the location of the central target and re-directing it towards the newly cued peripheral target location rather than difficulties correcting the on-going motor command through feedback-based mechanisms- *ala* the predictive/internal modeling account that I have proposed.

Unfortunately, the experimental paradigm adopted in the current thesis does not allow one to precisely distinguish between the possible contributions of predictive control difficulties and/or disinhibition of attention to double-step reaching. In order to address the issue, I propose that future research adopt a modified version of the double-step paradigm. Specifically, after completing the traditional double-step task, children could undertake an additional modified version of the task aimed at specifically investigating their ability to inhibit and shift attention from an invalid to valid spatial location (see Boulinguez & Nougier, 1999). This second condition would involve identical task parameters to the traditional double-step reaching task for non-jump trials. That is, upon presentation of the central target children would be required to reach and touch it as quickly as possible. The initial stages for jump trials would also be the same as for the traditional double-step task. However following target perturbation children would not be required to reach toward the newly cued target and
instead would cease their movement and respond vocally stating that the target had jumped to the left or right periphery as quickly as possible (Boulinguez & Nougier, 1999). A successful jump trial response would require that children disengage attention from the central target location and re-direct it towards the correct peripheral location. However, since no actual re-direction of the moving limb towards the new target would take place, demands on predictive modeling would be minimal. Accordingly, vocal responses to target perturbation would provide insight into the integrity of attentional inhibition/disengagement systems relatively independent of predictive modeling capacities (Boulinguez & Nougier, 1999). Since actual double-step reaching requires attentional disengagement/inhibition from the central to peripheral target but (according to computational modeling) also places great demands on predictive modeling mechanisms, comparison of the performance of children with DCD during the actual reaching and vocalized response versions of the double-step reaching task would allow one to disentangle the role/s of predictive modeling mechanisms and attentional inhibition/disengagement in the slower double-step reaching demonstrated by children with DCD. For example, if effect size comparisons for children with DCD during the traditional and vocalized double-step reaching tasks showed that they were similarly disadvantaged when physically and vocally responding to unexpected target perturbation compared to controls, it would suggest that the slower reaching trajectory corrections demonstrated by those with DCD might result predominantly from difficulties disengaging attention from invalid to valid target locations. This is because both versions of the task require attentional inhibition/disengagement; hence a deficit would manifest in performance of both versions of the task. However, while vocal responses to target perturbation would place minimal demands on predictive modeling mechanisms, actual re-direction of
reaching trajectory would place great demands on this system. Thus, if DCD children experienced both difficulties disengaging attention from invalid to valid target locations and employing predictive modeling mechanisms, then the latter difficulty would presumably compound performance deficits during actual double-step reaching. Hence, actual re-direction of reaching following target perturbation would be differentially affected compared to a vocalized response.

Despite the limitations of the double-step reaching task adopted here, I nonetheless argue that on the balance the predictive modeling deficit hypothesis offers the most cogent framework for explaining the performance of children with DCD. In support, the current paradigm contained measures of both online control- which I have argued is dependent on the integrity of predictive modeling mechanisms (i.e. MT Diff score and time to correction; see Chapter 3)- and attentional inhibition/disengagement (i.e. CTE; see Chapter 2). The overall effect sizes for group comparison on the key measures of online control were far larger than for the key indicator of attentional inhibition. Specifically, the effect sizes for MT Diff score and time to correction were very strong (partial $\eta^2$ typically exceeded 0.30), while comparatively moderate for CTE (partial $\eta^2$ was generally around 0.10). Accordingly, while it is plausible that a deficit in attentional inhibition might contribute to the slower time to correction and jump trial performance in DCD, in the present thesis it does not appear to explain the extent of the online control deficit demonstrated by children with DCD. Still, additional work is necessary to more accurately differentiate the contributions of predictive modeling and/or inhibitory/attentional difficulties in the slower trajectory corrections observed in children with DCD.

**Intervention research.** As I discussed earlier in this chapter, efforts to develop effective remediation of clumsiness have thus far been largely unsuccessful.
One avenue which has provided promising preliminary results is an imagery training protocol administered via DVD (Wilson et al., 2002). While MI training provides a promising avenue for improving predictive modeling in DCD and might thusly prove effective in improving motor control and learning in children with DCD, only 11 of the 54 children who comprised the motor impaired group presented with movement below the minimum quantitative cut-off for diagnosis of DCD (i.e. the 15th percentile) in Wilson and colleagues’ formative study. Given evidence suggesting that the efficacy of intervention in children with DCD might be mediated by symptom severity (Williams et al. 2008) additional work is required to determine the circumstances under which MI may prove effective in improving predictive (forward) modeling in DCD. Earlier, I highlighted evidence that MI cueing strategy (i.e. visual or verbal) might influence training efficacy and that different groups find different cueing strategies more effective (Hovington & Brouwer, 2010). Future work must identify how MI training can be structured to promote improved predictive modeling and motor skill in DCD and whether intervention needs to be tailored according to the severity of impairment. For example are visual, verbal or combined MI cueing strategies most effective at improving motor difficulties in DCD and is this issue mediated by symptom severity?

Furthermore, in Chapter 1, I highlighted the distinction between two broad categories of intervention for DCD: process-oriented and task-oriented (Wilson, 2005). For a more detailed discussion, see chapter 1, but briefly, process-oriented approaches presume that motor difficulties are the result of deficit at the level of putative processes or mechanisms that are thought to be important to age-appropriate movement. Intervention focuses on trying to ameliorate these deficits to improve motor function. Alternatively, task-oriented approaches aim to alleviate difficulties
that children experience with specific tasks such as walking, or handwriting. Though historically, *task-oriented* approaches have enjoyed greater empirical success (see Chapter 1), recent technological and methodological advances in the cognitive neurosciences have provided the field with greater scope for understanding the reciprocal relationship between the development of brain, cognition and action (Wilson, 2005). Using this framework, a number of empirically supported brain-behavior models have been developed, each explaining the neuro-cognitive basis of one, or a group, of motor deficits typical of DCD—such as the IMD hypothesis (Wilson et al., 2004; Williams et al., 2006, 2008), impaired movement timing involving the cerebellum (Castelnau et al., 2007; Lundy Ekman et al., 1991), and disinhibition of motor responses related to fronto-striatal circuitry (Mandich et al., 2002). Importantly, each of these models offers scope for targeted intervention. Though clearly limited, preliminary evidence of contemporary process-oriented interventions based on these brain-behavior models has been promising (i.e. MI training to improve predictive modeling and skill acquisition in DCD, Wilson et al., 2002). A key issue moving forward is whether these, and other, brain-behavior models can continue to be refined to the point that they can form the basis of valid and accessible intervention programs. Alternatively, will ecologically based task-oriented intervention approaches continue to provide clinicians’ with (arguably) the more effective framework?

**Alternative aetiological accounts of DCD.** Given the heterogeneity of DCD, it would clearly be remiss to suggest that addressing predictive modeling difficulties will resolve the motor issues experienced by all children with DCD. Indeed, a sub-group of roughly 20% of children with DCD in each of the samples that I employed were able to complete double-step reaching with similar efficiency to age-matched
controls (as indicated by their mean MT\textsubscript{Diff} and TC values being within age-matched control CI\textsubscript{95\%} limits) suggesting preserved predictive modeling. In light of strong evidence suggesting the existence of sub-groups of DCD (Hoare, 1994; Macnab et al., 2001; Tsai et al., 2008), it is likely each is subserved (at least in part) by distinct neuro-cognitive deficits. While few neuro-cognitive accounts of DCD have been subjected to as rigorous experimental scrutiny as the IMD account of DCD, I have highlighted a number of plausible alternative accounts in Chapter 1 which show promise in clarifying the aetiological basis of motor difficulties in DCD [i.e. impaired temporal control related to the cerebellum (Lundy-Ekman et al., 1991; Castelnau et al., 2007); impaired inhibitory control implicating fronto-striatal circuitry (Mandich and colleagues, 2003)]. While each of these theories enjoys a degree of empirical validity, many lack rigorous and independent validation simply by virtue of their recent inception. Hence, validation of these alternative theories is dependent on continued investigation. The importance of continued investigation lies in the fact that these brain-behavior models will form the basis of future process-oriented intervention development. Furthermore, since each theory attempts to explain a cluster (or type) of motor symptom experienced by children with DCD, an interesting question is whether the nature of neuro-cognitive impairment provides adequate grounds for sub-typing children with DCD. In other words, are sub-types of DCD subserved by distinct profiles of neuro-cognitive deficit? This issue has important implications for assessment and screening of clumsy children. Also of great relevance to
Summary and conclusion

The studies in this thesis have demonstrated that children with DCD suffer from a reduced capacity to implement corrections online; this evidence has clarified disparate findings in the developmental literature surrounding the nature of ROC in DCD (see Wilmut et al. 2006 and Plumb et al 2008). Interpreted from a modern neuro-computational perspective, results suggest that children with DCD may experience difficulties using predictive estimates of limb position in order to adjust movement commands, in-flight (i.e. predictive modeling). Specifically, these children may experience difficulties correcting movements in real time on the basis of error information which arises from the disparity between the expected consequences of the movement (modeled with respect to the initial target position) and actual sensory information arising from the visual perturbation. Furthermore, similarities in the double-step reaching profile of children with DCD and younger controls suggest that atypical ROC (and hence predictive modeling) in children with DCD may reflect a developmental immaturity involving the PPC, or parieto-cerebellar axis more broadly. Importantly, taken in the context of earlier work on predictive modeling in children with DCD (Williams et al., 2006, 2008; Wilson et al., 2001; 2004), the results of this thesis provide compelling support for the view that predictive modeling may be impaired in children with DCD and represents another small step towards validation of the IMD hypothesis.

The suggestion that impaired predictive modeling might reflect a developmental immaturity indicates that children with DCD likely possess the capacity to attain age-appropriate levels of predictive modeling (and motor skill), though intervention remains paramount to achieving this end. Accordingly, best practice principles from physical therapy indicate that treatment might best be focused
at helping children obtain age-appropriate levels of motor skills (c.f. developing compensatory strategies to minimize impairment). MI training was discussed as a promising intervention framework for improving predictive modeling and motor skill in DCD. However, despite supportive preliminary evidence on the efficacy of MI training at improving skill acquisition in DCD (Wilson et al., 2002), continued investigation is required to better establish the conditions under which MI training might prove most effective at ameliorating motor difficulties in DCD, and whether these might be mediated by symptom severity.

Directions for future research were also considered. Importantly, I suggested the need for longitudinal work to better establish how individual differences in predictive modeling might explain motor competence. Furthermore, I argued for continued investigation into the impact of commonly co-occurring developmental disorders such as ADHD and LD on symptom presentation in DCD and for future work to investigate whether ‘pure’ and co-morbid (e.g. DCD/ADHD) cases of DCD might be characterized by distinct neuro-cognitive impairment. Limitations associated with using traditional double-step reaching tasks to isolate the contribution of predictive modeling mechanisms to online control were also discussed and suggestions for future investigation proposed.

Finally, while data presented in this thesis provides strong evidence that atypical predictive modeling may contribute to poor ROC in DCD, it is implausible that all children with DCD suffer from an underlying deficit in predictive control. Indeed, ROC was preserved in a small sub-group of the DCD samples that I assessed. This heterogeneity is a hallmark of DCD and demonstrates the need for continued investigation into alternative neuro-cognitive accounts of DCD and alternative treatment methods.
In conclusion, the studies presented in this thesis provide compelling evidence that ROC is impaired in children with DCD. From a computational perspective, these results may reflect a reduced ability to implement predictive modeling strategies, as per the IMD hypothesis. This difficulty is likely to reflect immaturity involving parieto-cerebellar cortices.
References


*Neuropsychologia, 44*, 2594-2606.


*Adapted Physical Activity Quarterly, 11*, 115-129.


*Developmental Science, 9*, 1-20.


References


References


References


References


References


References


Appendix A. Published Articles

Online motor control in children with developmental coordination disorder: chronometric analysis of double-step reaching performance

C. Hyde and P. Wilson

Discipline of Psychology, School of Health Sciences, RMIT University, City Campus, Melbourne, Australia

Accepted for publication 20 April 2010

Abstract

Background Although there are a number of plausible accounts to explain movement clumsiness in children (or developmental coordination disorder (DCD)), the cause(s) of the disorder remains an issue of debate. One aspect of motor control that is particularly important to the fluid expression of skill is rapid online control (ROC). Data on DCD have been conflicting. While some recent work using double-step reaching suggests no difficulty in online control, others suggest deficits (e.g., based on sequential planning). To help resolve this debate, we suggest two things: use of recent neuro-computational models as a framework for investigating motor control in DCD, and more rigorous investigation of double-step reaching. Our working assumption here is that ROC is only visible through the seamless integration of predictive (or forward) models of movement and feedback-based mechanisms.

Aims The aim of this chronometric study was to explore ROC in children with DCD using a double-step reaching paradigm. We predicted slower online adjustments in DCD based on the argument that these children manifest a core difficulty in predictive control.

