Hot Executive Function in Children with Developmental Coordination Disorder

Submitted by

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Statement of Sources

This thesis contains no material published elsewhere or extracted in whole or in part from a thesis by which I have qualified for or been awarded another degree or diploma. No other person's work has been used without due acknowledgement in the main text of the thesis. The nature of any other assistance received in the pursuit of the research and preparation of the thesis and the extent of collaborations with another person or persons have also been acknowledged. This thesis has not been submitted for the award of any degree or diploma in any other tertiary institution. All research procedures reported in the thesis received the approval of the relevant Ethics/Safety Committees (where required).

Signed:

Shahin Rahimi-Golkhandan

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List of Tables

List of Figures

List of Acronyms

Acronym	Meaning
ABD	Atypical Brain Development
ADHD	Attention Deficit/Hyperactivity Disorder
ANOVA	Analysis of Variance
APA	American Psychiatric Association
ASD	Autism Spectrum Disorders
BMI	Body Mass Index
BOLD	Blood Oxygen Level Dependent
BRIEF-A	Behavior Rating Inventory of Executive Function-Adult
CCC-R	Cognitive Complexity and Control - Revised
CE	Commission error
CFA	Confirmatory factor analysis
CI	Confidence interval
CN	Cognitive neuroscience
CNS	Central nervous system
DAMP	Deficits in Attention, Motor control, and Perception
DCCS	Dimensional Change Card Sort
DCD	Developmental Coordination Disorder
DL-PFC	Dorsolateral prefrontal cortex
DSM	Diagnostic and Statistical Manual of Mental Disorders

Acronym	Meaning
DTI	Diffusion tensor imaging
EF	Executive function
ER	Emotion-regulation
FA	Fractional anisotropy
FC	Functional connectivity
fMRI	Functional Magnetic Resonance Imaging
HDT	Hungry Donkey Task
IGT	Iowa Gambling Task
IMD	Internal Modelling Deficit
IP	Information processing
ISI	Inter-stimulus interval
L-PFC	Lateral prefrontal cortex
MABC	Movement Assessment Battery for Children
MAND	McCarron Assessment of Neuromuscular Development
MANOVA	Multivariate Analysis of Variance
NDI	Neurodevelopmental Index
OE	Omission error
OFC	Orbitofrontal cortex
PFC	Prefrontal cortex
PPC	Posterior parietal cortex

Acronym	Meaning
RT	Reaction time
SES	Socioeconomic status
SLI	Specific Language Impairment
SMH	Somatic Marker Hypothesis
TD	Typically-developing
VM-PFC	Ventromedial prefrontal cortex
WM	Working memory
WCST	Wisconsin Card Sorting Test
WMN	White matter network
WISC-IV	Wechsler Intelligence Scale for Children-Version IV

Abstract

Deficits of cool executive function (EF) have been shown in children with motor problems (or Developmental Coordination Disorder—DCD), with implications for the planning of goal-directed action. However, there is little if any work on the possible link between DCD and hot EF. Given that hot EF predicts important developmental outcomes and underlie age-appropriate cognitive and social functioning, it was the broad aim of my thesis to investigate hot EF in DCD using state-of-the-art measures designed for children.

EF is an umbrella term that refers to a set of neurocognitive processes involved in conscious and effortful control of thought, emotion, and behaviour. Broadly, it can be divided into cool and hot EF. Cool EF is mainly subserved by lateral prefrontal cortex (L-PFC), enlisted when one deals with abstract and decontextualised stimuli. In contrast, hot EF is linked to ventromedial prefrontal cortex (VM-PFC), active in many real-life situations that are characterised by high affective involvement; here, one needs to consider or reappraise the emotional/motivational significance of stimuli and refrain from impulsive actions.

Study 1: Participants were 14 children with DCD and 22 typically developing (TD) children aged between 6 and 12 years. Motor skill was assessed using the McCarron Assessment of Neuromuscular Development (MAND). The measure of hot EF was the 100-trial version of the Hungry Donkey Task (HDT). Participants select from a set of four doors which provide gains or losses in rewards. Two doors (A & B) are disadvantageous in the long run (high immediate reward, but high loss overall), while the other two (C & D) are advantageous (low immediate reward, but low loss overall). **Results:** DCD group performed significantly worse than the controls on the HDT, making more selections from A and B, and less from C and D. While both groups chose doors with low frequency loss (B & D) more often, the DCD group opted for door B which yields high infrequent loss, but also high reward. **Discussion:** The HDT, as a proportional reasoning task, can be adequately performed

if one considers frequency of loss on each choice as the dominant dimension, and the amount of loss as the subordinate dimension. Both groups considered frequency of loss as the dominant dimension; however, DCD group opted for a choice which yields infrequent loss, but also high reward. Therefore, constant reward, instead of probabilistic loss, served as the subordinate dimension of choice for the DCD group. This suggests DCD group may find it harder to resist rewarding stimuli, and points to potential deficits of hot EF in this cohort. The aim of the second study was to determine whether this apparent deficit of hot EF in DCD is explained selectively by a heightened sensitivity to rewarding stimuli, or is due to a general deficit of inhibitory control.

Study 2: Participants were 12 children with DCD and 28 TD children aged between 7 and 12 years. In addition to the MAND, children completed two versions of a go/no-go task tapping cool and hot EF. The cool version presented male and female faces with neutral expressions. Stimuli for the hot version were fearful and happy faces. Both expressions were used as targets and non-targets in each version, giving four conditions. **Results:** Like earlier studies, all children responded faster to happy faces. Both groups showed comparable accuracy in response to go targets, and also had similar commission errors, except when the no-go stimulus was a happy face. The DCD group had significantly higher false alarms than controls, and failed to withhold a response to happy faces on more than 50% of the trials. **Discussion:** These results suggest a heightened sensitivity to emotionally significant distractors in DCD. This type of impulsivity may undermine self-regulation in DCD, with possible implications for adaptive function and emotional well-being. High sensitivity to appetitive, rewarding cues undermines the ability to control thoughts and actions, and, consequently, disrupt self- and emotion-regulation. However, an additional aspect of emotion-regulation and an important predictor of adaptive functioning in hot, affective contexts is the ability to approach negatively valenced stimuli. The ability to approach

negative stimuli can be measured experimentally by response time on a go/no-go task. However, these effects can be moderated by the choice of no-go stimuli. The use of happy no-go faces on 30% of the trials in the second study may have created an approach bias that also influenced responses to go stimuli. Moreover, fearful faces might not be the best expression to represent a *negative* stimulus, particularly for children. In study 3, I optimised the assessment of emotion-regulation and sensitivity to reward by pairing emotional expressions with neutral ones, and enlisting negative stimuli (i.e., sad faces) that were discriminable by children.

Study 3: The aim of the third study was twofold: First, to investigate potential differences between TD and DCD groups in approaching negatively valenced stimuli. Second, to obtain more accurate measures of sensitivity to reward in DCD. The participants were 12 children with DCD, and 24 TD children aged between 7 and 12. Children completed two blocks of an emotional go/no-go task in which neutral facial expressions were paired with either happy or sad faces. Each expression was used as both, a go and no-go target in different runs of the task. **Results:** There were no group differences in the ability to approach sad faces; however, the DCD group made significantly more commission errors to happy no-go faces. Analysis of reaction time, omission errors, and d', which measures sensitivity to each facial expression, showed that this difference was not attributable to emotion recognition difficulties in DCD. **Discussion:** Taken together, the particular pattern of performance in DCD does not suggest any differences between children with DCD and their TD peers in the ability to approach negatively valenced stimuli; however, it confirms earlier reports of (hot) EF deficits in DCD. Apart from the *general* negative effects of reduced inhibitory control in 'hot' contexts, EF deficits may *specifically* disrupt psychological and physical well-being in DCD by interacting with the environmental factors and stressors specific to this disorder.

Conclusion: The three studies reported here are the first ones that investigated and showed deficits of hot EF in DCD. The findings not only enhance our understanding of the cognitive mechanisms underlying DCD, but also point to the dire need to modify existing interventions for children with motor coordination problems. In sum, deficits of hot EF in DCD are linked to the interaction of cognitive control and emotion processing networks which may be disrupted in DCD or delayed in development. Emotionally significant, rewarding stimuli constitute a higher load on inhibitory control than neutral or negative stimuli; and children with DCD, who already have reduced response inhibition, find it more difficult to modulate their approach to rewarding stimuli when the task demands that this behaviour be inhibited.

The recent evidence of hot EF deficits in DCD suggests interventions that aim to improve EFs, in addition to enhancing functioning in the motor domain are probably more successful in improving quality of life in children with DCD. To elaborate how EF interventions could enhance both physical and psychological well-being in DCD, I developed a conceptual model which depicts how EF, particularly inhibitory control, may mediate the relationship between DCD and internalising problems. Emotional problems, in turn, mediate the relationship between DCD and physical consequences of the disorder. Follow-up research may extend the study of hot EF to adolescents and adults with DCD, and use neuroimaging techniques to enhance our understanding of the neural pathways underlying (hot) EF deficits in DCD.

Chapter 1: Literature Review

1.1 Overview

This chapter begins with an overview of the importance of motor development in shaping later developmental outcomes, particularly cognitive and social functioning. My review discusses the overlapping timescales of motor and cognitive development, and shows how typical development of motor function facilitates the acquisition of knowledge and the interactions with other individuals. I then describe the challenges posed by atypical motor development, with particular focus on the impact of Developmental Coordination Disorder (DCD), a relatively common neurodevelopmental disorder. The characteristics of DCD are reviewed, including its diagnostic markers, prevalence, comorbidity, prognosis, and the neural correlates. Different categories of research in DCD are reviewed next. In relation to current etiological accounts of DCD, I provide an in-depth review of the neurocognitive accounts of DCD (esp. deficits of predictive control and executive function – EF), and describe neural systems that have been implicated in both motor and cognitive development. I then present a review of the contemporary developmental theories of EF. A distinction is made between cool and hot EF, explained by the neural correlates and developmental trajectories of each. The importance of hot EF in predicting various developmental outcomes is highlighted. Deficits of cool EF are shown to be prominent in DCD, including deficits of response inhibition, working memory (WM), sustained attention, and set-shifting. Neural systems implicated in DCD are also associated with deficits of hot EF, which may have adverse psychosocial consequences. However, there is very little if any data on hot EF in this cohort. I conclude the chapter by translating this knowledge gap into a set of key aims that are the focus of my work in this thesis.

1.2 The Interactive Nature of Development: Relationship between Cognition and Action

Our understanding of the world relies on the ability to navigate the environment, manipulate objects, and interact with others (Pezzulo, 2011). This view is in line with the

embodied cognition hypothesis which posits that cognitive processes are shaped by the interaction of the body with the environment. More specifically, the evolution of the brain shows that our neural systems were first and foremost in the service of perceptual and motor processing which afforded immediate, on-line interactions with the environment, and sculpted human cognition. Thus, the embodiment perspective suggests that cognition is not centralized and abstract, but rather has deep roots in sensorimotor processing (M. Wilson, 2002), and that common biological bases underlie both motor and cognitive control (P. H. Wilson, 2015). A range of different motor skills, which are essential for daily living activities, enable such an embodied cognition. In spite of this close relationship between motor and cognitive abilities, these two aspects of development have often been studied separately (Leonard & Hill, 2015). There is, however, an increasing awareness about the impact of development in one domain on the other.

Fundamental motor skills that infants learn and refine early in development predict their social and cognitive abilities later in life (Gonzalez et al., 2014). For instance, the transition from crawling to walking improves infants' ability to actively explore the environment, increases their opportunities for learning, and facilitates the sharing of those experience with other individuals, resulting in more advanced social interaction behaviours (Clearfield, Osborne, & Mullen, 2008; Karasik, Tamis-LeMonda, & Adolph, 2011; Leonard & Hill, 2014). In a similar vein, motor performance during infancy has been shown to predict cognitive functioning at 1 year (Metgud, Patil, & Dhaded, 2011). Findings such as these suggest that disruptions or delays in motor and/or cognitive development can have far-reaching consequences on the other aspects of development.

Existing neurobiological data also show how seemingly unrelated domains of motor, cognitive and social function become interdependent. For instance, sucking and feeding skills during infancy as well as speech production in early childhood are subserved by overlapping

neural networks that are also involved in the development of cognitive control later (Koziol, Budding, Chidekel, 2011). The basic perceptual and motor skills learned in infancy activate similar, or even identical, brain regions as the more complex skills acquired in adulthood (Johnson, 2011). Thus, even though the complexity of skills required for adaptive functioning increases dramatically as children age (e.g., from basic sensorimotor skills to complex cognitive ones), the maturation of neural networks underlying both motor and cognitive control affects functioning across the lifespan in different domains (Johnson, 2011).

The relationship between the development of motor and cognitive control has been demonstrated in different studies. For example, children with learning disabilities aged between 7-12 years perform more poorly than their same-age typically-developing (TD) peers on measures of locomotor and object-control skills as well as reading and mathematical abilities (Westendorp, Hartman, Houwen, Smith, & Visscher, 2011). In a similar vein, gross motor skills, measured between birth and 4 years of age, predicted scores on the Wechsler Intelligence Scale for Children-Version IV (WISC-IV; Wechsler, 2004) in primary school, even after controlling socioeconomic status (SES) (Piek, Dawson, Smith, & Gasson, 2008). Another longitudinal study by Bart, Hajami, and Bar-Haim (2007) discovered that motor abilities at ages 5-6 year predicted different cognitive skills (e.g., scholastic adaptation) and social behaviours (e.g., disruptive, withdrawn, prosocial) a year later. Ommundsen, Gunderson, and Mjaavatn (2010) also found a direct relationship between motor functioning at 6-7 years and social status – among a group of peers – between the ages of 9-10 years. This could explain reduced participation of children with motor coordination problems in social play and their high rates of social isolation (e.g., Bar-Haim & Bart, 2006).

1.2.1 The overlapping timescales of development and activation. Converging evidence suggests there is an overlap between the timescales of motor and cognitive development (Diamond, 2007). The emergence of cognitive control abilities related to

behaviour planning and response inhibition coincides with the development of motor control and visuomotor coordination in the early period of life (Gonzalez et al., 2014). An example of this is the gradual improvement between the ages of 3-10 years in prospective motor control. Using an end-state comfort paradigm, children are asked to pick up a wooden sword which is placed in six different orientations, and fit it inside a hole. The ability to adopt an awkward initial grasp in order to achieve a final end-state comfort develops gradually over childhood, with biggest improvements observed between the ages of 3 and 4 years, as well as 9 and 10 years (Jongbloed-Pereboom, Nijhuis-van der Sanden, Saraber-Schiphorst, Crajé, & Steenbergen, 2013). This age-related increase in anticipatory action planning and response inhibition on a motor task suggests an interactive relationship between cognition and action, which is mirrored in the close interrelation of brain regions involved in motor (e.g., the cerebellum) and cognitive (e.g., the prefrontal cortex [PFC]) control (Leonard & Hill, 2015). Middle childhood is marked by an expansion of the scope of goal-directed action and the reorganisation of underlying neural networks (P. H. Wilson, 2015). The reorganisation of motor control temporally overlaps with the expansion of cognitive control systems. Over this period, there is an increasing level of neural coupling between the frontal, control networks and the posterior regions implicated in motor control (Johnson, 2005).

Indeed, existing literature suggests the cerebellum and the PFC are structurally and functionally related (Koziol et al., 2011). To illustrate, both novel motor and cognitive tasks result in the co-activation of the cerebellum and the PFC (Tal Saban, Ornoy, Parush, 2014). Some of the frontal regions, such as the dorsal premotor cortex, are also activated during both cognitive control and motor control/learning tasks. The premotor cortex is involved in planning, selection, organisation, and execution of actions (Abe & Hanakawa, 2009; Hanakawa, 2011). Diamond (2000) argued that the motor and cognitive deficits seen in a

range of neurodevelopmental disorders are actually driven by this close interrelation and co-activation of the neural systems underlying cognitive control and motor skills.

1.2.2 Atypical development. From the perspective of the '*interactive specialisation*' (Johnson & Munakata, 2005), the negative consequences of disruptions or a delay in the development of one of these interacting systems are not limited to that particular aspect of development, but have compounding effects on the other systems, too. A neurological disruption at an early age in one specific area could underlie cognitive and/or motor deficits linked to a range of neurodevelopmental disorders, even if the core symptoms of those disorders are seemingly disparate. For instance, attention deficit/hyperactivity disorder (ADHD), autism spectrum disorder (ASD), and dyslexia vary in terms of their main characteristic symptoms. However, all of these neurodevelopmental disorders have also been linked to difficulties in the motor domain (Diamond, 2000). An altered developmental trajectory of the frontal regions, associated with higher cognitive functions, could disrupt motor development in these cases. More specifically, concomitant deficits of motor and cognitive control are linked to the disruption of functional connectivity between nodes of a white matter network (WMN), or between two interacting networks (Sripada, Kessler, & Angsdadt, 2014).

The interactive relationship between motor function and cognitive control is not limited to a particular developmental stage. To illustrate, more than half of the university students with motor difficulties also show deficits in control of goal-directed action (Kirby, Sugden, Beveridge, Edwards, & Edwards, 2008). Additionally, delayed or disrupted development of motor control and learning may be an important basis for the detection of later cognitive impairments (Butcher et al., 2009; Iverson, 2010; Piek et al., 2008). Thus, an informed understanding of the nature of this relationship would facilitate detection and remediation of later deficits of cognitive control (Gonzalez et al., 2014).

The co-existence of motor and cognitive deficits in children with DCD provides good evidence of the interactive relationship between motor control/learning and cognitive functions (Leonard & Hill, 2015). DCD is a neurodevelopmental disorder characterized by difficulties learning motor skills, despite the opportunity to do so (APA, 2013). This disorder is associated with deficits in motor control, *as well as* a range of disturbances in the cognitive domain, particularly deficits of response inhibition, visuospatial memory, and executive attention (P. H. Wilson, Ruddock, Smits-Engelsman, Polatajko, & Blank, 2013). In the next section I provide a detailed description of the diagnostic characteristics of DCD, its prevalence, prognosis, comorbidity, as well as the main neural correlates of the disorder that have also been implicated in the development of cognitive control. I will then review the main categories of research in DCD, and focus mainly on the neurocognitive accounts of DCD.