Methods Participants were a group of 17 children with DCD and 32 typically developing children aged between 7 and 12 years. Visual targets were presented on a 17-inch LCD touch screen inclined to an angle of 15° from horizontal. The children were instructed to press each target as it appeared as quickly and accurately as possible. For 80% of the trials, the central target location remained unchanged for the duration of the movement (non-jump trials), while for the remaining 20% of trials, the target jumped at movement onset to one of the two peripheral locations (jump trials). Reaction time (RT), movement time (MT) and reaching errors were recorded.

Results For both groups, RT did not vary according to trial condition, while children with DCD were slower to initiate movement. Further, the RT of children with DCD was prolonged to a far greater extent on jump trials relative to controls, with a large effect size. As well, children with DCD committed significantly more errors, notably a reduced ability to inhibit central responses on jump trials.

Conclusion Our findings help reconcile some disparate findings in the literature using similar tasks. The pattern of performance in children with DCD suggests impairment in the ability to make rapid online adjustments that are based on a predictive (or internal) model of the action. These results pave the way for future kinematic investigation.

Keywords developmental coordination disorder, double-step reaching, temporal modeling, online motor control

Correspondence Peter Wilson, Discipline of Psychology, School of Health Sciences, RMIT University, City Campus, PO Box 247, Melbourne, Vic. 3001, Australia. E-mail: paxwilson@rmit.edu.au

© 2010 Blackwell Publishing Ltd

222
Introduction

Movement clumsiness in children (or developmental coordination disorder [DCD]) is not a trivial condition, with an estimated prevalence of between 5 and 10% (Frith et al. 2003). Despite this, the underlying causes of DCD remain poorly understood. From a cognitive neuroscience perspective, recent experimental work has examined the function of motor control networks that might be compromised in DCD. The hypotheses to emerge are both interesting and varied: impaired interhemispheric communication (Simmondsen et al. 1999), poor movement timing associated with cerebellar dysfunction (Williams et al. 1992; de Castellano et al. 2007), impaired motor adaptation perhaps implicating multimodal integration (Kagerer et al. 2004) and impaired predictive control (namely, internal modeling deficit; Wilson et al. 2004; Williams et al. 2006, 2008).

These different perspectives underline the importance of continued experimental work.

One aspect of motor control that has been associated with DCD and that is particularly important to the development of skill is rapid online control (ROC). Data on DCD, however, have been conflicting. While some recent work using double-step reaching suggests no difficulty in online control (e.g., Plumb et al. 2008), others suggest deficits (e.g. based on sequential pointing; Wilmot et al. 2006). To help resolve this debate, we propose: (i) the use of current neuro-computational models as a framework for investigating DCD; and (ii) a more controlled study of double-step reaching. Our working assumption here is that ROC is only visible through the seamless integration of predictive (or forward) models of movement and feedback-based mechanisms. This argument is used to help explain some of the irregularities we see in children with DCD in terms of their adjustments to target perturbation.

ROC in children with atypical motor development (DCD)

One of the mainstays of the investigation of ROC has been the perturbation paradigm, specifically the so-called double-step paradigm (DSP). Participants are required to reach and touch (or grasp) one of a number of possible targets. For the majority of trials, the target remains the same for the duration of the movement, while for a small number of trials, the target changes (or ‘jumps’) unexpectedly at, or shortly after, movement onset; hence, the participants must adapt their movement ‘on the fly’. This approach has a rich history in the cognitive neurosciences, informed initially by traditional dual-process models of motor control (see Elliott et al. 2001), but more recently by computational models (Castello et al. 1996; Desmurget et al. 1999). Whereas traditional dual-process models view motor control as occurring in independent feedback (or ballistic) and feedback (or forward) phases, cognitive neuroscience models now view movement as being controlled by an integrated system: a view supported by a number of studies illustrating that corrections to movement can be made within the order of 100 ms of movement onset (Castello et al. 1996; Paulignan et al. 1999; Furne et al. 2003), more quickly than (external) feedback-based mechanisms are able to accommodate. Briefly, in the case of ROC, a fast internal feedback system is used to correct movements in flight, with minimal lag (e.g. Desmurget & Grafton 2000, 2003). ROC is visible to the extent that the nervous system can predict the future location of the moving limb using a forward (internal) model that integrates efferent and afferent signals (Wolpert 1997; Desmurget & Grafton 2003; Jeannerod 2002). Forward models use the input of a motor command to the ‘plant’ to predict the position of the body. This type of control circumvents delays associated with processing sensory feedback because the position of the moving hand has changed appreciably by the time sensory feedback can be used to alter the unfolding motor command. For instance, using a double-step (or perturbation) task where targets jump by up to 10° at movement onset, trajectories can be altered smoothly and early, detectable in as little as 30–40 ms after lift-off (e.g. Desmurget & Grafton 2003), and not with two distinct movements (as the dual process model). Moreover, these corrections can occur in the absence of any non-visual sensory information (e.g. Bard et al. 1999), as seen in de-afferented patients, or without vision of the moving hand. Forward models are thought to contribute to movement control by anticipating the sensory consequences of movement, allowing the motor system to detect discrepancies between predicted movement and that generated by actual sensory inflow; error signals are thus generated to correct the ongoing motor command (Desmurget & Sirigu 2009).

To our knowledge, only Plumb and colleagues (2008) have used a double-step reaching paradigm to compare the performance of children with DCD and controls, with interpretation based largely on a dual-process model. The authors split movement into feedback- and forward-back stages; time to peak speed was suggested to represent the point at which reaching ceased to be under forward-control, with feedback-based mechanisms controlling the remainder of the movement occurring in phase. This interpretation is based on the assumption that time constraints prevent feedback mechanisms from influencing the initial stages of reaching as per dual-process models. The children were required to stand and complete the task using a
hand-held stylus. However, the task was modified for children with DCD who had difficulty managing the stylus and performing the task from a standing position. While children with DCD took longer to complete the task (mainly, movement time (MST)) and spent more time in the declarative (feedback-based) phase of movement, there was no differentiable effect of condition (jump vs. no jump). The authors suggested that these results were indicative of a generalized deficit in producing movements rather than a specific online control problem.

However, this interpretation is flawed by the fact that the two groups were performing different versions of the same task; children with DCD were seated, unlike controls, and used a much thicker stylus. By the authors’ own admission, these modifications simplified the postural and grip demands of the task for the DCD group. It is well documented that children with DCD have particular difficulty completing complex movements (i.e. Wilson & McKenzie 1988; Wilmot et al. 2006). By simplifying the task for the DCD group alone, we argue that any comparison between conditions (jump and non-jump) is confounded. Further, the theoretical framework (als the dual-process model) does not acknowledge the growing concern that feedback mechanisms are integrated with feedforward mechanisms continuously throughout movement, and not just at the end point (Stauniers & Knill 2003, 2005).

**Double-step reaching: a window into ROC**

Two recent studies have investigated the performance of children with DCD on sequential double-step tasks, the first based on a computational framework (Katschansky et al. 2001; Wilmot et al. 2006). The first used an oscillement paradigm that required children to make rapid saccades to two targets that were presented sequentially — the double-step saccade task (DSTT). Katschansky et al. compared the performance of children with DCD and age-matched controls, arguing that impaired forward modelling in DCD would lead to dysmetria on the second saccade. Children first fixated a central point. The first target then appeared at a lateral location for 140 ms, followed immediately by the second (at another location) for 100 ms. The participants were required to generate sequential saccades from fixation to the first and then second target. Because the second target was extinguished before initiation of the first saccade, a spatial dissonance between the retinal coordinates of the second target and the required saccadic motor coordinates arises. Hence, in order to make an accurate second saccade, a forward model of the eye movement must be generated to predict the end point of the first eye movement. As predicted, the authors found that children with DCD were dysmetric on second saccades. Their performance suggested a reduced ability to program saccade sequences using a forward model, unlike age-matched controls. Although these results suggest an impaired ability to implement internal modelling strategies (ala the IMD hypothesis), the DSTT is an oscillatory paradigm and one that does not engage mechanisms for correcting movements rapidly, in flight.

More recently, Wilmot and colleagues (2006) investigated the coordination of hand and eye coupling during a sequential reaching task. Children with DCD and age-matched controls were required to reach for targets under three conditions: a single-touch condition during which children reached and touched a single target; a double-touch condition where participants touched two targets presented sequentially; and a ‘double-off’ condition, like the ‘double’ condition except that targets were extinguished 250 ms after movement onset. The results showed that while the two groups did not differ on simple aiming, children with DCD showed longer MTs and reduced accuracy when making sequential movements. Increased fixation time prior to movement in the DCD group suggested greater reliance on visual feedback control compared with controls. This increased fixation was also suggested to disrupt use of the efference copy which must be ‘buffered’ if fixation is prolonged: the longer the fixation, the less accurate the initial efference copy will be as the environment and task change.

The poor coupling between eye and hand movements is consistent with the slower, more variable reaching seen in children with DCD; however, the sequential pointing paradigm does not permit inferences about ROC. Rather, children could have completed the initial motor command in its entirety before then generating a second motor command to complete the two-step sequence (i.e. reaching from the first to the second target). That is, for double-step trials, the motor command for the first stage of reaching (i.e. reaching to the first target) does not need to be corrected after movement onset. Instead, a second motor command can be generated for reaching from the first and second targets. By comparison, rapid online corrections of the sort seen in double-step perturbation require in-flight modulation of the motor command.

The aim of the study presented here was to address the existing controversy about the nature of online control in children with DCD, entailing a double step reaching task. A chronometric approach was adopted here as a viable exploratory tool — deficits to online control are manifest as a selective delay in MT to perturbation trials (Desmurget & Grafon 2003). This pattern of performance is also evident in disorders that are known to impact forward modelling and ROC (e.g. optic ataxia seen in parietal patients; Grea et al. 2002; Glover 2003). Hence,
we predicted that children with DCD would display delayed MT and greater response errors on jump trials relative to typically developing children.

Methods

Participants

The sample consisted of 17 children (nine girls and eight boys) between the ages of 7 and 12 years who met the research criteria for DCD and 27 age-matched controls (13 girls and 14 boys). Mean age for the DCD group was 9.68 years (SD = 1.7), and 9.83 years (SD = 1.6) for the controls. All children were right-handed dominant with the exception of four children in the DCD group and one control child. The children were screened using a two-step procedure (as per Wilson et al. 1997). Principals from two state primary schools in suburban Melbourne were approached and invited to participate in the study. Classroom teachers from these schools identified children whom they considered to show poor movement skill for their age (i.e., they demonstrated significant difficulty completing everyday tasks such as handwriting, using classroom utensils (i.e., scissors, pencils, etc.) and/or physical education activities as per Diagnostic and Statistical Manual-IV diagnostic criteria, criterion B; this was corroborated by physical education teachers. These children were then assessed using the McCarron Assessment of Neuromuscular Development (MAND) (McCarron 1997); those scoring below the 10th percentile were included in the DCD group (see Greve et al. 2001) (criterion A). Exclusion criteria were a past or current diagnosis of ADHD or known learning, neurological or physical disorder (criterion D). As the children were recruited from mainstream primary schools and had not been diagnosed with a learning disorder, they were assumed to have IQ levels within the normal range (Greve et al. 2001) (criterion D). Control children were considered to have age-appropriate levels of motor skill by teachers and a MAND total score above the 20th percentile. The RMIT University Human Research Ethics Committee and Research Branch of the Victorian Government Department of Education and Early Childhood Development approved the research project, and all parents gave informed consent.

Apparatus

Double-step reaching paradigm

The DSPP was used to assess online motor adaptation. The Virtuoso Software Package (Dassault Systemes, Villeneuve d’Ascq, France) was used to develop the task display, which was presented on a 17-inch LCD touch screen (TFT, Necco, Incheon, Korea). The monitor was mounted at an angle of 15° from horizontal on a tabletop. The (black) surface surrounding the monitor was matched to the color of the monitor border and visual display to reduce the contrast within the participant’s visual field. The display consisted of a green circle at the bottom centre of the monitor which acted as a ‘home base’, and three yellow possible target locations (each 2 cm in diameter) presented in a semi-circular formation across the top of the screen. These possible targets were spaced 20° apart at the coordinates of –20°, 0° and 20° with respect to the ‘home base’. The distance between the centre of the home base and each target was 30 cm.

The participants were seated unrestrained on a height-adjustable chair in a dimly lit room. Lighting levels did not permit children to receive visual feedback of their moving limb (Farne et al. 2003). The index finger of their dominant hand was placed on the ‘home base’ which was positioned approximately 5 cm anterior to their midline. Their non-dominant hand rested comfortably beside the monitor. An assessor stood behind the participant throughout the task to ensure that they remained attentive and provided further instruction when necessary.