1.3 Developmental Coordination Disorder (DCD)

1.3.1 History of the term. Since the early 1960s when terms like ‘developmental clumsiness’ were used to describe children with motor learning difficulties, a number of other labels have been used, many reflecting the particular assumptions of the researcher or period. Some of the more common terms have included: clumsy child syndrome (Gubbay, 1975), sensory integrative dysfunction (Ayres, 1972), developmental dyspraxia (Cermak, 1985), physical awkwardness (Miyahara & Register, 2000), and perceptual motor dysfunction (Laszlo & Sainsbury, 1993). In Scandinavia, motor coordination problems in children have been conceptualised as part of broader syndromes such as ‘Deficits in Attention, Motor control, and Perception’ (DAMP, Gillberg, 2003) and before that, ‘minimal brain dysfunction’ (Wender, 1973). However, in an attempt to standardise the terminology and use a term that properly describes the condition, there has been a general consensus since 1994 to use the diagnostic term DCD – first introduced in the third edition of the Diagnostic and

Statistical Manual of Mental Disorders (DSM-3; APA, 1987) – to refer to motor problems that occur independently of other conditions (Polataijko, Fox, & Missiuna, 1995). Recent consensus statements (e.g., Blank, Smits-Engelsman, Polatajko, & P. H. Wilson, 2012) have also favoured the use of DSM diagnostic criteria to describe children whose motor coordination problems are not attributed to a known medical cause, but interfere with their activities of daily living and academic achievement.

1.3.2 DCD according to DSM-5. According to the most recent edition of DSM (APA, 2013), children with DCD often experience a range of motor problems such as delay in achieving developmental motor milestones (e.g., walking, sitting), and deficits in fine and/or gross motor skills (e.g., issues with handwriting or performance in sport). DCD is also associated with deficits in the acquisition/control of motor-related activities, despite the opportunity to do so (Criterion A). The motor performance of children with DCD is often slower, less accurate, and more variable than that of their same age TD peers (Piek et al., 2004; Zwicker, Missiuna, Harris, & Boyd, 2012). These challenges reduce the participation of children with DCD in physical activities, and consequently lead to poor physical fitness and higher rates of obesity in this cohort. DCD is indeed a chronic neurodevelopmental disorder in which motor coordination difficulties significantly disrupt daily living activities and academic achievement (Criterion B). Motor coordination problems are manifested at an early age, and include difficulties with learning typical childhood motor skills such as walking, running, catching, throwing, tying shoes, riding a bicycle, handwriting, and so forth (Criterion C). The disorder is not due to a general medical condition (Criterion D); and – unlike other motor disorders such as cerebral palsy – the intelligence levels of children with DCD are in line with what is expected of them given their chronological age (Kirby, Sugden, & Purcell, 2014). However, despite average or above average intelligence, difficulties with self-care, writing, typing, painting, drawing, and play disrupt the performance of children

with DCD in academic settings. Table 1 presents the most recent diagnostic criteria of DCD as presented in DSM-5 (APA, 2013).

Table 1.1

The Diagnostic Criteria for Developmental Coordination Disorder in DSM-5

Criterion	Description
A	The acquisition and execution of coordinated motor skills is substantially below that expected given the individual's chronological age and opportunity for skill learning and use. Difficulties are manifested as clumsiness (e.g. dropping or bumping into objects) as well as slowness and inaccuracy of performance of motor skills (e.g. catching an object, using scissors or cutlery, handwriting, riding a bike or participating in sports).
B	The motor skills deficit in criterion A significantly and persistently interferes with activities of daily living appropriate to chronological age (e.g. self-care and self-maintenance) and impacts academic/school productivity, prevocational and vocational activities, leisure and play.
C	Onset of symptoms is in the early developmental period.
D	The motor skills deficits are not better explained by intellectual disability (intellectual developmental disorder) or visual impairment and are not attributable to a neurological condition affecting movement (e.g. cerebral palsy, muscular dystrophy, degenerative disorder).

1.3.3 Prevalence. Recent reviews of research in DCD (e.g., Zwicker et al., 2012) suggest the disorder is generally diagnosed in about 5-6% of school-aged children. However,

there is still debate about the exact prevalence of DCD. The use of different selection criteria and assessment tools across clinical and research studies could be one of the factors influencing this figure. For instance, the choice of different cut-off points on motor screening tools, or not checking against *all* of the diagnostic criteria – in Table 1.1 – would inflate the rates. Moreover, different lifestyle across cultures, and the use of different terms to describe this condition also determine the reported prevalence (Cairney, Veldhuizen, & Szatmari, 2010; Zwicker et al., 2012). However, even on conservative estimates of prevalence (i.e., 5-6%) and high levels of comorbidity with other developmental disorders, DCD is not a trivial disorder (Deng et al., 2014).

1.3.4 Comorbidity. Neurodevelopmental disorders, such as DCD, ADHD, and ASD, constitute a group of conditions that frequently co-occur with one another. The onset of these disorders, which are characterised by developmental deficits that affect cognitive and motor milestones as well as social and academic achievement, is in the early development period – before children reach school age. DCD, stereotypic movement disorder, and tic disorders belong to a class of neurodevelopmental disorders known as motor disorders. Thus, motor impairment per se cannot be the basis of DCD diagnosis. The clinical diagnosis of DCD requires a synthesis of medical and developmental history, physical examination (i.e., tests of visual and neurological function), school or workplace report, as well as the individual's performance on standardised and psychometrically sound measures of motor ability (APA, 2013). However, even in children with a clinical diagnosis of DCD, the possibility of secondary diagnoses is high. Indeed, the diagnosis of 'pure' DCD (i.e., without comorbid problems) is the exception rather than the rule (Peters & Henderson, 2008). DCD often overlaps with a range of other neurodevelopmental disorders including ADHD, ASD, and learning disabilities, particularly dyslexia and specific language impairment (SLI) (Kirby et al., 2014). Notably, about half of the children diagnosed with DCD also show the core

symptoms of ADHD such as impulsivity, attention deficits, and hyperactivity (Zwicker, Missiuna, & Boyd, 2009); and, about a third to half of children with SLI could be diagnosed with DCD, too (Kaplan, Crawford, Wilson, & Dewey, 1997). Likewise, more than 50% of those with dyslexia experience significant motor coordination problems (Iversen, Berg, Ellertsen, & Tonnessen, 2005). The co-occurrence of neurodevelopmental disorders, not only exacerbates symptom severity, but also requires greater caution in ascribing difficulties in daily activities to motor impairment (APA, 2013). However, even though there is some overlap between the neural networks implicated in DCD and other comorbid conditions (e.g., ADHD, SLI), neuroimaging data indicate these conditions should still be regarded as discrete disorders (Cairney, 2015).

1.3.5 Prognosis. Although the difficulties with the acquisition and execution of motor skills are evident from an early age (e.g., delay in transition from crawling to walking), problems with motor coordination often go unnoticed until they affect children's academic performance in primary school (Missiuna & Campbell, 2014). As a result, DCD is mostly diagnosed between the ages of 6 to 12 years (Barnhart, Davenport, Epps, & Nordquist, 2003). Some early studies (e.g., Fox & Lent, 1996; Sellers, 1995) suggested that children with DCD would outgrow their difficulties with motor control and learning; however, longitudinal research has shown that in around one half of cases motor difficulties persist into adolescence (Cantell, Smyth, & Ahonen, 2003; Hellgren, Gillberg, Gillberg, & Enerskog, 1993; Losse et al. 1991) and adulthood (Cousins & Smyth, 2003; Drew, 2005; Fitzpatrick & Watkinson, 2003; Kirby, Edwards, & Sugden, 2011; Missiuna, Moll, King, Stewart, & Macdonald, 2008). For individuals with persistent DCD, new challenges are faced during the transition to adolescence and early adulthood when higher demands are placed on academic and occupational functioning (e.g., driving a car, working with machinery, etc.). It is estimated

that DCD and its consequences continue to disrupt daily living activities in adulthood for about 75% of those who did not receive interventions as children (APA, 2013).

Moreover, the severity of motor impairment and comorbidity also determine the prognosis of DCD. To illustrate, children with mild motor impairments may experience remission of symptoms in adolescence (Cantell et al., 2003). McLeod, Langevin, Goodyear, and Dewey (2014) argued that an increase in functional connectivity with age between the primary motor cortex and the fronto-parietal networks involved in cognitive control, memory and visuospatial imagery underlies this symptom remission. Enhanced connectivity within fronto-parietal circuits increases inhibitory control and the ability to modulate motor responses in-flight; and consequently improves motor control. In contrast, brain development is disrupted to a greater extent among those with comorbid DCD and ADHD, in that no age-related increase in functional connectivity occurs between the primary motor cortex and any other brain structures (McLeod et al., 2014). This could be one of the factors explaining higher severity of DCD-related symptoms in children with comorbid diagnoses (Missiuna & Campbell, 2014) compared to those with a single diagnosis.

1.3.6 Neural correlates. There is converging evidence that problems with motor coordination and skill issues in DCD are linked to dysfunction (e.g., hypoactivity) in brain regions involved in motor learning (e.g., cerebellum, PFC, parietal cortex, and basal ganglia) or disruption of connectivity between neural networks (Ferguson, Jelsma, Versfeld, Smits-Engelsman, 2014; Zwicker, Missiuna, Harris, & Boyd, 2010). Some of these networks, including the frontal lobe, the cerebellum, and the basal ganglia, have also been implicated in the development of cognitive control (Diamond, 2000). In this section, I provide an overview of the main neural correlates of DCD.

1.3.6.1 Cerebellum. Children with DCD often do not perform as well as their TD peers on tests of cerebellar function. At a neurological level, they show impaired performance

on finger-to-nose touching and rapid alternating hand movement tasks (Ivry, 2003; Lundy-Ekman & Ivry, 1991). Children with motor coordination difficulties also show reduced performance in rhythmic finger tapping tasks (e.g., Piek & Skinner, 1999). This pattern of performance in DCD indicate deficits in precursors of motor control that are associated with cerebellar activity, namely motor adaptation and timing of motor responses (Cantin, Polatajko, Thach, & Jaglal, 2007; Ghez & Thach, 2000; Kagerer, Contreras-Vidal, Bo, & Clark, 2006). The cerebellum, which acts as an adaptive controller (Barlow, 2002), utilises feedforward information (error signals) and modifies motor activity by updating neural representations (internal models) of movement whenever there is some disparity between intended and actual movement (Imamizu et al., 2000; P. H. Wilson et al., 2013). This in-flight adjustment of motor activity by the cerebellum occurs during both novel and learned motor tasks (Ghez & Thach, 2000). Moreover, the slowness and reduced accuracy of movements in DCD have been linked to the poor postural control and impaired timing of more distal musculature – the two functions that are closely linked to the activity of the cerebellum (Estil, Ingvaldsen, & Whiting, 2002; Piek & Skinner, 1999). More specifically, the cerebellum has been implicated in the sequences and timing of muscle contraction (Barlow, 2002) and learning of timed motor responses (P. H. Wilson et al., 2013).

Immaturities in the development of the cerebellum, which underlie the aforementioned deficits of motor timing in DCD (Cantin et al., 2007), are commonly associated with prematurity (Diamond, 2000). Given that the cerebellum develops later and more slowly than most of the other brain regions (Ivry, 2003), it is highly vulnerable in developmental disorders. A maturational lag, or some developmental insult and microstructural damage to the cerebellum leads to reduced motor functional performance. I will review the current hypothesis about cerebellar involvement in DCD (i.e., '*deficits of predictive control*') under 'etiological accounts of DCD' – section 1.4.3.

1.3.6.2 Parietal cortex. Suboptimal function of the parietal cortex, which is involved in the processing of sensorimotor transformation and motor learning, has been linked to impaired motor performance in DCD (Ferguson et al., 2014; Kashiwagi & Tamai, 2013; P. H. Wilson, Maruff, & McKenzie, 1997). Children with DCD often have difficulties executing imagined movements, for example, predicting the duration of imagined actions as a function of task difficulty. This performance pattern suggests impairment in the processing of an efference copy, a process that is subserved by the parietal lobe and its reciprocal connections to frontal and cerebellar cortices (Maruff, P. H. Wilson, Trebilcock, & Currie, 1999; P. H. Wilson, Maruff, Ives, & Currie, 2001). Indeed, performance of children with DCD on a visually guided mental imagery tasks is similar to that of patients with parietal lobe damage (Sirigu et al., 1996).

A recent functional magnetic resonance imaging (fMRI) study (Kashiwagi, Iwaki, Narumi, Tamai, & Suzuki, 2009) also supports parietal involvement in DCD. Using a visually-guided tracking task, children with DCD showed significantly less activation of the left superior and inferior parietal lobules than the control group. Finally, the parietal cortex appears to be involved in the emotion recognition difficulties of children with DCD, too (Adolphs, Damasio, Tranel, & Damasio, 1996; Cummins, Piek, & Dyck, 2005). To illustrate, Adolphs and colleagues (1996) showed that patients with lesions of right inferior parietal cortex had difficulty recognising negative facial expressions such as sadness and fear.

1.3.6.3 Prefrontal cortex. The planning, execution, and control of motor responses also rely on a fully functioning PFC, which is involved in cognitive and behaviour control, as well as decision-making. Hypoactivity of the attentional network in DCD including the dorsolateral prefrontal cortex (DL-PFC) (Querne et al., 2008) would compromise the initial stages of motor learning and action planning (Brown-Lum & Zwicker, 2015). Further,

reduced prefrontal involvement may contribute to the high comorbidity of attentional problems with DCD (Deng et al., 2014).

Importantly, atypical recruitment of frontal regions in DCD varies according to the stage of learning. To illustrate, children with DCD who had to learn a trail-tracing task showed more activity in the frontal, parietal, and temporal regions than their TD peers. The DCD group also activated twice as many regions as controls (Zwicker et al., 2010). These findings suggest that those with motor coordination difficulties had to direct more effort and recruit more attentional resources when first learning the task (Brown-Lum & Zwicker, 2015). By comparison, after practicing the fine motor trail-tracing task for 3 days, children with DCD showed hypoactivity across a broad network of regions involved in motor learning, including the right DL-PFC, bilateral inferior parietal lobules, and the cerebellum (Zwicker, Missiuna, Harris, & Boyd, 2011). This finding is supported by Querne et al. (2008) and suggests potential disruption of the motor and sensory pathways in DCD, particularly fronto-cerebellar and parieto-cerebellar pathways (Adams, Lust, Wilson, & Steenbergen, 2014; Brown-Lum & Zwicker, 2015).

1.3.6.4 Basal ganglia (striatum). The exact role of basal ganglia in the presentation of DCD symptoms is not fully understood (Groenewegen, 2003). There is some suggestion that difficulties in force control, at least among a subgroup of children with DCD, could be due to an abnormal functioning of basal ganglia (Lundy-Ekman & Ivry, 1991). However, at least one fMRI study failed to find support for this hypothesis (Ferguson et al., 2014). On the other hand, the striatum – a subcortical part of the forebrain essential for motor learning – has been implicated in DCD. Querne and others (2008), for instance, reported reduced path coefficients between the striatum and the parietal cortex in a go/no-go task, and suggested that an altered pattern of connectivity between the striatum and the parietal cortex may undermine inhibition of motor responses in DCD.

1.3.6.5 Summary. The cerebellum, parietal and prefrontal cortices, as well as basal ganglia are among the neural networks that have been implicated in DCD. Some of these neural networks have overlapping functions in that deficits observed in DCD (esp. difficulties with motor control and learning, as well as cognitive control) have been linked to more than one of them. For instance, both the cerebellum and the parietal cortex are involved in the reduced ability of children with DCD to utilise and update internal models of action. Indeed, it is important to synthesise this information in order to provide a unified account of DCD. In the next section, I will discuss attempts to provide such a unified account. More specifically, I will briefly review different types of research in DCD, and focus on contemporary aetiological accounts of DCD, particularly the main neurocognitive accounts.

1.4 Research in DCD

In addition to meta-analyses, research studies in DCD can be broadly grouped into three categories: Descriptive research, which aims to introduce the main characteristics and consequences of the disorder as well as the comorbidities; intervention research, which evaluates the effectiveness of various remediation programs and methods; and aetiological studies, which attempt to unravel the underlying causes of DCD. The focus of this section is on aetiological research, particularly the current hypotheses regarding the underlying neurocognitive mechanisms of DCD.

1.4.1 Descriptive research. There are different varieties of descriptive research in DCD. Some focus on the prevalence and prognosis of the disorder (e.g., Lingam, Hunt, Golding, Jongmans, & Emond, 2009) while others report the effects of DCD on activities of daily living (e.g., Summers, Larkin, & Dewey, 2008), participation in physical activities (e.g., Jarus, Lourie-Gelberg, Engel-Yager, & Bart, 2011), psychosocial adjustment (e.g., Missiuna et al., 2014), and academic success (e.g., Dewey, Kaplan, Crawford, & B. Wilson, 2002). The identification of possible subtypes of DCD, with reference to the comorbidity of disorder and

behavioural performance of children with motor impairments, is another aspect of descriptive studies (e.g., Tsai, P. H. Wilson, & Wu, 2008). Descriptive research has shed light on a range of motor and psychosocial consequences of DCD, the impact of the disorder on daily life, its heterogeneous nature, and the lifelong nature of the condition for a significant proportion of children. This information has guided the choice of interventions, and formed the basis for aetiological inquires into DCD.

1.4.2 Aetiological research. Aetiological accounts of DCD can be chronologically and broadly sorted into developmental delay, neurological impairments, information processing (IP), and the more recent neurocognitive accounts. More specifically, motor impairment in children with coordination problems was initially attributed to a transient and general maturational delay (Geuze & Börger, 1993). However, converging evidence showed that for about half of the children, DCD persists into adolescence and adulthood, particularly in the absence of intervention (Cantell et al., 2003; Losse et al. 1991; Missiuna et al., 2008). The '*developmental delay*' account was later challenged by those suggesting that what is now known as DCD is due to a generalised non-specific deficit at a neurological level (Gillberg, 2003; Kaplan, Crawford, Cantell, Kooistra, & Dewey, 2006; Visser, 2003). An important limitation of the '*neurological impairment*' accounts was that they failed to specify the exact neurobiology of the disorder (Visser, 2003); therefore, limiting their usefulness to inform clinicians. However, the IP (i.e., cognitivist) (P. H. Wilson & McKenzie, 1998), and the cognitive neuroscientific approaches (Castelnau, Albaret, Chaix, & Zanone, 2007; Mandich, Buckolz, & Polatajko, 2002; Sigmundsson, Ingvaldsen, & Whiting, 1997; P. H. Wilson et al., 1997) have now become the prominent views about the causality of DCD. In this section, I review the main hypotheses about the aetiology of DCD – focusing specifically on cognitivist and neurocognitive accounts of DCD.

1.4.2.1 Cognitivist approach. By and large, the main focus of experimental work on causation has been on IP and neurocognitive factors associated with poor motor control and learning in DCD. The aim of the cognitivist approach has been to explain behavioural symptoms of DCD by identifying a set of internal cognitive processes that support those behaviours (P. H. Wilson et al., 2013). The IP approach uses a computer metaphor to explain processing of perceptual and motor information in the brain. More specifically, it assumes that the brain – just like a computer – processes information in a series of stages; and that a disruption of processing within one or more of these stages underlies deficits of motor and cognitive control in DCD (P. H. Wilson & McKenzie, 1998). The IP approach examines the integrity and processing capacity of putative cognitive stages by manipulating processing demands on these stages, and observing changes in the speed, accuracy, or efficiency of action. The cognitivist perspective has linked DCD to deficits in different perceptual and motor processes, namely visuospatial processing, kinaesthetic perception, and cross-modal perception (P. H. Wilson & McKenzie, 1998). However, the IP approach has been criticised for the lack of ecological validity, and not accounting for the interactive, parallel function of neural networks. This limitation could be one of the reasons for the failure of this approach to provide a comprehensive account of DCD and properly inform interventions; and eventually giving way to the neurocognitive approaches (P. H. Wilson, 2005).