Procedures

At the beginning of each trial, the ‘home base’ was illuminated; the children were instructed to touch and hold the ‘home base’ with their (dominant) index finger. After a random interval of between 500 and 1500 ms, the home base was extinguished, which coincided with illumination of the central target. The children were required to reach and touch the middle of the target as quickly and accurately as possible, until it disappeared. To ensure that visual attention was initially oriented to the same location for each trial, the participants were instructed that the central target location would always be lit first, but that the target may jump to one of two other (peripheral) targets during movement (Castiello et al. 1998, 1999). During these jump trials, the children were asked to follow and touch the middle of the lit target. After each successful trial, the children were instructed to return to the ‘home base’ and wait the next target. Correct touches resulted in a short auditory tone, while no sound was emitted for incorrect touches. Where a trial resulted in error, the participants were instructed to continue touching the screen until they did so within the target boundaries and (hence) heard the tone.

For 80% of the trials, the central target remained illuminated for the duration of the movement. For the remaining trials (20%), the central target was initially illuminated, but at the
point of finger lift-off from the ‘home base’, the central target was
turned off; but re-appeared immediately at one of the two lateral
locations (jump trials). The participants were instructed to
reach and touch the target that was currently lit until the
auditory tone was emitted and the target extinguished.

Prior to the commencement of the task, the assessor mod-
elled the instructions for a jump and non-jump trial. The par-
ticipants then completed eight practice trials (six non-jump and
two jump). If the experimenter deemed it necessary, additional
practice trials were administered until it was clear that the par-
ticipants understood the instructions. The participants then
completed two blocks of 40 trials that were separated by a 1-min
break. Each block consisted of 32 non-jump trials and eight
jump trials (four to each side) which were presented in a
pseudo-randomized order.

Time-dependent measures were reaction time (RT), mea-
sured by the time between target display onset and finger lift-off
from the ‘home base’; and MT, measured by the interval
between lift-off and placement of the finger on the target loca-
tion. The difference between the mean MT for jump and non-
jump trials (MT - MT) was also calculated. Three types of response
error were also recorded: touchdown errors (TDEs) were
recorded when children touched outside the target boundary,
determined by the centre of pressure; anticipatory errors (AEs)
were recorded for responses that were initiated (i.e. lift-off from
the home base) before a target location was illuminated or for
RTs within 150 ms (see Wilson et al. 1997). Finally, target touch
errors (TTEs) were recorded for finger touches that were made
initially to the central target on jump trials.

Design and analysis

For each child, mean RT and MT, and total errors for each index
were calculated. The timed measures were based on legitimate
responses only; trials for which an error occurred were
removed. For jump trials, timed responses were collapsed over
target location (i.e. left and right). Preliminary analysis revealed
that condition (i.e. non-jump and jump trials) had no effect on
total number of AEs, and hence they were collapsed over jump
and non-jump trials. A criterion of 50% correct responses on
jump trials (or 8 out of 16) was deemed a minimum sampling
requirement. Of all correct responses, outliers were defined as
responses where RT or MT was >3 SD or <3 SD from the child’s
mean for that condition. For the error trials and outliers com-
bined, an average of 24 (33%) and 13 (16%) trials were removed
from the DCD and control groups, respectively. Data from five
children were not included in the analysis because they were
unable to understand the task rules.

Mean RT, MT and TDE data were compared between groups
for jump and non-jump trials using separate two-way analysis
of variance (ANOVA) (2 [group] × 2 [condition]). AE and TTE
were compared between groups using independent t-tests. Mea-
sures of effect size (partial η²) were used to temper the results of
significance tests. Finally, we also investigated the effects of age
on online control within each group. Here, we compared MT,AE
scores of younger (7–9 years) and older (10–12 years) children
within DCD and control groups.

Data were tested for violations of normality and homogeneity
of variance. For both MT and AE, tests of normality were
transformed using a logarithmic transformation. This transfor-
mation did not alter the effects of interest, and in light of
ANCOVA and t-tests being robust to violations of the assump-
tions of normality and homogeneity of variance (Lindman
1974; Tabachnik & Fidell 1996), the data were not transformed
for the final data set.

Results

RT

Mean RTs (±SE) for each group are displayed in Fig. 1. The
two-way ANOVA on mean RT revealed no significant interac-
tion between group and condition, F(1,9) = 0.60, P = 0.44, partial
η² = 0.01. Averaged across condition, the mean RT for children
with DCD and controls was 553 and 509 ms, respectively, and
was not shown to differ statistically, F(1,9) = 2.75, P = 0.10, partial
η² = 0.26. The main effect for condition was also not significant,
F(1,9) = 0.65, P = 0.43, partial η² = 0.01; averaged across group,
mean RT for jump and non-jump trials was 529 and 534 ms,
respectively.

![Figure 1: Mean reaction time (RT ± SE) for developmental coordination
disorder and control groups.](image-url)
Appendix A

Published Articles

6 C. Hyle and F. Wilson

MT

Analysis of group differences

Mean MT (±SE) for each group is shown in Fig. 2. The two-way ANOVA on mean MT revealed a significant interaction between group and condition, Wilk’s Λ = 0.75, F(2, 13) = 13.84, P = 0.001 and large effect size; partial η² = 0.25. Also significant was the main effect for condition, Wilk’s Λ = 0.03, F(2, 13811.98, P = 0.000, partial η² = 0.97. The main effect for group was not significant, F(2, 47) = 2.42, P = 0.06, partial η² = 0.08; averaged over condition, mean MT for the DCD and control groups was 690 and 634 ms, respectively.

Tests of simple effects showed no group difference for ‘non-jump’ trials, t(20) = 0.75, P = 0.06, partial η² = 0.01; however, children with DCD were shown to be significantly slower on jump trials, t(20) = 2.89, P = 0.01, partial η² = 0.17; mean MT for controls was 778 ms for ‘jump trials’ compared with 867 ms for the DCD group. The difference between the mean MT for jump and non-jump trials (MTdiff) was also compared between groups. The average MTdiff score for the DCD group was significantly higher than controls, t(20) = 3.72, P = 0.001, partial η² = 0.25, 355 ms compared with 285 ms.

Analysis of individual differences

Individual mean MTdiff scores within each group are presented in Fig. 3. There appeared to be a distinction between groups at approximately 300 ms with the majority of children with DCD exceeding this time and most controls under it. The C15% of controls was between 263 and 300 ms, compared with 325 and 382 for the DCD group. The MTdiff score for 13 out of 17 (76%) of the DCD group exceeded the upper C155% limit of the control group (or 300 ms).

Analysis of MTdiff scores within each group (DCD and control)

Independent t-test failed to reveal a significant difference for mean MTdiff scores between younger (7–9 years of age) and older (10–12 years of age) control children, t(20) = 1.23, P = 0.23, partial η² = 0.06; means were 303 and 275 ms, respectively. Younger children with DCD produced significantly larger MTdiff scores than older children with DCD, t(20) = 3.01, P = 0.01, partial η² = 0.38; means were 397 and 322 ms, respectively.

Errors

Mean AE and CTE scores (±SE) for each group are displayed in Fig. 4. The two-way ANOVA on mean TDE revealed significant main effects for condition, Wilk’s Λ = 0.36, F(2, 47) = 76.39, P = 0.000, partial η² = 0.65, and for group, F(2, 13811.12, P = 0.002, partial η² = 0.21. The interaction of group and condition was not significant, F(2, 47) = 0.53, P = 0.47, partial η² = 0.01. Independent t-tests revealed that the children with DCD committed significantly more CTEs, t(20) = 4.31, P = 0.000, partial η² = 0.31, while there was no difference between the groups on AE, t(20) = 1.83, P = 0.07, partial η² = 0.07.

Discussion

Using a double-step reaching paradigm, this study investigated the ability of children with DCD to make rapid online
adjustments. As predicted, the MTs of these children were significantly prolonged, relative to controls, when the location of targets was shifted at the point of lift-off. As well, the DCD group committed more movement errors. Children with DCD also showed a trend for slower movement initiation. The pattern of results suggests that children with DCD show impairment in the ability to update predictive (forward) models of movement online. This view is discussed in detail below.

RT

The absence of any effect for condition (i.e., non-jump vs jump trials) on RT was expected because the target perturbation did not occur until after movement lift-off (post RT). Hence, task requirements up to the point of lift-off were identical for both conditions; rather, by definition, online control would be exerted after lift-off and maximally in response to target perturbation.

There was a trend for children with DCD to be slower to initiate movement compared with controls (P = 0.10). This finding is in agreement with other work that shows consistently that children with DCD are generally slower to respond to external cues (Henderson et al. 1993; Wilson & McKenzie 1998). RT is one measure used to infer the speed of neural transmission which reflects the integrity of the central nervous system. Others have also made the point that in the case of movement, RT may reflect the time necessary to plan (and initiate) the impending motor command (Desmurget et al. 2004). In DCD, it is possible that the general effect on RT reflects both low transmission times and a defect in planning. However, our study was not designed to tease this apart, but rather to posit control issues that bear on MT data. This is now discussed.

MT

Like earlier studies using the DSP (Castilho et al. 1999; Farne et al. 2003; Plumb et al. 2008), MT increased significantly for jump trials. This is attributed to two key factors; firstly, the increase in movement amplitude required to complete the task; and secondly, the increase in task complexity related to online correction of movement. Assuming that the initial motor command is accurate, non-jump trials allow reaching to unfold largely unchanged. While some online modulation is expected for simple reaching, the motor command is imperfect (Desmurget & Grafion 2006), demands on this system are minimal. Conversely, in response to target perturbation, discrepancies between the predicted location of the hand (in relation to the target) and its actual location based on the flow of visual sensory information must first be detected and the resultant error signals integrated with the ongoing motor command to alter the movement path.

As predicted, the MTs of children with DCD were roughly equivalent to those of controls on trials where target location remained fixed. This finding accords with Wilmut and colleagues (2006) who showed the MT of children with DCD to be almost identical to controls for simple aiming movements. However, we showed that children with DCD were differentially affected by target jumps, relative to non-jump trials as indicated by the larger MT jump score compared with control children and the presence of a strong interaction effect. Inspection of Fig. 3 illustrates that the two groups performed similarly on the non-perturbed trials (mean difference = 22 ms), whereas the DCD group showed impaired performance when completing jump trials (mean difference = 90 ms); this pattern was confirmed by an analysis of simple effects. Further, examination of individual differences showed that over 70% of the DCD group were disadvantaged by the jump condition relative to controls. Taken together, these results suggest that although children with DCD were able to plan and perform simple aiming movements as effectively as their typically developing peers, they were less efficient at adapting their movements online to target perturbation. This pattern of results rules out the possibility that the impaired performance exhibited in DCD children may reflect a general motor deficit as such impairment would be expected to affect reaching performance irrespective of trial condition (cf. Plumb et al. 2008).

The presence of a significant interaction effect on MT stands in contrast to the results of Plumb and colleagues (2008) who also compared children with DCD and controls on a DSP. Importantly, in Plumb et al.’s study, the two groups performed somewhat different versions of this task control children
conducted the task while standing, whereas children with DCD were seated and used a finger stylus. In our study, both groups were seated and all other task parameters were identical (i.e., both groups were required to point and touch the target using their dominant index finger). Task modifications of the type used by Plumb and colleagues would result in a comparative increase in task complexity for controls by virtue of the added degrees of freedom that must be accommodated when planning a movement from a standing position, with all its postural constraints. Given that the magnitude of condition effects are in the order of milliseconds, any change in task complexity, no matter how small, would likely impact the pattern of results ( esp., interactions with group) whether the dependent measure is time or accuracy based. Thus, it is possible that the failure to observe an interaction in Plumb et al.’s study may be caused by differences in task complexity between the two groups which obscure an underlying issue with online motor control in DCD.

Again, rapid and efficient online correction in response to target jumps requires children to integrate error signals that are generated when the predicted location of the hand (in relation to the target) fails to match its actual location based on the flow of visual sensory information—a internal feedback loop that enables fast online corrections. The prolonged MT observed in DCD on jump trials may reflect impairment in the ability to either generate this error signal following target perturbation, or integrate it seamlessly with the ongoing motor command. This notion that children with DCD have an impaired ability to utilise internal models of movement has been canvassed previously (Wilson et al., 1997, 2001; Williams et al., 2006) and is discussed further below. While for simple (straight line) movements in perspective space, the motor deficit seen in DCD may not be as apparent, particularly when initial fixation and target location are aligned (as per Valli et al., 2006). For targets that are displaced finitely, however, trajectory planning might present more difficulty for these children.

**Analysis of age differences within each motor group**

There are little data on the development of ROC per se; however, goal-directed reaching undergoes significant maturation between the ages of 7 and 12 years. Over this period, the development of reaching follows a non-monotonic trajectory, characterized by an increase in proficiency with a temporary re-organization of control at around 8 years of age (Heyes, 1997; Pellizzi & Inherit, 1996). Indeed, at this age, performance efficiency has been noted to decline (e.g., Pellizzi & Inherit, 1996) and then follow an improving trend after about 9 years of age. Accordingly, we undertook additional analysis to investigate the development of ROC by comparing younger (i.e., 7–9 years of age) and older (10–12 years of age) children in the control and DCD groups, respectively, on MT.<br>

Interestingly, for children with DCD, ROC was less developed in younger children compared with older. This suggests that while impairment continues throughout development for children with DCD, they may ‘catch up’ to typically developing children, at least to a degree, throughout development. For typically developing children, there was no significant change with age on our measure of online control (cf. Pellizzi & Inherit, 1996). Thus, even younger control children possess a degree of competence with respect to ROC. The lack of difference between younger and older control children on our measure of online control is at odds with the general observation that movement efficiency and control improve from the ages of 7 to 12 years. However, as noted above, work on goal-directed reaching suggests an increase in proficiency at around 9 years of age as feedback and feedforward mechanisms become better integrated. Inclusion of 9-year-olds in the ‘younger group’ might therefore dilute the between-group comparison here as their performance compensates for some of the more immature response patterns seen in 7-year-olds, for example. Moreover, the slightly greater variability in the younger control group is consistent with the view that 9-year-olds are using a more sophisticated mode of control than 7- to 8-year-olds. These age comparisons are limited by small sample size, and longitudinal investigation is warranted to further explore these interesting developmental trends.

**Movement errors**

The number of TDEs committed by both groups increased significantly for the jump trials. Because task complexity increases during jump trials, this effect was expected. Interestingly, although the DCD group produced significantly more TDE than controls overall, no interaction effect was observed. The general insensitivity of the DCD group is in line with earlier research (Wilson et al., 1997). However, it was also reasonable to expect that they would be more disadvantaged than controls on jump trials as online control demands increased; but this was not the case. Taken together, a reduced ability to implement ROC (as shown in MT data) may bias children with DCD towards a speed-accuracy trade-off when dealing with target shifts; rather than making additional errors, they may compensate for impaired online control by slowing their movements to maintain some semblance of accuracy during jump trials. Interestingly, this type of pattern is also seen in older adults (Shamway-Cook & Woodacres, 2007, p. 487).
The DCD group also made more CTEs than controls, perhaps suggesting a problem of response inhibition. This type of error implies a failure to suspend or alter trajectory in response to the shift in target location, before a pre-planned reach has completely unfolded. In the main, the number of CTEs we see in healthy adult populations and typically developing (older) children is very small (in the order of <1% of trials). This makes the current result, with moderate effect sizes, noteworthy. There are two possible explanations for the current group difference. First, because successful correction of movement is dependent on the efficient integration of forward estimates of limb end point (relative to target) with the actual sensory estimates, the CTE data could be interpreted as a disruption of predictive control in DCD. However, the average number of CTEs was in order of two to three out of 16 jump trials—perhaps too few to draw strong inferences about online control. The alternative explanation is to attribute the higher number of CTEs to a reduced ability to inhibit or modulate responses to what was a highly salient stimulus (the centrally cued location).

Interestingly, difficulties inhibiting shifts of visual attention to compelling cues have been reported previously in DCD (Wilson et al. 1997; Wilson & Maruff 1999; Mandich et al. 2003; Wimmer et al. 2007). Most of this data are drawn from the covert orienting of visuospatial attention task (COVAT), a variant thereof. The work of Wilson and colleagues, in particular, showed that children with DCD manifest deficits in the ability to disengage voluntary attention from invalidly cued locations. Others have detected similar patterns of performance, but interpreted the results somewhat differently, implicating inhibitory control per se (e.g., Mandich and colleagues). The co-occurrence of inhibitory and movement skill problems has been well documented in both younger (Levesley et al. 2006) and older (Piek et al. 2007) children. And the overlap is even greater in children with comorbid DCD and ADHD (Serganit et al. 2006). It is likely that the ability to modulate action planning by inhibitory control might be reduced in both groups, placing limits on motor learning (see also Burkley 1997). Developmentally, we simply need more longitudinal data to unravel the dynamic relationship between inhibition and movement control in children.

Interestingly, children with DCD also tended to make more AIs (although this difference just failed to reach significance, $P = .07$). This type of error represents the ability to maintain sustained attention and to prevent responses before locations are cued, rather than the ability to actively disengage attention from invalid cues, as per the CONAT. However, we return to the possibility that inhibitory control may be compromised to some extent in DCD, at least to the extent that it concerns organization of a movement response to spatial targets. In other work, Piek and colleagues (2007) have found that children with DCD were poorer on a range of executive tasks that measured response inhibition, working memory and set shifting—they were slower and more variable. It remains to be seen whether deficits of this type also impact more specific aspects of executive control (e.g., error monitoring). Taken together, results for measures of error highlight the need for further investigation of the role of inhibition in DCD symptomatology, as well as the particular role, if any, it plays in impaired online control.

**Impaired internal modelling: implications and the role of the posterior parietal cortex (PPC)**

Converging measures have highlighted the role of the PPC in online control and forward modelling. It has been suggested that the PPC (and its downstream connections) may be the prime network for processing internal representations of action (for a good review, see Desmurget & Sirigu 2009), and is strongly implicated in the planning and execution of target-directed reaching (Andersen & Buneo 2000). More specifically, PPC is thought to subserve integration of forward estimates of limb end point with optic flow, and generators of the error signals by which the unfolding movement command can be modulated online (Desmurget et al. 1999).

Curiously, there are striking similarities between the pattern of online control displayed by children with DCD in the present study and adult patients with optic ataxia during double-step reaching. Optic ataxia is associated with damage to the PPC, resulting in impairment to rapid online adjustments when reaching (Pasdelix et al. 2006; Glover 2003); it is argued that this problem reflects difficulty integrating feedback-based information with the efference copy (namely, internal modelling) (see Glover 2003). Like the DCD children we tested, impaired online control in patients with PPC lesions is inferred from elevated MT in response to target jumps (Gre a et al. 2002); by comparison, responses are relatively preserved for simple reaching in patients with bilateral PPC lesions. As well, kinematic data have revealed that these patients have a propensity to reach towards the original goal and then generate a second movement towards the new target location, compared with the smooth corrections observed in healthy adults.

The notion that children with DCD show impairment in the ability to generate and/or monitor internal models of movement (implicating PPC) has been argued by a number of studies using motor imagery and oculo motor paradigms (Wilson et al. 1997, 2001; Williams et al. 2006). Interestingly, the performance
patterns observed in these studies mirror with remarkable consistency those seen in adults with lesions of the PPC. Notably, on measures of movement imagery, covert orienting and sequential eye movements, there are similarities between the performance of children with DCD and adults with ideomotor apraxia, a disorder associated with left parietal lobe damage (Butsuen et al. 2005). Whether this (neural) locus is a site of developmental deviation or delay in DCD is a question of continued focus in our group.

Limitations
As was highlighted in the introduction, the present study was designed to assess rapid online motor control in DCD as a probe to the mechanisms subserving impaired motor control in the disorder. Hence, we acknowledge the limitations of using chronometric data in isolation of kinematic data to examine mechanisms of motor control. Although global measures of performance like MT and errors can provide useful information about the integrity of control mechanisms, they provide little information about the subtle changes in control that are shown at different points in the movement cycle. That said, the large effect size observed for the interaction between group and condition on MT, together with the absence of any group effect on RT, does support the suggestion that the rapid online adjustments necessary to maintain speed and efficiency of movement are compromised in DCD. These results pave the way for future work that explores the kinematics of ROC in DCD.

Although there appears to be converging evidence that dysfunction at the level of PPC may explain a deficit of cognitive deficits observed in DCD, the implications of results presented here for neurocognitive models should be interpreted with caution. Inferences regarding the neural locus of internal modelling mechanisms are presently almost entirely based on adult data. With the exception of a recent EEG study conducted by Laut and colleagues (2006) and de Castella and colleagues (2008), very little neurophysiological or neuroimaging data exist on DCD. Because the cortical regions of most interest in the present investigation, the parietal and frontal cortices, follow a more protracted developmental trajectory than other brain regions – the current consensus being that the parietal lobe approaches maturity between the ages of 10 and 12 years, while the frontal lobe continues to mature well into the late teens (Casey et al. 2005; Leonard & Giedd 2006) – it is likely that these regions would be still unfolding in the children we tested. The upshot is that we need to temper any conclusions drawn about the integrity of these emerging neural networks in DCD, particularly when performance trends are interpreted with reference to mature adults on similar tasks. Neuro-imaging data are required to better understand how these networks change with age in both typical and atypical development.

Conclusion
The children with DCD displayed a reduced ability to adapt their movement online in response to target perturbation, manifest as delayed MT and increased response error. This pattern of performance was consistent with the view that the impaired online control found in DCD may, at least partly, be caused by an impaired ability to update predictive (forward) models of movement (Deurman et al. 2003). On a number of paradigms assessing aspects of internal modelling, children with DCD show a similar response pattern to patients with lesions of the PPC. This has led to the hypothesis that DCD may be attributable to dysfunction at the level of PPC. Notwithstanding some of the limitations of chronometric data, results presented here clarify some inconsistencies in the DCD literature, and lay the foundation for more rigorous kinematic analysis and functional imaging in future research.

Key messages
- DCD affects movement in between 5 and 19% of children.
- There are a number of plausible, yet diverse aetiological accounts of DCD; online motor control is particularly important to the development of skill.
- DSPs developed and interpreted from a modern neurocomputational perspective are a valid method for investigating online motor control.
- Our findings showed that the children with DCD were able to perform simple goal-directed reaching, as well as their age matched peers, but experienced greater difficulties adopting their movement online.
- From a neurocomputational perspective, impaired online control suggests some difficulty with internal modelling, likely at the level of PPC.

Acknowledgements
We would like to acknowledge Ray Druckman for his technical support throughout the development of the paradigm, and Assoc Prof Paul Maruff from CogState Ltd, Melbourne, Australia, for his assistance throughout the study.

© 2018 Blackwell Publishing Ltd. Frontiers in child health and development.
Appendix A

Published Articles

References


Appendix A

Published Articles


Dissecting online control in Developmental Coordination Disorder: A kinematic analysis of double-step reaching

Christian Hyde, Peter H. Wilson

School of Psychology, School of Health Sciences, RMIT University, Melbourne, Australia

Abstract

In a recent study, children with movement clumsiness (Developmental Coordination Disorder, DCD) were shown to have difficulties making rapid online corrections when reaching, demonstrated by slower and less accurate movements to double-step targets (Hyde & Wilson, 2011). These results suggest that children with DCD have difficulty using predictive estimates of limb position when making rapid adjustments to movement, in-flight. However, kinematic data alone does not provide strong evidence for this hypothesis. It remains unclear whether early (and rapid) control parameters or post-correction stages of the movement trajectory are affected. This, the overarching aim of this study was to conduct a kinematic analysis of double-step reaching in order to isolate the different control parameters that might explain the slower and less accurate double-step reaching performance of children with DCD. Participants were a new sample of 15 children with DCD aged between 8-12 years and 13 age-matched controls. Children were required to reach and touch one of three possible targets presented at the coordinates 28°, 0°, and 20° on a 17-in. LCD touch screen. For most trials (95%) the target remained stationary for the duration of movement (non-jump trials), while for the remainder (5%) the target jumped randomly to one of two peripheral locations at movement onset (jump trials). Consistent with earlier work, children with DCD were shown to initiate reaching compared to controls and showed longer MT and more errors on jump trials. Kinematic data showed that while the two groups did not differ on time to peak velocity or acceleration, children with DCD were slower to correct reach trajectory on jump trials. No group differences were observed on less kinematic measures, e.g., post-correction time. The pattern of results support and extend earlier work showing deficits in ROC in DCD. From a computational perspective, delayed corrections to the reach trajectory suggests some difficulty integrating information about the target perturbation with a predictive (or forward) estimate of limb position relative to the initial target. These conclusions are discussed, along with directions for future research.

© 2011 Elsevier Inc. All rights reserved.

1. Introduction

The development of online motor control is thought to be crucial to the smooth and flexible control of action. In typically developing children, the nature of online control alters with the changing constraints of maturation and experience (Hyde & Wilson, 2011). Important among these changes is the capacity of the nervous system to make rapid changes in trajectory, in-flight, should movement be perturbed in some way or should something in the environment change. This form of rapid online control (ROC) is thought to be visible to the extent that the nervous system can predict the future location of the moving limb using a forward internal model (Desmurget & Grafton, 2003; Jeannerod, 2006; Woolpert, 1997). This forward estimate of limb position provides a means of rapidly integrating effector and afferent signals — sometimes referred to as an internal feedback loop — thereby speeding responses to any changes in the environment during the course of movement (Desmurget & Grafton, 2003). This type of control is illustrated clearly in cases where the goal of a task changes as the movement is being performed, e.g., reaching for a pen as it rolls away. Experimentally, this scenario can be simulated using a double-step perturbation procedure whereby the movement target shifts to another location at movement onset. From a computational perspective, the initial state of the limb is defined by visual and proprioceptive coordinates and the target by visual coordinates. As the movement is generated, a corollary discharge encodes a copy of the movement commands (via efference copy) which is used to
predict how the limb will move in response to the motor command; the predicted consequences are specified in an (internal) forward model (Shadmehr, Smith, & Krakauer, 2016). In neural terms, a functional loop between parietal cortex and the cerebellum is thought to monitor these forward estimates of limb position and correct any diverging motor commands online should the action deviate from expectations (Blumenfeld & Sirigu, 2009; Shadmehr & Krakauer, 2006).