1.4.2.2 Neurocognitive accounts of DCD. Cognitive neuroscience (CN) is a multi-disciplinary approach, which integrates brain and behaviour under a single conceptual scheme. The broad aim of CN is to map neural networks that support action, cognition, and emotion (P. H. Wilson e al., 2013). Unlike the IP approach, neurocognitive accounts of DCD are based on the premise that action is the result of multiple interacting networks in the brain. The experimental methods are similar to that of the cognitivist approach in that the processing demands of putative mechanisms are manipulated, and the changes in speed,

accuracy, or efficiency of action are recorded. The main difference, however, is that the neural structures are also inferred through the use of neuroimaging techniques or neuropsychological comparisons. For instance, if the behavioural performance in DCD is similar to that of children and/or adults with specific brain lesions, those structures or networks are also implicated in DCD. The earliest studies that adopted the neurocognitive approach to unravel the aetiology of DCD can be traced back to late-1990s (e.g. Sigmundsson et al., 1997; P. H. Wilson et al., 1997). Since then, a large number of studies using the CN perspective have emerged. The findings of these studies have converged into three neurological accounts of DCD: deficits of predictive control of action, problems with rhythmic coordination and timing, and reduced cognitive control – aka executive function (EF).

1.4.2.2.1 Predictive control. Deficits of predictive control – sometimes referred to as internal modelling deficit (P. H. Wilson, 2005) – is a prominent neurocognitive account of DCD. In this case, predictive control refers to real-time control of motor output as well as the ability to learn and use internal models of actions (i.e., predictive mapping). The basic premise of internal modelling is that the motor system is capable of estimating its own behaviour. Predictive control of action relies on the interaction of two internal models: Motor commands necessary for achieving a desired goal state are generated by the *inverse* model (i.e., the controller). The *forward* model, on the other hand, receives an efference copy of the motor command and predicts the future state of the moving limb(s). If there is a discrepancy between real-time sensory feedback and forward estimates, error signals are generated to correct motor command in-flight (P. H. Wilson et al., 2013; Wolpert, Ghahramani, & Flanagan, 2001). At a neurological level, rapid online correction of motor output is subserved by a functional loop between the parietal cortex and the cerebellum that monitors forward estimates of limb(s) position and sensory feedback, and – in the case of discrepancy between

the two – corrects motor commands online (Blakemore & Sirigu, 2003; Shadmehr & Krakauer, 2008). The maturation of fronto-parietal and parieto-cerebellar loops underlies the control of movement using internal models (P. H. Wilson, 2005).

Deficits of predictive control in DCD have been supported by converging evidence from different lines of research. Some of these findings include the reduced ability of children with DCD to voluntarily control their covert attention (Tsai, Pan, Cherng, Hsu, & Chiu, 2009; P. H. Wilson & Maruff, 1999; P. H. Wilson et al., 1997), as well as their impairments in motor imagery (e.g., mental rotation of limbs) (P. H. Wilson, Maruff, Williams, Lum, & Thomas, 2004) and grip force modulation (Hill & Wing, 1999). More recently, Hyde and P. H. Wilson (2011) reported reduced goal-directed reaching in children with DCD using a double-step reaching paradigm. Neuroimaging data also show hypoactivity of left posterior parietal cortex (PPC) and the somatosensory cortex in DCD (Kashiwagi et al., 2009). Indeed, the internal representation of one's body schema (Ogawa & Inui, 2007), as well as hand control and motor imagery (Gerardin et al., 2000) have been linked to left PPC.

1.4.2.2.2 Motor timing. The second converging neurocognitive account of DCD is that a deficit in the timing of motor responses underlies coordination problems in this cohort. Impairments in rhythmic coordination of movement result from difficulties in inter- and intra-limb coupling and stability (P. H. Wilson et al., 2013). More specifically, the cerebellum, which is involved in the adaptive control of movement in response to contextual changes, regulates the timing of motor output and affects the relationship between agonist and antagonist bursts in muscle recruitment. Impairment at the cortico-cerebellar level (i.e., within the cerebellum and in its reciprocal connections to motor and sensory cortices) appears to disrupt rhythmic coordination of action. Evidence to support this particular account of DCD comes from measures of motor timing such as finger tapping, in which children must maintain a stable finger tapping rhythm first in the presence of some auditory cue and then

after its removal (e.g., De Castro Ferracioli, Hiraga, & Pellegrini, 2014; Piek & Skinner, 1999).

1.4.2.2.3 Executive function (EF). Children with DCD show a wide range of deficits in cognitive control – or EF. EF is indeed an umbrella term (Diamond, 2000; Zelazo & Carlson, 2012), which broadly refers to a set of neurocognitive processes implicated in conscious and effortful control of thought, emotion, and behaviour (*I will discuss the construct of EF and its dimensions in more detail in the next section*). Generally, EF is comprised of response inhibition, WM, and mental-flexibility. A recent meta-analysis of DCD studies (P. H. Wilson et al., 2013) from 1998 to 2011 reports large effect sizes (Cohen's *d*) across a range of standardised and experimental measures of WM (1.07), inhibitory control (1.03), and executive attention (i.e., planning and cognitive flexibility) (1.46) in children with DCD. In a similar vein, difficulties of children with DCD in reading, writing, and calculating have been linked to their deficits of sustained attention, and an inability to filter out distractions (Ferguson et al., 2014). The P. H. Wilson and colleagues (2013) meta-analysis also suggests EF deficits are more severe in DCD than ADHD. The more recent studies – not included in the meta-analysis – support this conclusion (Leonard & Hill, 2015).

1.4.2.3 Summary. Early aetiological accounts of DCD suggest a generalised pattern of delay or impairment as causes of this disorder, and fail to isolate loci of focus for guiding theory and intervention development. Moreover, the basic premise of the IP approach is not in line with contemporary theories regarding the interactive relationship of neural networks. Neurocognitive approach, on the other hand, has used robust experimental and neuroimaging methods, and identified deficits of predictive control, motor timing, and EF as the main aetiological accounts of DCD. Having said that, a comprehensive aetiological account of DCD is yet to emerge. To illustrate, the role of affect in motor functioning of children with DCD is not yet clear. Indeed, the ability to regulate emotional responses during motor activity

– which is rarely conducted in abstract, decontextualised lab-based settings – facilitates motor output. Deficits in this area could disrupt the process of motor learning and control. There is, however, paucity of information regarding this aspect of functioning – which relies on EF (esp. inhibitory control) – in children with DCD. Thus, the investigation of EF in affective contexts is vital for enhancing our understanding of DCD and its associated symptoms in both motor and cognitive domains.

In the next two sections, I first present an overview of the contemporary developmental theories of EF, and explain the distinction between 'cool' and 'hot' EF; then, I describe existing literature on cool EF deficits in DCD, and discuss evidence that suggests DCD may also be linked to reduced EF in hot, affective contexts.

1.5 Current Conceptualisations of EF

Historically, the construct of EF has been derived from the study of PFC lesions (Zelazo & Müller, 2011). The main challenge conceptualising EF using this approach is that EF amounts to a list of abilities that appear to underlie numerous and diverse deficits associated with damage to the PFC, including impairments of inhibitory control, planning, attention, feedback utilisation, cognitive flexibility, and so forth (Müller & Kerns, 2015). More recently, developmental theories of EF have attempted to conceptualise this construct more clearly.

1.5.1 Developmental theories of EF.

1.5.1.1 Inhibition accounts. Early efforts (e.g., Luria, 1966) to explain the development of EF focused on age-related changes in the ability to inhibit prepotent responses. Several contemporary conceptualisations of EF (e.g., Dempster, 1992; Diamond, 2013) view inhibition as an independent process, which underlies the ability to control thoughts, behaviour, and emotion. The main premise here is that deficits of cognitive control are often manifested as perseverative errors, which indicate an immature or inefficient

inhibitory control mechanism. For instance, age-related decline in the capacity of WM has been linked to a reduced inhibitory control among the elderly, which makes them more vulnerable to proactive and retroactive interference (Hedden & Park 2001, Solesio-Jofre et al., 2012) and distraction (Zanto & Gazzaley, 2009). Although inhibition accounts have remained popular (e.g., Kirkham, Cruess, & Diamond, 2003), they are bound by significant limitations. For instance, these accounts fail to specify which situations become challenging for children at different ages. The construct of inhibition itself is very heterogeneous, and researchers need to clarify what type of inhibition (e.g., cognitive inhibition, behavioural inhibition, etc.) they are referring to when discussing the development of EF (Müller & Kerns, 2015). Moreover, this view is too simplistic to account for different phenomena that are linked to the construct of EF – for example, performance on EF tasks with minimal demands on the inhibition mechanism. Finally, the concept of inhibition may explain why a response is withheld, but it fails to elucidate why the correct response is generated (Zelazo & Müller, 2011). Therefore, it seems that inhibition is a necessary but not a sufficient account of EF development (Müller, Dick, Gela, Overton, & Zelazo, 2006).