In the case of visual perturbations, a forward model of limb position relative to the target is generated and compared with sensory afference which signals actual target location. Any mismatch is thought to generate an error signal that is used to update limb trajectory. More precisely, computational modeling suggests that rapid online corrections are organized by “superimposing” a dynamic error signal onto the outgoing feedforward motor command (Cavina-Parola, Yavuz, & Kalaska, 2009). These online adjustments are tuned to the dynamic kinematic properties of the moving limb and circumvent the processing delays associated with sensorimotor feedback loops (Hanazag, Vetter, Johansson, & Wolpert, 2001). Indeed, this form of predictive control is ideally important because the position of the moving limb has changed appreciably by the time sensory feedback alone can be used to alter motor commands. Importantly, a number of studies show that smooth online corrections are disrupted when the involvement of posterior parietal cortex (PPC) is disturbed by lesion (Cela et al., 2002) or through TMS (Desmurget et al., 1998). Parietal regions are thought to be crucial in updating forward estimates of limb position, particularly where vision of the moving hand itself is not available. In a similar vein, rapid online adjustments are also necessary during the early stages of movement or when the moving limb itself undergoes some external perturbation which forces it to maneuver, off course. Ascending cerebellar pathways are thought to monitor somatic perturbations of this type, detecting with minimal time lag the discrepancy between the predicted dynamic properties of the limb in response to the motor command and its actual behavior. Indeed, the cerebellum has been referred to as a “somatic event detector” to highlight its vital role in motor control (Weisz & King, 2008).

In general, healthy young adults have little difficulty adjusting their movement in-flight in response to visual and mechanical perturbation, suggesting seamless use of predictive control. Indeed, the ability to correct errors is regarded as one of the most significant achievements in motor control during childhood and over the course of adolescence where biomechanical constraints are changing rapidly as a result of maturation (Chowdury, Champion, Hird, & Blakemore, 2006). The significance of prediction in development is highlighted by the fact that children with movement difficulties (or Developmental Coordination Disorder—DCD) show poor coupling of hand and eye movements during target-directed reaching (Wittem, Watson, & Brown, 2008) and improved online adjustments (to visual perturbation) (Hyde & Wolpert, 2011). Briefly, DCD is characterized by a deficit in fundamental motor skill in the absence of neurological or physical impairment, a feature which distinguishes it from common development disorders of movement such as Cerebral Palsy (Pearson-Jones, Pink, & Levy, 2014). Until recently, the evidence on the nature of ROC in DCD was mixed.

Work by Wittem et al. (2006) demonstrated that children with DCD were as efficient when reaching to a single target but were slower and less accurate when reaching sequentially from one target to another. The DCD group spent more time focusing targets generated sequentially before initiating hand movements, which led to an increase in error. This pattern suggested difficulties in feedforward control, but did not examine ROC directly in response to target perturbations. More recently, Funky and colleagues (2008) suggested that ROC in reaching was preserved in DCD. They found that the effect of target perturbation on movement time was similar for both DCD and non-DCD groups. However, Plunk also acknowledged that there were methodological limitations in this study; group comparisons were compromised by the fact that DCD and control groups performed different versions of the same task (i.e., children with DCD were seated and used a large pointing stylus, while control children stood and used a smaller stylus).

To address these conflicting accounts of online control in DCD, we recently examined double-step reaching while controlling all task parameters (Rhyde & Wolpert, 2011). A chronometric analysis showed that children with DCD were more disadvantaged by target jumps, manifest as slower and more error-laden performance on jump trials compared with typically developing children. We explained this pattern of performance from a neuro-computational perspective. Here ROC is thought to be implemented by integrating predictive (or feedforward) and feedback based mechanisms efficiently. This argument accounts for recent evidence showing that feedback based mechanisms are used continuously throughout the movement cycle (Sauter & Knill, 2001, 2003) rather than simply towards the end of movement vis-a-vis the old dual-component model of reaching (for a review see Elliott, Helsen, & Chau, 2001). We argued that slower and less accurate double-step reaching in DCD may reflect a difficulty using predictive (or forward) models to rapidly update movement plans; this has been expressed previously under the internal modeling deficit (IMD) hypothesis (Williams, Thomas, Maruff, Butson, & Wilson, 2006; Williams, Thomas, Maruff, & Wilson, 2008; Wilson et al., 2004). A major limitation of chronometric analysis alone is that it does not allow one to dissect the subtle transitions in motor control that occur at different time points in the movement cycle (i.e., the question of whether control parameters are affected early or late in the movement trajectory. For jump trials, due to time delays associated with processing non-visual and visual sensory feedback, reaching is thought to rely heavily on predictive control during the early phase of movement, ap to the point when early kinematic markers are expressed (i.e. time to peak acceleration and velocity; dFA and dPV). These early markers together with the first detectable change in movement trajectory are thought to reflect the integration of real-time sensory feedback with the ongoing motor command. More precisely, in circumventing processing delays, sensory signals are thought to be conveyed as an extension of the initial position (relative to the initial target) allowing discrepancies to be detected rapidly in real-time, and corrective signals generated to adjust the movement trajectory. Though online corrections preserved over the entire movement cycle, demands on this system are maximal during the early phase of double-step reaching when the larger scale changes in trajectory are implemented in response to target perturbation, and reduced during the later (post-correction) phase of reaching which serves mainly to break the limb as it captures the target at its new, fixed location (see Wolpert & Flanagan, 2001).}

Other work using the double-step task has revealed distinct patterns of deficit based on early kinematic markers. In patients with optic ataxia, for instance, corrections to the reach trajectory after target perturbation occur significantly later than in healthy adults suggesting difficulties using internal feedback control to update the motor command (Cela et al., 2002). Desmurget and Grafton argue that the posterior parietal cortex and its reciprocal connections to the cerebellum may support these early corrections: for visually-guided reaching, predictive models for limb position are thought to be generated and/or monitored at the level of PPC (see Desmurget & Sirigu, 2005). These forward estimates enable the system to respond rapidly to self-in-target relations change during the course of a movement, as when targets shift their location. The PPC is one site where comparisons between the expected location of the limb (with respect to the target) and that indicated
by actual sensory inflow occurs, and the error signal generated (Deumert et al., 1999). The cerebellum is another site involved in monitoring forward models, particularly for detecting and correcting unexpected somatic events (Blakemore & Sirigu, 2003).

From a neuro-computational perspective (e.g., Deumert & Graf ton, 2005; Deumert & Sirigu, 2006; Wolfpert, Ghahramani, & Hafiz, 2001) rapid online correction during reaching is thought to be dependent on two processes: the first concerns the capacity of the motor system to monitor the presence of any error between the predicted limit trajectory (according to a forward model) and actual limb position based on sensory inflow; and second, the integration of a resultant error signal with the ongoing motor command to alter trajectories in-flight and with minimal time lag. Importantly, these control processes can be inferred from kinematic landmarks in reaching. Adult data for double-step reaching show that early kinematic markers (i.e. DFA and IV) occur earlier for jump compared with non-jump trials (Castello, Paulignan, & Jeannerod, 1981; Farrel et al., 2007; Paulignan, MacKenzie, Martinet, & Jeannerod, 1991) suggesting very rapid online adjustment (i.e. at around 100 ms, too fast for sensory feedback control alone which can take upwards of 200 ms (Flinn, Blakemore, & Wolfpert, 2000)). Earlier budding is then correlated with the subsequent change in trajectory (i.e. Farrel et al., 2007) that occurs some 150–200 ms after these initial landmarks. Thus, DFA and IV may mark a period during which discrepancy between the expected and actual sensory consequences of movement is initially detected (i.e. Farrel et al., 2007; Paulignan et al., 1992). Fast visual channels associated with so-called visual-motor functions (between dorsal parietal and motor cortex) are likely to support this (see Pizzol, Blumfeld, Lueck, Yarn, & Rossetti, 2006). This information must then be used to re-calibrate the movement and adjust the ongoing motor command; the earliest detectable change in trajectory toward the new target is thought to represent this process (Dobrovoliski, Bickford, Lueck, Carnahan, & Jingling, 2002; van IJzendoort, Catcher, Geuze, Steenbergen, & Bouma, 2007). Dissociation between the error detection and trajectory correction processes is further highlighted by evidence from optic ataxic patients who experience difficulty correcting ongoing movement following target perturbation (Cota et al., 2002) despite a preserved capacity to detect error (Pizzol et al., 2006).

The broad aim of the current study was to investigate ROC in children with DCD using both kinematic and chronometric analysis of double-step reaching. More specifically, we hoped to break down the kinematic analysis into earlier and later phases of processing in order to isolate the different control parameters that might explain the slower and less accurate performance in DCD. Given the fundamental imperfections we have observed in DCD on other aspects of predictive control (e.g., double-step sarcodes, reported in Kuchma et al., 2002), we predicted that performance deficits would manifest on early markers of double-step reaching.

2. Method

2.1. Participants

The sample consisted of 13 children (nine girls and four boys) between the ages of 8 and 12 years of age who met the research criteria for DCD and 11 age-matched controls (six girls and seven boys). This sample was different in that used in Hyde and Wilton (2011). Mean age for the DCD group was 10.5 years (SD = 1.7) and 10.3 years (SD = 1.4) for the controls. All children were right-handed dominant with the exception of four children in the DCD group, as determined by performance on the McGarron Assessment of Neuromatocellular Development (MANN; McGarron, 1997).

Children were screened using the same procedure adopted in our earlier investigation and approved by the RMFT HREC and the Victorian Government Department of Education and Early Childhood Development. Principals from two state primary schools in suburban Melbourne were approached and invited to participate in the study. Classroom teachers from these schools identified children whom they considered to show poor movement skill for their age (i.e. they demonstrated significant difficulty completing everyday tasks such as handwriting, using classroom utensils [i.e. scissors, pencils, etc.] and/or physical education activities; as per DSM-IV diagnostic criteria, Criterion C), this was corroborated by physical education teachers. These children were then assessed using the MANN: those scoring below the 10th percentile were included in the DCD group (see Geuze, Jongmans, Schoemaker, & Smits-Engelsman, 2001) (Criterion C). Parents were not asked to indicate whether their child had been referred for or were receiving movement therapy. Exclusion criteria were a past or current diagnosis of ADHD or known learning, neurological or physical disorder (Criterion C). As the children were recruited from mainstream primary schools and had not been diagnosed with a learning disorder, they were assumed to have IQ levels within the normal range (Geuze et al., 2001) (Criterion D). Control children were considered to have age-appropriate levels of motor skill by teachers and a MAAD total score above the 20th percentile.

2.2. Apparatus

2.2.1. Double-step reaching paradigm

The double-step reaching paradigm was used to assess online motor adaptation. The Virtuos Software Package (Dassault Systèmes, Villeurbanne, France) was used to develop the task display which was presented on a 17 in. LCD touch screen (TFT, Neste, Tashcan, Korea). The monitor was mounted at an angle of 15° from horizontal on a tabletop, which ensured that targets were freely visible throughout the movement. The (black) surface surrounding the monitor was matched to the color of the monitor beam and visual display to reduce the contrast within the participant’s visual field. The display consisted of a green circle at the bottom center of the monitor which acted as a ‘home base’ and three (yellow) possible target locations (each 2 cm in diameter) presented in a semi-circular formation across the top of the screen. Three horizontally spaced were spaced 20° apart at the coordinates of 20°, 0°, and 20° with respect to the ‘home base’. The distance between the center of the home base and each target was 30 cm. Participants wore a thin polyester glove on their reaching (i.e. dominant) hand. An electromagnetic sensor was attached to the position of the fingernail of the index finger of the glove using Velcro; the underside of the gloves index finger was removed to maximize tactile feedback. Participants were seated unstrained on a height adjustable chair in a dimly lit room. Lighting levels did not permit children to receive visual feedback of their moving hand (Farrel et al., 2003). The index finger of their dominant hand was placed on the ‘home base’ which was positioned approximately 5 cm anterior to the umbilicus at the midline. Their non-dominant hand was extended comfortably beside the monitor. An assessor stood behind the participant throughout the task to ensure that they remained attentive and to repeat instruction when necessary. Kinematic data for reaching was recorded using the HMD of Birds motion tracking system (Ascension, VT, USA) sampled at 100 Hz. Raw data was converted into three dimensional coordinates (X, Y, Z) with the Y-axis representing the distance component of movement, X-axis the direction component, and Z-axis the depth component. Acceleration, velocity and reaching trajectory profiles for each trial were recorded from this data. See Fig. 1 for typical non-jump and jump reaching plots for children with DCD and age-matched controls.
Appendix A

Published Articles

2.3. Procedure

At the beginning of each trial, the ‘home base’ was illuminated; children were instructed to touch and hold the ‘home base’ with their (dominant) index finger. After a random interval of between 500 and 1500 ms (to minimize anticipatory responses) the home base was extinguished, which coincided with illumination of the central target. Children were required to reach and touch the middle of the target as quickly and accurately as possible, until it disappeared. To ensure that visual attention was initially oriented to the same location for each trial, participants were instructed that the central target location would always be at the bottom of the screen, but that the target may jump to one of two other (peripheral) targets during movement (Castello, Bennett, Bondfjeld, Lim, & Peppard, 1999; Castello, Bennett, & Chambers, 1998). During these jump trials, children were asked to follow and touch the middle of the target.