1.5.1.2 Working memory accounts. The proponents of this approach suggest that changes in the capacity of WM underlie the development of EF (Case, 1985; Morton & Munakata, 2003). More specifically, age-related development of WM capacity manifests itself in differences in the ability to control attention in a goal-directed manner (i.e., executive attention) and inhibit irrelevant, prepotent responses or distractions (Redick, Heitz, & Engle, 2007). Thus, inhibitory control is not viewed as an independent process, but rather a side effect of the development of WM. In short, the WM account, which does not treat EF as a superordinate construct, attempts to derive other component processes of EF from changes in WM capacity (Müller & Kerns, 2015). Similar to the inhibition accounts, this approach is

also considered too simplistic to account for the complexity of EF processes and various behavioural deficits associated with EF impairments (Zelazo & Müller, 2011).

1.5.1.3 Working memory plus inhibition accounts. The first two accounts are examples of 'narrowing' approaches in that the development of EF is attributed to a particular component process of EF (Müller & Kerns, 2015). The third account, however, adopts a 'widening' approach, and considers the development of *both* WM and inhibitory control relevant to the conceptualisation of EF (Diamond, 2002; Roberts & Pennington, 1996). According to this approach, WM and inhibition have an interactive relationship whereby the activation of mental representations relevant to a particular goal-directed task results in an automatic inhibition of irrelevant actions. Given that both processes are subserved by the same pool of executive resources (i.e., DL-PFC), reduced EF could be due to either an increase in WM load, which leaves few resources available for inhibition of prepotent responses, *or* an increase in the significance of alternative, irrelevant choices that put more demands on WM. Even though this account is more complex than the 'narrowing' ones, it is not yet clear whether it can explain the wide range of phenomena linked to EF (Stuss, Eskes, & Foster, 1994).

1.5.1.4 Factor-analytic approaches. The factor-analytic account, which is popular in the area of developmental psychology (Müller & Kerns, 2015), has used confirmatory factor analysis (CFA) to investigate whether EF has a unitary structure or is a multi-dimensional construct with dissociable component processes. The seminal work of Miyake and colleagues (2000), which has since become a template for many EF studies, identified three moderately correlated factors in adults: inhibition of prepotent responses, mental flexibility (set-shifting), and updating and monitoring of mental representations (i.e., WM). The use of CFA in pre-schoolers identified a unitary structure for EF (Fuhs & Day, 2011; Wiebe et al., 2011; Willoughby, Wirth, & Blair, 2012); however, there is also evidence that a two-factor solution,

comprising WM and inhibition, could be a better fit at this age, both for TD (e.g., Miller, Giesbrecht, Müller, McInerney, & Kerns, 2012; Usai, Viterbori, Traverso, & De Franchis, 2014) and atypically developing children (Schoemaker et al., 2012). Studies of school-aged children either support the three-factor model of Miyake and others (e.g., Lehto, Juujärvi, Kooistra, & Pulkkinen, 2003), or suggest a two factor solution, with WM and a combination of inhibition and set-shifting (Huizinga, Dolan, & van der Molen, 2006; Van der Ven, Kroesbergen, Boom, & Leseman, 2013). The three-factor model has also been found in adolescents (e.g. Lee, Bull, & Ho, 2013). Indeed, age-related changes in the structure of EF are in line with the *interactive specialisation* view in that initially undifferentiated neural systems become more specialised in their functions as a result of the interaction between the individual and the environment (Johnson & Munakata, 2005).

1.5.1.5 Computational approaches. The computational account, which has received significant attention in recent years, is based on the premise that the development of WM underlies the development of other executive processes. This approach relies on neural network modelling, which essentially simulates the properties and functions of neural systems in a mathematical form in order to examine the effect of changes in brain processes on cognition and behaviour (Munakata, Chatham, & Snyder, 2013). Morton and Munakata (2002) used the *connectionist model* to explain age-related changes on EF tasks. This neural model includes an input layer, an output layer, and a layer of hidden units. Each layer is also composed of interacting units. Feedforward connections establish the link between the input and the hidden layers, and also between the hidden and the output layers. According to the Hebbian learning rule, the connections between units that are simultaneously active eventually become stronger. Feedforward connections between layers are indeed latent memory traces that reflect the strength of learned associations. Based on this model, age-related increases in the strength of excitatory connections of PFC units improve the ability to

hold an active memory representation and override the activation of latent traces; and, consequently, drive changes in component processes such as inhibitory control. For instance, inhibition of prepotent responses on a colour naming Stroop task depends on the ability to override default responses (i.e., read the word) by holding an active representation of task rules (i.e., identify the ink colour). Thus, inhibition is not an independent process, but the outcome of maintaining mental representations of abstract information in neural networks (Munakata et al., 2011). However, Müller and Kerns (2015) argue that the concept of 'abstract representation' needs to be elaborated more clearly by neural network models.

1.5.1.6 Hierarchical and functional approaches. This account proposes that the development of EF is due to age-related changes in different levels of control, particularly the complexity of strategies used to solve problems. The *cognitive complexity and control theory – revised* (CCC-R; Zelazo, Müller, Frye, & Marcovitch, 2003) suggests that all children formulate plans to solve problems. The plans at different ages vary in terms of the hierarchical complexity of the rules underlying them. This developmental trend in the complexity of rule use corresponds to the development of EF. The pre-school period is characterized by notable increases in the complexity of rule system. For instance, 3-year old children can use a pair of simple rules (e.g., 'if green then here, if blue then there'), but fail to integrate higher-order rules in their decision-making (e.g., 'if sorting by colour, if green then here, if blue then there; but if sorting by shape, if car then here, if boat then there). Bunge and Zelazo (2006) suggested the ability to reflect on simple rules in order to formulate more complex ones is subserved by lateral regions of prefrontal cortex (L-PFC). As children reprocess simpler rule and integrate higher-order ones into their rule system, they activate a hierarchically complex network of PFC regions (Zelazo & Cunningham, 2007). The reprocessing stage fulfils the functions of WM and inhibition in that children keep rules in

mind, and – by formulating complex rules – avoid regressing to simpler, irrelevant rules (Müller & Kerns, 2015).

1.5.1.7 Summary: The current state of affairs. There is now evidence to support all of the aforementioned developmental theories of EF. However, none of these approaches can be considered a comprehensive account of EF. To illustrate, it is not clear whether the development of EF is characterised by qualitative changes (i.e., hierarchical levels of rule complexity), or is simply due to quantitative improvements such as an increase in inhibitory control and enhanced activation of mental representations. Moreover, the processes that promote the development of EF are still not clearly understood. Finally, the impact of factors other than complexity, particularly the degree of affective involvement, on performance on EF tasks needs to be elaborated. I will now turn to a more recent conceptualisation of EF that addresses this latter limitation of developmental theories of EF by making the distinction between 'cool' and 'hot' EF.

1.5.2 Two dimensions of EF: Cool and hot EF. EF has been traditionally assessed using tests of WM, inhibition, and set-shifting (Miyake et al., 2000). These tasks that are often administered in lab settings include emotionally-neutral stimuli, and encompass 'cool', cognitive aspects of EF. Cool EF tasks present scenarios in affectively-neutral contexts that often have little resemblance to everyday problems where there is a need to deal with stimuli that are not decontextualised and abstract, but emotionally and motivationally meaningful (Zelazo & Müller, 2011). For instance, Wisconsin Card Sorting Test (WCST; Grant & Berg, 1948), which presents cards that vary on three dimensions (i.e., shape, colour, and number), requires test-takers to discover the rules for sorting these cards correctly. The task is not associated with any obvious rewards or punishers, and there is little to be gained or lost (Zelazo & Carlson, 2012).

A more recent conceptualisation of cognitive control extends the construct of EF to the higher-order control processes that underlie goal-directed action in emotionally and motivationally significant situations (Zelazo & Müller, 2011). 'Hot' EF is generally elicited by problems that involve delaying gratification, resisting temptation, reappraisal of the motivational significance of a stimulus, or suppression of particular emotions or behaviours. Thus, the tasks assessing hot EF (e.g., delay discounting, or gambling/card tasks) involve some obvious rewards and losses, and often integrate tempting stimuli that must be avoided. To illustrate, success on the Iowa Gambling Task (IGT; Bechara, Damasio, Damasio, & Anderson, 1994), which is one of the most widely used measures of hot EF, relies on one's ability to forgo options that are initially rewarding but lead to an overall loss; and instead opt for those options that are only rewarding in the long run. The study of EF development using more affectively-relevant hot situations may lead to more ecologically valid measures and a proper understanding of decision-making in real-life, where one's actions are often influenced by various motivational and emotional factors.

1.5.2.1 Neural correlates of cool and hot EF. The motivational significance of stimuli or contexts determines the type of EF and the neural systems that are activated. Cool EF is linked to L-PFC, while hot EF is subserved by orbitofrontal cortex (OFC) and other medial regions, collectively referred to as ventromedial prefrontal cortex (VM-PFC) (Happaney, Zelazo, & Stuss, 2004; Zelazo & Müller, 2002). The important difference between VM-PFC and L-PFC is that the former belongs to a fronto-striatal network with extensive connections with amygdala and other limbic system structures. The VM-PFC, therefore, is involved in the regulation of affective and non-affective information, as well as the regulation of actions in motivational contexts (Zelazo & Müller, 2011).

Further support for the construct of hot EF comes from neuroscientific research into the function of OFC, which is involved in the flexible reappraisal of the affective significance

of stimuli (Rolls, 2004). The common feature of various tasks that depend on an intact OFC is that success relies on the ability to modify mental representations of specific stimulus-reward associations. This includes measures of reversal learning, delay discounting, and gambling (Happaney et al., 2004). These tasks that are used as measures of hot EF either require one to reconsider the value of an immediate reward relative to a larger delayed reward (i.e., delay discounting), or reappraise options that are seemingly advantageous at the start, but turn out to be disadvantageous in the long run (i.e., gambling tasks).

Converging evidence from clinical observations and animal studies suggests lesions of DL-PFC and OFC result in different deficits. More specifically, a distinction is made between the lateral syndrome – linked to reduced attention, WM, and planning (Knight & D'Esposito, 2003) – and the orbital syndrome – associated with impulsivity and social disinhibition (Fuster, 2008). Indeed, data from lesion studies show that hot EF is dissociable from cool EF in that deficits of hot EF – operationalised as poor performance on measures of gambling (e.g., Bechara et al., 1994), risky decision making (e.g., Rogers et al., 1999), or delay discounting (e.g., Elliott, Frith, & Dolan, 1997) – can occur in the absence of cool EF deficits, and vice versa. To illustrate, both adult and paediatric patients (Bechara, 2004; Eslinger, Flaherty-Craig, & Benton, 2004) with OFC lesions perform poorly on the IGT, and have problems in their daily lives; however, they perform similarly to otherwise normal individuals on cool EF tasks, such as the WCST.

1.5.2.2 Development and dimensionality of cool and hot EF. In general, EF, particularly WM, emerges early in the development – around 12 months of age (Zelazo & Müller, 2011). EFs develop gradually between infancy and early adulthood (Best & Miller, 2010; Johnson, 2012); however, not all EFs follow the same developmental trajectory. For instance, while WM develops early, inhibitory control is fairly difficult for young children (Diamond, 2013), and cognitive flexibility, which builds on WM and inhibitory control,

emerges much later in the development (Davidson, Amso, Anderson, & Diamond, 2006; Garon, Bryson, & Smith, 2008). EF develops most rapidly during developmental stages when the neural networks involving PFC undergo significant structural and functional changes. This occurs mainly during preschool period (2-5 years), as well as adolescence; however, EF continues to develop well into adulthood (Carlson, Zelazo, & Faja, 2013).

Developmental studies suggest the development of hot EF may lag behind that of cool EF. For example, Hooper, Luciana, Conklin, and Yarger (2004) administered two cool EF (i.e., digit span, and go/no-go) and one hot EF (i.e., the IGT) tasks to a group of children and adolescents aged 9-17 years. Although the results revealed age-related improvements in performance on all three tasks, the biggest age-related improvement on cool EF tasks happened between the two youngest groups, while only the oldest group of participants (14-17 years old) had optimal performance on the hot EF task. Likewise, Prencipe and others (2011) reported that adult-like levels of performance on the IGT, administered to a group of 8-15 year olds, are reached at a later age compared to performance on a cool EF task. There were also weak correlations between hot and cool EF tasks in both studies.

The performance on hot EF tasks is subserved by much of the same basic neural circuitry which also underlies cool EFs; however, the fact that hot EF also involves interference from bottom-up affective and reward processing regions such as the ventral striatum (Prencipe et al., 2011) may explain the developmental lag in hot EF (Hodel, Brumbaugh, Morris, & Thomas, 2015). In other words, the cool, cognitive control networks might not be mature enough to modulate the effects of emotionally-significant stimuli. Apart from this, the involvement of the striatal regions in hot EF also highlights the importance of the integrity of fronto-striatal networks in the development of hot EF.

Additionally, even though lesions studies, neuroscientific research, and behavioural data from developmental studies support the dissociation of hot and cool EF, the development

of these two aspects of EF in children is not yet properly understood. To illustrate, a handful of studies reported that hot and cool EF load onto two distinct, but correlated, factors (e.g., Brock, Rimm-Kaufman, Nathanson, & Grimm, 2009; Carlson, Moses, & Breton, 2002; Davis-Unger & Carlson, 2008; Willoughby, Kupersmidt, Voegler-Lee, & Bryant, 2011), while others (e.g., Allan & Lonigan, 2011; Sulik et al., 2010) failed to identify anything more than a single unitary construct for EF in childhood.

There is some suggestion that the distinction between cool and hot EF starts to emerge around 6 years of age (Zelazo & Carlson, 2012). This view is supported by neurobiological research (e.g., Johnson, 2011) which shows that neural systems that are relatively undifferentiated in infancy and early childhood become more specialised with experience, and serve different functions as part of a developmental process of adaptation. This is also similar to how cool EFs become distinct processes as children age. For instance, WM and cognitive flexibility load onto separate factors and can be differentiated in older children (e.g., Huizinga et al., 2006; Lehto et al., 2003) and adults (Miyake et al., 2000), but not in young children (Wiebe, Espy, & Charack, 2008; Wiebe et al., 2011).

1.5.2.3 Issues with the assessment of cool and hot EF. Although the two dimensions of EF are dissociable in patients with brain lesions, they normally work together as part of a system that facilitates adaptation to contextual changes. Given that VM-PFC and L-PFC are parts of a larger interactive functional system with overlapping neurocognitive networks, it might be impossible to design ‘pure’ measures of cool and hot EF (Hongwanishkul, Happaney, Lee, & Zelazo, 2005). Right ventrolateral PFC, for instance, appears to be involved in adaptive functioning in a wide range of hot and cool contexts (Aron, Robbins, & Poldrack, 2004). Thus, performance on many EF tasks could be due to the combination of cool and hot EF (Hongwanishkul et al., 2005). Zelazo and Müller (2011) even suggest that

simply adding motivationally significant stimuli to a standard rule-use task – generally used to assess cool EF – yields an assessment of hot EF.

Indeed, the ability to contextualise emotionally significant problems, reflect upon them, and rephrase them in abstract terms has proved a useful method for dealing with challenges in affectively-laden contexts (Zelazo & Cunningham, 2007). For instance, reflecting upon the non-arousing qualities of a marshmallow, such as its colour or shape, and thinking of it as a cotton ball or a white cloud enables children to delay gratification for a longer period of time (Mischel, Shoda, & Rodriguez, 1989). Another example for the interaction of cool and hot EF is the involvement of WM in the performance of the IGT. Given that individuals need to keep track of wins and losses associated with each option on the IGT, some studies found a direct relationship between WM capacity and net score on the IGT (e.g., Hinson, Jameson, & Whitney, 2002). In sum, it appears that both hot and cool cognitive processes are integral parts of hot EF tasks; and that differences in cool EF, especially inhibitory control, may contribute to deficits of hot EF (Hodel et al., 2015).

1.5.2.4 Significance of cool and hot EF. Many of the skills deemed necessary for success (e.g., creativity, self-control, discipline, flexibility) as well as the ability to take time to decide what to do next, maintain sustained attention, and perform in novel and challenging contexts rely on a combination of cool and hot EF. EF in childhood is also a significant predictor of various important developmental outcomes (Carlson & Zelazo, 2011). More specifically, cool EFs have been repeatedly linked to school readiness, classroom functioning, and academic achievement in general (e.g., Blair, 2002; Blair & Razza, 2007; Diamond, Barnett, Thomas, & Munro, 2007; Fabes, Martin, Hanish, Madden-Derdich, & Anders, 2003; Gathercole & Pickering, 2000; Kurdek & Sinclair, 2000; Rimm-Kaufmann, Pianta, & Cox, 2000).

Hot EF, on the other hand, is a better predictor of real-world functioning and different developmental outcomes (Brock et al., 2009; Hongwanishkul et al., 2005; Willoughby et al., 2011). To illustrate, the follow-up research to the seminal Stanford Marshmallow Experiment (Mischel, Ebbesen, & Zeiss, 1972) showed that the children who were able to delay gratification at age four had better self-control, concentration, academic performance, interpersonal skills, and frustration tolerance as adolescents (Mischel et al., 1989; Shoda, Mischel, & Peake, 1990). This group of high-delayers were also less likely to use recreational drugs in adulthood (Ayduk et al., 2000), and performed better than the low-delayers in suppressing responses to positive social cues (i.e., happy faces) when they were asked to (Casey et al., 2011). Moreover, better self-control – which substantially overlaps with hot EF – in childhood is linked to higher SES and physical health, and lower likelihood of substance dependence and criminal conviction in adulthood, even after controlling for social class of origin and IQ (Moffitt et al., 2011). These longitudinal studies highlight the significance of hot EF in predicting various behavioural patterns across lifespan. In a similar vein, hot EF deficits have been linked to poor ability to anticipate future consequences of actions, reduced impulse- and self-control, and consequently, poor decision-making in situations with high emotional and/or motivational significance. Reduced hot EF increases the likelihood of disruptive or externalising problem behaviours, and interferes with learning ability and academic achievement (Raver & Zigler, 1997; Rimm-Kaufman et al., 2000; Shonkoff & Phillips, 2000).

1.6 Cool and Hot EF in DCD

1.6.1 Cool EF impairments. There is now converging evidence linking reduced motor coordination to deficits of EF. This is based not only on behavioural data but also recent work into the neurobiological mechanisms of motor and cognitive development in both typically and atypically developing children. Some estimates suggest that about half of the

children diagnosed with DCD also suffer from reduced EF (Sugden, Kirby, & Dunford, 2008; Willcutt, & Pennington, 2000). More specifically, DCD is associated with deficits in different cool EF tasks.

1.6.1.1 Response inhibition. Deficits of response inhibition in DCD disrupt the ability to plan and monitor motor responses (Livesey et al., 2006; Piek et al., 2004; P. H. Wilson et al., 1997), detect and correct errors (Lord & Hulme, 1988; Mandich et al., 2002), and make adjustments based on performance feedback. Difficulties with coordinating movements on tasks that are complex (Piek & Coleman-Carman, 1995), involve cross-modal integration (P. H. Wilson & McKenzie, 1998), a trade-off between speed and accuracy (Vaessen & Kalverboer, 1990) or a time delay (Dwyer & McKenzie, 1994) have been linked to reduced response inhibition in DCD (Piek et al., 2004). According to Mandich and colleagues (2002), there is a direct relationship between the severity of DCD symptoms and deficits of inhibitory control.