After each successful trial, children were instructed to return to the ‘home base’ and await the next target. For 80% of total trials (80), the central target remained illuminated for the duration of the movement. For the remaining trials (20%), the central target was initially illuminated, but at the point of finger lift-off from the ‘home base’, the central target location was extinguished but re-appeared immediately at one of the two peripheral locations (jump trials). Participants were instructed to reach and touch the target that was currently lit until the auditory tone was emitted and the target extinguished.

Prior to testing, the assessor modeled task performance. Participants then completed eight practice trials (six non-jump and two jump). If the experimenter deemed it necessary, additional practice trials were administered until it was clear that the participants understood the instructions. Participants then completed two blocks of 40 trials that were separated by a 1 min break. Each block consisted of 12 non-jump trials and eight jump trials (four to each side) which were presented in a pseudo-randomized order.

Timed measures were: reaction time (RT), measured by the time between target display onset and finger lift-off from the ‘home base’; movement time (MT), measured by the interval between lift-off and placement of the finger on the target location. The difference between the mean RT for jump and non-jump trials (MTRed) was also calculated. Three types of response error were also recorded: first, touchdown errors (TE1) were recorded when an initial contact was outside the target boundary; second, anticipatory errors (AE) were recorded for responses with initial contact (i.e., lift-off from the ‘home base’) before a target location was illuminated or the RTs within 150 ms (see Wilson, Manzol, & McKeon, 1997). Finally, correct touch errors (CTE) were recorded for touches made initially to the central target on jump trials.

Four kinematic measures were recorded: time to peak acceleration (TPA); time to peak velocity (TPV); time to correction of movement trajectory (TC), and post-correction time (PCT). TC was measured as the time of movement correction away from the initial (central) target towards the correct target during ‘jump’ trials. This was determined manually using a 2D (x by y) representation of each reaching trajectory for jump trials to identify the time at which the hand deviated away from its (virtually) straight-line path and toward the caudal peripheral target (Hsiao et al., 2000; van Bruaendt et al., 2007). Negligible punctuation and inpation of the lower arm occur during the course of the movement; hence, there was no need to adjust these measures. PCT was determined for jump trials as the time elapsed from TC to the completion of movement.

2.4. Design and analysis

Kinematic data were filtered off-line using a fourth order Butterworth filter with a cut-off of 10 Hz. For jump trials, timed responses were collapsed over target location (i.e., left and right). For each child, mean values were recorded for each dependent measure; trials on which errors occurred were counted but not included in the analysis of chronometric and kinematic data. Preliminary analysis revealed that condition had no effect on total numbers of AE committed and hence they were collapsed over ‘jump’ and ‘non-jump’ trials. For inclusion in parametric analysis, all children met a minimum of 50% correct responses on jump trials (or 8 out of 10). For each dependent measure, outliers were defined as responses ±3 SD from the child’s mean for that condition. An average of 24 (90%) and 17 (74%) trials were removed from the DCD and control groups, respectively.

Separate two-way ANOVA (2 [Group] × 2 [Condition]) were conducted on mean values for RT, MT, TPV, TPE and TCE. Independent t-tests were used to compare groups on AE, CTE, MTRed, TC, and PCT. Measures of effect size (partial $\eta^2$) were used to temper the results of significance tests.

3. Results

3.1. Chronometric analysis

3.1.1. Reaction time

Mean RTs (ms) for each group are displayed in Fig. 2. The two-way ANOVA on mean RT revealed no significant interaction effect between group and condition, $F(1, 24) = 0.19$, $p = .66$, partial $\eta^2 = 0.03$. Averaged over condition, the mean RT for the DCD group (572 ms) was significantly longer than the control group (494 ms), $F(1, 24) = 8.87$, $p = .004$, partial $\eta^2 = 0.28$. The main effect for
Appendix A

Published Articles

condition was not significant, \( F(1, 24) = 0.03, p = .93, \) partial \( \eta^2 = 0.000; \) averaged across group, mean RT for ‘jump’ and ‘non-jump’ trials was 533 and 532 ms, respectively.

3.1.2. Movement time

Mean MT’s (ms) for each group are shown in Fig. 3. The two-way ANOVA on mean MT revealed a significant interaction between group and condition, \( \text{VARS} A = 0.06, F(1, 24) = 0.53, p = .47, \) partial \( \eta^2 = 0.03. \) Also significant was the main effect for condition, \( \text{VARS} A = 0.34, F(1, 24) = 0.13, p = .72, \) partial \( \eta^2 = 0.06, \) while that for group was not, \( F = 1.23, p = .24, \) partial \( \eta^2 = 0.05. \)

Tests of simple main effects failed to show a significant group difference on non-jump trials, \( F(24) = .42, p = .51, \) partial \( \eta^2 = 0.01, \) but children with DCD (855 ms) were significantly slower on ‘jump’ trials than control children (815 ms), \( F(24) = 2.10, p = .05, \) partial \( \eta^2 = 0.08. \) The mean MT difference score for the DCD group (338 ms) was significantly higher than controls (260 ms), \( F(24) = 3.24, p = .003, \) partial \( \eta^2 = 0.0). \)

3.1.3. Analysis of individual differences

Individual differences on MT difference scores for each group are shown in Fig. 4. There appeared to be a distinction between groups at approximately 300 ms: most children with DCD exceeded this time while most controls did not. The Qmax for the DCD group was between 302 and 373 ms compared with 225 and 295 ms for controls. Nine of the 13 children with DCD (or 69%) exceeded the upper Qmax of the control group (i.e., 205 ms) while three of the 13 control children (or 23%) exceeded the lower Qmax of the DCD group (i.e., 932 ms).

3.2. Kinematic analysis

3.2.1. Time to peak acceleration

The two-way ANOVA on mean iPA revealed no significant interactive effect between group and condition, \( F(1, 24) = 0.34, p = .71, \) partial \( \eta^2 = 0.01. \) Averaged over condition, the mean iPA for DCD children and controls (176 and 160 ms, respectively) did not differ statistically, \( F = 1.50, p = .23, \) partial \( \eta^2 = 0.06. \) The main effect for condition was also not significant, \( F = 0.75, p = .34, \) partial \( \eta^2 = 0.03, \) averaged over group, mean iPA for ‘jump’ and ‘non-jump’ conditions was 171 and 166 ms, respectively.

3.2.2. Time to peak velocity

The two-way ANOVA on mean iPV revealed no significant interaction between group and condition, \( F = 1.98, p = .26, \) partial \( \eta^2 = 0.01. \) Averaged over condition, the mean iPV for DCD children and controls (159 and 179 ms, respectively) did not differ statistically, \( F = 0.80, p = .38, \) partial \( \eta^2 = 0.01. \)

3.2.3. Time to correction (TC)

Mean IC (ms) for each group is displayed in Fig. 5. Independent t-test revealed that children with DCD took significantly longer to initiate movement correction on ‘jump’ trials than control children, \( t(24) = 3.46, p < .01, \) partial \( \eta^2 = 0.12. \)

3.2.4. Post-correction time (PCT)

Independent t-test showed no significant difference in time spent in the declarative phase of ‘jump’ trials for children in the DCD and control groups, respectively, \( t(25) = 1.60, p = .05, \) partial \( \eta^2 = 0.08. \)

3.3. Errors

Averaged across condition, children with DCD (mean = 1.8) committed significantly more TDs than controls (10.2), \( F(1, 24) = 4.76, p = .04, \) partial \( \eta^2 = 0.17. \) The two-way ANOVA on TDs failed to reveal a significant effect for condition, \( F(1, 24) = 1.24, p = .28, \) partial \( \eta^2 = 0.05, \) nor was the condition by group interaction significant, \( F(1, 24) = 0.13, p = .73, \) partial \( \eta^2 = 0.01. \) Finally, there were no significant group differences on AE, \( F(24) = 0.19, p = .68, \) partial \( \eta^2 = 0.002 \) (M = 1.10 for DCD, and 1.04 for control), or CT, \( F(24) = 1.30, p = .07, \) partial \( \eta^2 = 0.013 \) (M = 1.77 for DCD, and 0.61 for control).

Fig. 3. Mean movement time (MT & SD) for DCD and control groups.

Fig. 4. Mean MT difference scores for DCD and control children.

Fig. 5. Mean time to correction of movement initiation for DCD and control children.
Appendix A

Published Articles

4. Discussion

In an earlier chronometric study of double-step reaching, children with DCD showed a reduced ability to make rapid online corrections, demonstrated by slower and less accurate reaching on jump trials (Hyde & Wilson, 2011). However, it was not possible to determine whether the motor control issue was expressed early or late in the reach trajectory. The kinematic study presented here was designed to isolate early markers of online control that indicate use of forward estimates of limb position during double-step reaching.

As predicted, chronometric data showed that children with DCD were generally slower to initiate movements compared with controls, an attribute of movement that has been reported repeatedly (e.g., Henderson, Rose, & Henderson, 1982; Wilson & McKenzie, 1998). Importantly, their reaching movements were significantly slower and less accurate on trials when the target unexpectedly jumped at reach onset (see also Hyde & Wilson, 2011). Our kinematic data clarify the underlying nature of this impairment. On jump trials, reaching is thought to rely heavily on predictive modeling during the early phase of movement, which is expressed as reduced FA and IPV compared with non-jump trials. Interestingly, no group differences were observed on IPA or IPV. However, children with DCD were significantly slower to correct the trajectory of their reach away from the initial target location on jump trials (given by TC). The TC metric indicates the point at which fast internal feedback signals are integrated with the ongoing motor command (i.e., Dufresne et al., 2002; van Baardewijk et al., 2007). This process of rapid modification in limb trajectory occurs within the order of 250–350 ms in adults and can be considered a crucial outcome of motor prediction (Dufresne & Craigh, 2006). By comparison, no difference between groups was observed on PCT where demands for online control are reduced because of the elapsed time since target displacement. Taken together, this pattern of results suggests impairment in DCD of the ability to implement rapid online control in response to target perturbation. This hypothesis is discussed in detail below.

4.1. Chronometric analysis

4.1.1. Reaction time

No effect of condition (i.e., non-jump versus jump) on RT was observed. This result was expected since target perturbation occurred after movement initiation: RT was recorded prior to this point. No differences were observed between the two groups for either task. No differences in terms of reaction time were observed for either group. In general, children with DCD had a slower RT, but this was not statistically significant.

4.2. Movement planning

4.2.1. Early control parameters

Unlike performance from healthy adults, there was no significant difference between jump and no-jump reaching for either group on FA or IPV, and no overall group difference (see also Plum & Colledge, 2008). In adults, FA and IPV on jump trials (at around 100 ms) are lower than on non-jump trials (Castiello et al., 1991; Farnè et al., 2003; Fadda et al., 1991) and precedes the actual redirection of the limb by about 150–200 ms. Reduced FA, for example, implies some motor consequence of the target displacement—more specifically, the process of error detection per se (Farnè et al., 2003; Fadda et al., 1991). However, additional time is needed to integrate any error signal with the ongoing motor command which ultimately results in a change in limb trajectory, which is inferred from TC (Dufresne et al., 2002; van Baardewijk et al., 2007).

The pattern of performance observed in our study suggests that the powerful internal feedback loop which supports early error detection in adults may still be unfolding in primary-school-aged children. This hypothesis is supported by the only other developmental study of online control using a double-step reaching paradigm which also failed to show a condition effect on IA.
Appendix A

Published Articles

or IBM in 7–10 year olds (van Breukelen et al., 2007). However, on jump trials in this earlier study, the target disappeared 120 ms after movement onset and reappeared in the new location 120 ms later. Hence, the task used by van Breukelen may not fully capture the very early and rapid mechanisms we are tapping into in our study.

Slower TC in children with DCD may suggest a reduced ability to integrate internal feedback signals with the ongoing motor command (Van Breukelen et al., 2007). Current consensus suggests that the capacity to use this control system in an adult-like fashion emerges between the ages of 8 and 12 years in typically developing children (Hay, 1979; Hay & Redon, 1995; Pellizer & Hanter, 1996). Van Breukelen showed that 62% of typically developing children aged between 7 and 10 years met predefined accuracy demands by completing double-step reaching in a single movement (i.e., without reaching the intended target). Temporal values for TC in this study cannot be compared directly to ours or to the adult data due to variations in the task. Though no work has specifically compared the double-step reaching of healthy primary school-aged children with adults, the mean TC score of control children in our study (M = 328 ms) is at the lower end of the distribution of values for healthy adults. For target jumps that are coincident with finger/hand lift-off, some of the range of values reported for adults include the following: 286–339 ms (Babinski et al., 2002); 255–285 ms (Paulignan et al., 1991); 238–264 ms (Roquin & Harvey, 2008); and 309 ms (Sadeghia, 2006). It should be noted that participants in the Sadeghia study were instructed to reach at a ‘comfortable’ speed (rather than as quickly and accurately as possible), which may account for slower TC. Thus, while a degree of proficiency in the rapid online corrections of our control group, the system appears to undergo further refinement over adolescence and early adulthood. Whether the performance pattern of children with DCD reflects a deviation from the typical developmental trajectory or an immaturity of sorts is unclear from our data. This issue is the subject of current investigation in our laboratory.

Recent neurophysiological data from studies of human and non-human primates has clarified the specific neural networks that support predictive control, particularly those involved in sacral planning and for rapid adjustments of limb trajectory (Bikson & Sagar, 2003). The role of PPC in predictive control (particularly in the dorsolateral region) has been well documented for goal-directed movements to visually-defined targets. It has been shown, for example, that receptive fields in frontal eye field and PPC are updated in anticipation of a sacral (Kratzner, Biggin, & Milner, 1994). This forward estimate of sacral direction appears to provide a spatial (or egocentric) frame for planning limb movements (Arff, Donchin, Napolitana, & Shadmehr, 2002). For goal-directed reaching, the parietal cortex contributes to state estimation by integrating dynamic visual inputs that signal changes in the environment with forward estimates of the state of the limb and visual environment (Archambault, Caminiti, & Satzinger-Mayer, 2009; Shadmehr & Krakauer, 2003). In other words, when a visual target jumps, the unexpected sensory information must be integrated with the output of the forward model, otherwise the reach will continue along its original (but now redundant) path. The upshot is that output signals from PPC provide crucial error information that is then used by motor cortex to alter motor commands to the moving limbs. Our data suggest that the process of integration may be impaired in DCD.

Interestingly, the pattern of performance we see in DCD is very similar to that seen in optic ataxia, a disorder of parietal lobe function that is known to impact ROC (Gira & et al., 2002; Pisella et al., 2006). Gira showed that TPA and TPV during double-step reaching was similar to healthy controls yet patients were slow to correct the trajectory of their reaching following target perturbation suggesting difficulties integrating error information onto a corrective online response. This account of impaired ROC in optic ataxia is supported by evidence showing that patients are able to terminate movement in response to unexpected target perturbation as quickly as healthy controls (Pisella et al., 2000) – suggesting preserved error detection – yet cannot amend movement trajectories to a displaced target (Gira et al., 2002). This work supports a growing body of evidence that demonstrates similar performance profiles between children with DCD and patients with PPC damage on measures of predictive modeling (i.e., motor imagery ability, assessed using mental limb rotation) (Williams et al., 2006, 2008), the visually guided pointing task (Wilson, Macarff, Ives, & Carron, 2001) and double-step sacral task (ESST) (Katschinskas et al., 2001). For a more detailed discussion see Hyde and Wilson (2011).

While there is some converging evidence that “delay” in maturation of the PPC may explain the impairments in predictive control that we see in DCD, other work suggests that the cerebellum may also be implicated. For example, Hill and Wing (1998) infer deficits in predictive control based on evidence of impaired grip-force modulation in DCD. When lifting an object vertically, a child with DCD was shown to increase grip force earlier than controls. But also, in response to load perturbation, grip-force changes occurred later in DCD. Similar results have been observed by Pereira, Haissou and Fossberg (2000) and Pereira, Landgren, Gillberg and Forsgren (2001), with pronounced deficits in grip-force modulation seen in a sub-group of children with DCD. The pattern of deficit here is similar to that seen in cerebellar patients (see Wolpert, Miall, & Kawato, 1998). Finally, and more recently, Kapner and Collewijn (2006) have shown impaired visual-motor adaptation in DCD using prism displacement. Using a center-out pointing task involving a 60° rotation of visual feedback, typically developing showed strong adaptation effects after prolonged training regardless of whether an abrupt or gradual change in visual feedback was used. Children with DCD, however, showed no after-effects when exposed to gradual changes in feedback. This suggests a reduced ability to detect and correct movement errors over repeated trials—i.e., other words, a problem updating an internal model for the pointing movement. Again, the pattern of performance resembles that seen in cerebellar dysfunction (Miall & King, 2008).

4.2.2. Late control parameters: post-collision phase

The lack of group difference on PCT indicates that temporal control during the latter stages of reaching is relatively preserved in DCD. This is based on the assumption that once an initial adjustment to the movement trajectory is made in response to the target jump, demands on ROC are minimal for the remainder of the movement (see Wolpert & Flanagan, 2001). This argument is supported by observation that the accuracy of double-step reaching by adults is not affected by target perturbation (Castiello et al., 1991; Paulignan et al., 1991; Sadeghia, 2006); similarly, there was no effect of condition on TDE in our study. Further, visual inspection of reaching trajectory plots for each of our groups showed that once the initial corrections to trajectory were implemented in response to the target jump, movement followed a (virtually) straight-line path, like that seen on non-DCD trials (where ROc are also minimal). Reach trajectories of a similar shape on jump trials have been observed for healthy younger (Castiello et al., 1991; Farnb et al., 2002; Sadeghia, 2006) and older adults (Sadeghia, 2006; Sadeghia, Gautier, Boudon, Veccer, & Hotan, 2006), and right brain-damaged patients (Farnb et al., 2003). Taken together, data suggest that although children with DCD take longer to implement changes in trajectory (in response to perturbation), they are able to then capture targets with reasonable accuracy and efficiency. Both the post-collision phase of jump trials and simple reaching under no-jump conditions are characterized by relatively low demands on ROC. On balance there is support for the notion that simple
Appendix A

Published Articles

reaching is age-appropriate in DCD under stable environmental conditions.

4.2.3. Summary of kinematic findings

Taken together, data is consistent with the broad hypothesis that ROC – which relies on predictive estimates of limb position – is impaired in children with DCD. Specifically, slower correction of reaching trajectory from the initial target on jump trials may reflect difficulty integrating information about the target perturbation with the ongoing motor command. By comparison, control of the larger scale of movement (where deviation from rapid online adjustment are minimal) appears to be preserved.

4.3. Errors

4.3.1. Anticipatory errors

No significant group difference was observed on AU suggesting that children with DCD were able to inhibit inappropriate movement and/or maintain finger contact with the home base as well as control children (see also Hyde & Wilson, 2011). It is noteworthy, however, that others have reported inhibition problems in DCD using tasks of executive function (e.g., Fink, Dyk, Franks, & Convell, 2007). The particular parameters under which these problems might manifest is a topic of further investigation.

4.3.2. Touch down errors

As predicted, averaged across conditions, children with DCD committed significantly more TE’s when compared to controls. This result is in line with previous research showing decreased accuracy in DCD (Santos-Engelmann, Wensberg, & Dayen, 2003) as well as our earlier study using the same perturbation task (Hyde & Wilson, 2011).

4.3.3. Center touch errors

Children with DCD showed a trend for committing significantly more CTE’s though the effect was not significant (p = 0.07); this finding again accords with our earlier study. CTE’s reflect an individual’s capacity to correct an initiated motor response in following environmental changes in a timely fashion. Accordingly, these results suggest that children with DCD experience difficulty correcting or stopping movement in response to target perturbation prior to the initial motor command unfolding completely. To reiterate, as argued in Hyde and Wilson (2011) that there were two potential explanations for this pattern of performance: firstly, since efficient correction of reaching in response to a target jump requires the seamless integration of predictive models of movement with feedback based mechanisms, the increase in CTE’s seen in children with DCD may reflect a deficit in integrating predictive and feedback based mechanisms (ala the BMD hypothesis). However, the average number of CTE’s committed was roughly two out of 16 per child with DCD, limiting the capacity for drawing inferences about online control. The second, and more likely explanation for the increased CTE’s seen in children with DCD, is that they may suffer from an impaired ability to inhibit responses to compelling (though inappropriate) stimuli (see Hyde & Wilson, 2011 for a detailed analysis).

4.4. Future directions and limitations

There is now converging evidence that children with DCD have difficulty making rapid online adjustments to visual perturbations, suggesting an underlying problem with predictive motor control. Establishing whether this deficit reflects a developmental immaturity or deviance from typical development is important to our understanding of the role of predictive control in skill acquisition during childhood and the design of remedial programs. For example, in the case of developmental immaturity, it is assumed that children with DCD have the capacity to ‘catch-up’ to their same-age peers and hence remedial effort should focus on assisting children with DCD attain age-appropriate skill using conventional strategies (van Breukelen, Batchelor, Ceule, Boe, & Bourmaa, 2010). Conversely, in the case of deviance, it is assumed that impairment will persist without vigorous treatment (McConnel, 1998; van Breukelen et al., 2010) and that remediation would be better directed at the development of compensatory strategies that will minimize the degree of impairment (Miller & Buchholtz, 2006). Deviance versus immaturity can be established using well-designed cross-sectional studies comparing the motor performance of children with DCD and younger controls. If the performance of children with DCD is of a similar profile and level to that of younger (neurologically immature) controls, then developmental immaturity is inferred. Alternatively, if the pattern does not fit along a normal developmental trajectory, then deviance is inferred. This method, for example, has been used to establish developmental immaturity as the likely explanation for impaired postural control in DCD (Wanne, More-Williams, & Rambert, 1998), prone skills (Hill, Bishop, & McConnel-Smith, 1988) and covert-orienting of attention (Willott, Brown, & Wanne, 2007).

There is a strong case for use of neuroimaging methods to clarify the nature of any neurocognitive impairment in DCD. The speed and efficiency with which children with DCD can implement rapid online corrections appear to be compromised, but not to the same degree as parietal patients who exhibit prolonged TC (e.g., mean TC = 516 ms vs. 578 ms for DCD children) and often an inability to correct movement online at all (Gria et al., 2002). Thus, despite similarities in the pattern of performance between these two groups, it is unlikely that DCD is attributable to parietal lesions. However, the impairments in predictive control (incapacitated earlier under the BMD hypothesis—see Wilson, 2005), are compelling enough to suggest a likely neurodevelopmental delay at the level of PFC or the precuneus-parietal axis more generally (De Genes & Graf, 2003; De Genes & Strigo, 2009). There is a need for neuroimaging data to test this hypothesis.

Finally, given variability in the presentation of DCD, we cannot assert that a deficit in predictive control explains all cases of DCD. What we can say is that there is good evidence to suggest that most children with DCD have difficulties implementing forwards models, and that this affects their ability to alter movements in flight. Whether these difficulties reflect a developmental delay is an issue of ongoing investigation.

4.5. Conclusion

The present study replicated our earlier chronometric analysis of double-step reaching in DCD impaired ROC was indicated by increased movement time on jump trials. Kinematic data allowed us to isolate the locus of the impairment in DCD. Early kinematic markers (UP and IP) failed to show any group differences. However, children with DCD were significantly slower to correct the trajectory of their reaching away from the initial target on jump trials. No abnormalities were shown for the post-correction phase of jump trials: there were no group differences on PCT and TD errors. Taken together, there is evidence that children with DCD show some impaired online control during the early stages of double-step reaching. These early online changes in trajectory are predicated on the ability to generate and monitor forward estimates of limb position that specify the relative position of the hand and target. A reduced ability to integrate the resulting error signals with the ongoing motor command on jump trials may explain the longer TC in children with DCD, and hence their atypical pattern of performance. Intriguingly, the kinematics of double-jump reaching in DCD showed some similarities to parietal patients;
but, we urge caution when drawing strong inferences about the neuromotoric aspects of DCD. In some, the strong effect sizes observed in this and our previous study provide converging evidence for impaired predictive control in DCD. It is possible that this deficit is attributable to some immaturity at the level of the PFC or parieto-cerebellar axis.

Acknowledgments

We would like to thank Ray Duckman for his technical support throughout the development of the double-step task and Prof. Paul Mansell of CogState Ltd. for his assistance with equipment. For their cooperation and good humor during testing, we would also like to thank the staff and students of Tinternale and Westerfjell Primary Schools.

References


Appendix A

Published Articles


Appendix B.  Parent Plain Language Statement and Consent Form, Study 1, 2 & 3

INVITATION TO PARTICIPATE IN A RESEARCH PROJECT

PROJECT INFORMATION STATEMENT

Project Title:
- Assessing the strategies people use to complete movements and how these change with development

Investigators:
- Mr Christian Hyde (BSc, Grad. Dip. Psych) (PhD student), christian.hyde@student.rmit.edu.au.
- Dr Peter Wilson (Project Supervisor: Associate Professor, Psychology, RMIT University, peter.h.wilson@rmit.edu.au, (03) 9925 2906.

Dear Parent,

Your child has been invited to participate in a research project being conducted by RMIT University. This information sheet describes the project in straightforward language, or ‘plain English’. Please read this sheet carefully and be confident that you understand its contents before deciding whether or not you wish for your child to participate. If you have any questions about the project, please ask one of the investigators.

Who is involved in this research project? Why is it being conducted?

My name is Christian Hyde and I am conducting a research project with Associate Professor Peter Wilson in the School of Psychology towards a Doctorate of Philosophy (Psychology) at RMIT University. This means that I will be preparing a research report from the results of this study. I would like to invite your child to participate in this research. This project has been approved by the RMIT University Human Research Ethics Committee and the Research Branch of the Victorian Government Department of Education and Early Childhood Development.

Why has my child been approached?

The Principle of your child’s school, insert name, has agreed to allow us to approach students of (insert the name of the relevant school) to invite them to participate in our project.

What is the project about? What are the questions being addressed?

Our project “Assessing the strategies people use to complete movements and how these change with development” is investigating the ways that people perform movements and how the strategies people use change as they get older. The aim of our study is for us to develop a better understanding of how the strategies that people use to perform movements change over time as well as the reasons why some people have more difficulty performing movements than others. Approximately 60 participants are expected to be involved on the study.

If I agree for my child to participate, what will they be required to do?

The study will involve your child doing some computer-based tasks. The first task will involve them pointing and touching one of a number of targets on a computer screen as quickly and as accurately as possible. We will either be assessing the time it takes them to complete each task, or the way in which they move their arms to do so. The second task will involve them following a target around a computer screen. Here we will be assessing how quickly and accurately they can complete this task. The research will take approximately 30 to 45 minutes of your child’s time.
Appendix B  Parent Plain Language Statement and Consent Form

What are the risks or disadvantages associated with participation?
Occasionally, people find being assessed uncomfortable or upsetting. If at any stage during the study your child feels uncomfortable or upset because of something required of them by the research, they are encouraged to let the researcher know and the assessment will cease.

What are the benefits associated with my child’s participation?
There will be no direct benefit to your child for participating in this research. We hope that the findings of this study will help to further our knowledge about the way that people become efficient at performing movements. We also hope that the results will assist in understanding why some people have more difficulty coordinating movements than others.

There will be no financial benefit or reward for participating in this study.

What will happen to the information that my child provides?
All aspects of the study, including results, will be strictly confidential and only the researchers will have access to information on participants. To maintain confidentiality your child’s name will not appear on any of the data. A code number will be assigned to your child’s data. The consent forms which you will sign will not be kept in the same place as your child’s results so there will be no way to identify which results have been obtained from your child.

Storage of the data collected will adhere to the University regulations and be kept in secure storage for 5 years. A report of the study may be submitted for publication, but individual participants will not be identifiable in such a report, as only aggregated group data will be reported.

In order to assist with research examining movement development, your child’s anonymous data may be used for other projects in this area. All data will be completely anonymous and your child’s identity will not be disclosed.

What are my child’s rights as a participant?
As this study is completely voluntary you and your child are under no obligation to consent to participation and your child may withdraw at any stage for any reason. Your child has the right to ask questions regarding the project at any time.

Whom should I contact if I have any questions?
If you have any queries or would like to be informed of the aggregate research findings, please contact Dr Peter Wilson on (03) 9925 2906 or peter.h.wilson@rmit.edu.au. Should you or your child have any concerns about the conduct of this research project, please contact Dr Prof. Peter Wilson on the contact details above.

Yours sincerely,

Christian Hyde  Dr Peter Wilson

Any complaints about your child’s participation in this project may be directed to the Executive Officer, RMIT Human Research Ethics Committee, Research & Innovation, RMIT, GPO Box 2476V, Melbourne, 3001. Details of the complaints procedure are available at: http://www.rmit.edu.au/rd/hrec_complaints
Appendix B

Parent Plain Language Statement and Consent Form

**Portfolio**
**School of**

Name of participant:

Project Title:

Name(s) of investigators:

(1) Christian Hyde

(2) Dr Peter Wilson

---

1. I have received a statement explaining the tests/procedures involved in this project.

2. I consent to my child’s participation in the above project, the particulars of which - including details of tests or procedures - have been explained to me.

3. I authorise the investigator or his or her assistant to use with my child the tests or procedures referred to in 1 above.

4. I acknowledge that:
   
   (a) The possible effects of the tests or procedures have been explained to me to my satisfaction.
   
   (b) I have been informed that my child is free to withdraw from the project at any time and to withdraw any unprocessed data previously supplied (unless follow-up is needed for safety).
   
   (c) The project is for the purpose of research and/or teaching. It may not be of direct benefit to my child.
   
   (d) The privacy of the personal information my child provides will be safeguarded and only disclosed where I have consented to the disclosure or as required by law.
   
   (e) The security of the research data is assured during and after completion of the study. The data collected during the study may be published, and a report of the project outcomes will be provided to Dr Peter Wilson. Any information which will identify my child will not be used.

---

**Participant’s Parent’s Consent**

I consent to the participation of ____________________________ in the above project.

**Signature:**

(1)

(2)

**Date:**

**(Signatures of parents or guardians)**

**Witness:**

**Date:**

(Witness to signature)

---

Any complaints about your child’s participation in this project may be directed to the Executive Officer, RMIT Human Research Ethics Committee, Research & Innovation, RMIT, GPO Box 2476V, Melbourne, 3001. The telephone number is (03) 9925 2251.

Details of the complaints procedure are available from the above address.
Appendix C.  Child Plain Language Statement and Consent Form (A) for 7-12 year olds, Studies 1, 2 & 3

INVITATION TO PARTICIPATE IN A RESEARCH PROJECT
PROJECT INFORMATION SHEET- CHILD VERSION

January XXth, 20XX

This information sheet is for you to keep.

Hello, my name is Christian Hyde and I would like to invite you to participate in a project that I am conducting with Dr. Peter Wilson from RMIT University. The aim of this project is to learn about how children move.

What will I be doing?
You will be asked to do some activities that many children enjoy doing like threading beads, balancing on one leg, and jumping as far as you can. This project will also involve doing some activities on a computer. We will also ask you to quickly touch objects that light up on a computer screen. Finally, we will ask you to follow a red dot around a computer screen. We will measure how you go. This will help us learn more about how children do things and how they grow.

What if I don’t want to take part in the project?
You do not have to take part in this project if you do not want to. Also, if you do decide to join in the project but change your mind at any time, you are free to stop whenever you want. There will be no penalty if you decide to stop at any time during the project.

What if I do want to take part in the project?
Please sign the sign the form.

Thank you. 😊
Consent Form (Children)

I agree to take part in the project which has been described above.

I agree to joining in on some activities using a computer

I agree to doing some everyday activities such as balancing on one leg, and jumping as far as I can

And I know that I do not have to take part in this project and am free to stop whenever I want

Participant’s name

Signature

Date / /
Appendix D. Child Plain Language Statement and Consent Form (B) for 5-6 year olds, Study 3

INVITATION TO PARTICIPATE IN A RESEARCH PROJECT
PROJECT INFORMATION SHEET- CHILD VERSION

What is this project about?
- Learning about how children move

Who is running this project?
- Mr Christian Hyde & Dr. Peter Wilson from RMIT University
  peter.h.wilson@rmit.edu.au, tel. 9925-2906

What will I do?
You have been chosen to be part of a project about how school children learn new skills like catching, throwing, and jumping.

You will be asked to do some activities that most children really enjoy like threading beads, balancing on one leg, and jumping as far as you can. We will also ask you to quickly touch objects that light up on a computer screen. Finally, we will ask you to follow a red dot around a computer screen.

We will measure how you go. This will help us learn more about how children do things and how they grow.

Would you like to be part of the project?

<table>
<thead>
<tr>
<th>YES</th>
<th>OR</th>
</tr>
</thead>
<tbody>
<tr>
<td>No</td>
<td>NO!</td>
</tr>
</tbody>
</table>

Yes, I would like to do the activities. – Please sign your name □

Thank you. ☺

Name: ___________________________________ Date: _______________
(Signature of Principal Investigator)

Name: ___________________________________ Date: _______________
(Signature of Project Supervisor)
Appendix E. Adult Participant Plain Language Statement and Consent Form, Study 3

INVITATION TO PARTICIPATE IN A RESEARCH PROJECT

PROJECT INFORMATION STATEMENT

Project Title:
o Assessing the strategies people use to complete movements and how these change with development

Investigators:
o Mr Christian Hyde (BSc, Grad. Dip. Psych) (PhD student), christian.hyde@student.rmit.edu.au.
o Dr Peter Wilson (Project Supervisor: Associate Professor, Psychology, RMIT University, peter.h.wilson@rmit.edu.au, (03) 9925 2906.

Dear Participant,

You are invited to participate in a research project being conducted by RMIT University. This information sheet describes the project in straightforward language, or ‘plain English’. Please read this sheet carefully and be confident that you understand its contents before deciding whether to participate. If you have any questions about the project, please ask one of the investigators.

Who is involved in this research project? Why is it being conducted?
My name is Christian Hyde and I am conducting a research project with Associate Professor Peter Wilson in the School of Psychology towards a Doctorate of Philosophy (Psychology) at RMIT University. This means that I will be preparing a research report from the results of this study. I would like to invite you to participate in this research. This project has been approved by the RMIT Human Research Ethics Committee.

Why have you been approached?
You have been approached because you are a student of RMIT University.

What is the project about? What are the questions being addressed?
Our project “Assessing the strategies people use to complete movements and how these change with development” is investigating the ways that people perform movements and how the strategies people use change as they get older. The aim of our study is for us to develop a better understanding of how the strategies that people use to perform movements change over time as well as the reasons why some people have more difficulty performing movements than others. Approximately 60 participants are expected to be involved in the study.

If I agree to participate, what will I be required to do?
The study involves doing some computer-based tasks. The first involves pointing and touching one of a number of targets on a computer screen as quickly and as accurately as possible. We will either be assessing the time it takes you to complete each task, or the way in which you move your arms to do so. The second task involves following a target around a computer screen. Here we will be assessing how quickly and accurately you can complete this task. The research will take approximately 30 to 45 minutes of your time.
Occasionally, people find being assessed uncomfortable or upsetting. If at any stage during the study you feel uncomfortable or upset because of something required of you by the research, please let the researcher know.

**What are the risks or disadvantages associated with participation?**
Occasionally, people find being assessed uncomfortable or upsetting. If at any stage during the study you feel uncomfortable or upset because of something required of you by the research, please let the researcher know.

**What are the benefits associated with participation?**
There will be no direct benefit to you for participating in this research. We hope that the findings of this study will help to further our knowledge about the way that people become efficient at performing movements. We also hope that the results will assists in understanding why some people have more difficulty coordinating movements than others.

There will be no financial benefit or reward for participating in this study.

**What will happen to the information I provide?**
All aspects of the study, including results, will be strictly confidential and only the researchers will have access to information on participants. To maintain confidentiality your name will not appear on any of the data. A code number will be assigned to your data. The consent form which you will sign will not be kept in the same place as your results and so there will be no way to identify which results have been obtained from you.

Storage of the data collected will adhere to the University regulations and be kept in secure storage for 5 years. A report of the study may be submitted for publication, but individual participants will not be identifiable in such a report, as only aggregated group data will be reported.

In order to assist with research examining movement development, your anonymous data may be used for other projects in this area. All data will be completely anonymous and your identity will not be disclosed.

**What are my rights as a participant?**
As this study is completely voluntary you are under no obligation to consent to participation and you may withdraw at any stage for any reason. You have the right to ask questions regarding the project at any time.

**Whom should I contact if I have any questions?**
If you have any queries or would like to be informed of the aggregate research findings, Please contact Dr Peter Wilson on (03) 9925 2906 or peter.h.wilson@rmit.edu.au. Should you have any concerns about the conduct of this research project, please contact Dr Prof. Peter Wilson on the contact details above.

Yours sincerely,

Christian Hyde

Dr Peter Wilson
Appendix E

Science, Engineering and Technology
Health Sciences

Assessing the strategies people use to complete movements and how these change with development

Name(s) of investigators: (1) Christian Hyde
(2) Dr Peter Wilson

5. I have received a statement explaining the tests/procedures involved in this project.

6. I consent to participate in the above project, the particulars of which - including details of tests or procedures - have been explained to me.

7. I authorise the investigator or his or her assistant to use with me the tests or procedures referred to in 1 above.

8. I acknowledge that:

(d) The possible effects of the tests or procedures have been explained to me to my satisfaction.
(e) I have been informed that I am free to withdraw from the project at any time and to withdraw any unprocessed data previously supplied (unless follow-up is needed for safety).
(f) The project is for the purpose of research and/or teaching. It may not be of direct benefit to me.
(d) The privacy of the personal information I provide will be safeguarded and only disclosed where I have consented to the disclosure or as required by law.
(e) The security of the research data is assured during and after completion of the study. The data collected during the study may be published, and a report of the project outcomes will be provided to Dr Peter Wilson. Any information which will identify me will not be used.

Participant’s Consent

Participant: ____________________________ Date: ________________
(Signature)

Witness: ____________________________ Date: ________________
(Signature)

Any complaints about your participation in this project may be directed to the Executive Officer, RMIT Human Research Ethics Committee, Research & Innovation, RMIT, GPO Box 2476V, Melbourne, 3001. The telephone number is (03) 9925 2251.

Details of the complaints procedure are available from the above address.