1.6.1.2 Working memory. The performance of children with DCD on some measures of WM has been similar to that of same-age TD controls (Piek et al., 2004; Piek, Dyck, Francis, & Conwell, 2007); however, the DCD group took longer to complete the task. This may suggest that motor ability significantly predicts timing measures, but not the capacity of WM. In contrast, increased response times could still be due to the need for additional read-out time and/or short-term memory capacity (e.g., Anson, 1982). Other studies have linked DCD to reduced WM capacity, particularly visuospatial WM (Alloway, Rajendran, & Archibald, 2009; Ru Loh, Piek, & Barrett, 2011). Visuospatial WM is significantly poorer than verbal WM in children with motor coordination problems.

1.6.1.3 Cognitive flexibility. Although some studies report no significant differences between children with DCD and their TD peers in the ability to flexibly switch between different mental sets (e.g., Piek et al., 2007), others (e.g., Michel, Roethlisberger,

Neuenschwander, & Roebers, 2011; Wuang, Su, & Su, 2011) found DCD is associated with reduced mental flexibility.

1.6.2 Hot EF in DCD. What we know about EF deficits in DCD is limited to cool EF tasks. There is paucity of information about the ability of children with DCD to enlist different EFs, particularly inhibitory control, in 'hot', affective contexts. In this section, I provide empirical evidence which lays the foundation for the general hypothesis that deficits of EF in children with DCD extends to 'hot EF'. I review existing literature on a range of DCD symptoms that suggest children with DCD may also suffer from hot EF deficits. More specifically, I emphasise the high prevalence of psychosocial problems in DCD that have also been linked to reduced hot EF. I review neurobiological data on regions that have been implicated in *both* motor impairment and hot EF; and discuss how impairments in cool EF may jeopardise the performance of children with DCD on hot EF tasks. Finally, I highlight the role of preterm birth, as one of the risk factors of DCD, in increasing the risk for hot EF impairments in children with motor coordination problems.

1.6.2.1 Psychosocial difficulties in children with DCD. In addition to deficits of motor control and motor learning, DCD is associated with a range of significant co-occurring secondary emotional and mental health concerns that compromise psychosocial development and further disrupt daily living activities in children with motor coordination problems (Ferguson et al., 2014; Zwicker et al., 2012). Motor and cognitive control deficits in DCD may reduce participation and/or disrupt performance in group activities (e.g., team games and sport), and, consequently, lead to low perceived self-competence and limited social interactions with peers (Cummins et al., 2005). Children with DCD also perceive they have limited social support (Skinner & Piek, 2001). This in turn leads to feelings of frustration and social-isolation, as well as reduced self-worth and self-esteem, which may consequently

exacerbate symptoms of anxiety and depression (Francis & Piek, 2003; Schoemaker & Kalverboer, 1994; Skinner & Piek, 2001; Zwicker et al., 2012).

The psychosocial consequences of DCD worsen as children age (Skinner & Piek, 2001). Indeed, both cross-sectional (Dewey et al., 2002; Green, Baird, & Sugden, 2006; Skinner & Piek, 2001; Tseng, Howe, Chuang, & Hsieh, 2007) and longitudinal (Sigurdsson, van Os, & Fombonne, 2002) studies suggested that internalising disorders (e.g., depression and anxiety), and to a lesser extent externalising problems (Kanioglou, Tsorbatzoudis, & Barkoukis, 2005; Tseng et al., 2007) are common among children with motor coordination problems. To illustrate, motor coordination impairments were significantly related to lower perceived self-worth and anxiety symptoms in the Skinner and Piek (2001) study. Sigurdsson and others (2002) also reported that children considered as 'clumsy' or 'poorly coordinated' at age 7 were three times more likely at ages 11-16 to be rated as 'anxious' by their parents. Moreover, academic achievement and vocational opportunities in children with DCD are further hampered by their problems with time management, planning and organisation (Kirby et al., 2014). The comorbidity with other disorders also increases the risk of psychosocial maladjustment (Eggleston, Hanger, Frampton, & Watkins, 2012; Rasmussen & Gillberg, 2000). To illustrate, the severity of depressive symptoms in children with comorbid DCD and ADHD is about 3 to 5 times higher than that in children with DCD or ADHD only (Missiuna et al., 2014; Piek et al., 2007).

Missiuna and colleagues (2008) suggested a developmental trajectory in DCD in which basic motor concerns in early childhood or self-care, academic, and peer problems in middle childhood are replaced by more serious concerns about emotional and behavioural issues in late childhood and adolescence. Recurrent findings indicate the aforementioned psychosocial problems co-exist with DCD; however, causality has yet to be established (Missiuna & Campbell, 2014). Some of these issues (e.g., reduced self-care, self-regulation,

and planning, as well as high incidence of internalising and externalising behaviours) have also been linked to deficits of hot EF, and more specifically to reduced inhibitory control in emotionally-laden situations. Thus, a postulate is that reduced EF in affective contexts may contribute to psychosocial problems in DCD.

1.6.2.2 Neural networks implicated in both hot EF and motor control. A range of different studies have indicated that the experience and regulation of emotion, as well as the planning of action and execution of motor responses activate common neurological networks (Coombes, Corcos, Pavuluri, & Vaillancourt, 2012; Heimer & Van Hoesen, 2006; Hikosaka, Sesack, Lecourtier, & Shepard, 2008; Mauss, Bunge, & Gross, 2007; Mogenson, Jones, & Yim, 1980). Some of these overlapping neural networks involved in both EF and motor functioning include the frontal lobe, the cerebellum, and the basal ganglia (Abe & Hanakawa, 2009; Pangelinan et al., 2011; Schmahmann & Pandya, 2008). It is possible that impairments within these structures or disrupted connectivity of the neural pathways connecting them contribute to potential deficits of hot EF in DCD. For instance, Deng and others (2014) suggest that the extensive connections between ventral PFC and the emotion circuitry of the brain (Price, 1999) might account for emotional symptoms associated with DCD.

The cerebellum has also been implicated in regulating emotional responses. The high prevalence and co-occurrence of emotional and motor problems in DCD could be linked to common neurodevelopmental causes, such as cerebellar dysfunction (Cairney et al., 2010). The cerebellum is well-known for its role in motor coordination; however, some evidence suggest the cerebellum may also be involved in affective regulation (Schmahmann & Caplan, 2006; Schutter & van Honk, 2009). To illustrate, suppressing the function of medial cerebellum impairs the ability to modify emotional responses, and eventually increases negative mood (Schutter & van Honk, 2009). On the other hand, facilitating the function of medial cerebellum leads to mood elevations (Schutter, van Honk, d'Alfonso, Peper, &

Panksepp, 2003). The neuroanatomical basis of this finding is the reciprocal connections between the cerebellum and both the limbic system structures, such as the amygdala, and the PFC (Schutter & van Honk, 2009). In the case of children with DCD, Marien, Wacknier, de Surgeloose, de Deyn, and Verhoeven (2010) identified a disruption within the cerebello-cerebral network which contributes to the execution of planned actions, visuospatial perception, and regulation of emotion and mood.

In addition to the PFC and the cerebellum, the striatum, which is the major input station of basal ganglia, may also undermine hot EF in DCD. The striatum is involved in planning and modulation of movement pathways, and receives its input from the cerebral cortex (Van den Bercken & Cools, 1982). However, WM and cognitive flexibility also rely on an intact striatum (Voytek & Knight, 2010). Given that cool EFs facilitate performance on hot EF tasks – for instance on the IGT – a maturational lag or disruption of the striatum in children with DCD may undermine hot EF in this cohort.

1.6.2.3 The impact of cool EF deficits. Impairments of cool EF in DCD may also contribute to a reduced EF in motivationally significant contexts. According to van Duijvenvoorde, Jansen, Visser, and Huizinga (2010), intact affective decision-making relies on a mature balance between affective (hot) and cognitive (cool) processes. To illustrate, even though hot EF tasks mainly involve VM-PFC, they may also activate other brain regions such as DL-PFC, the anterior cingulate, the insula, the inferior parietal cortex, the thalamus, and cerebellum (Ernst et al., 2002). Cool EFs, such as WM and response inhibition, are subserved by many of these areas; for instance, the DL-PFC and the anterior cingulate (Braver, Barch, Gray, Molfese, & Snyder, 2001; Owen, 2000). DCD literature is ripe with examples of cool EF deficits in children motor coordination problems (e.g., P. H. Wilson et al., 2013). Therefore, one possibility is that the deficits of inhibitory control, WM, or cognitive flexibility may disrupt performance of the children with DCD on hot EF tasks.

More specifically, positively-valenced stimuli, such as rewards or happy faces, constitute a higher load on inhibitory control networks than the other stimuli that are not motivationally-salient (Lagattuta, Sayfan, & Monsour, 2011). Emotionally-significant stimuli increase the excitability of motor system and the likelihood of approaching stimuli and/or opting for immediate rewards (Chiu, Cools, & Aron, 2014). The ability to inhibit such prepotent responses relies on cognitive control networks that modulate the activation of motor system, and that prevent (irrelevant) motivational cues from biasing the motor system. An effective technique for facilitating delay of gratification is the use of reappraisal strategies – that is to re-evaluate the emotional/motivational significance of stimuli based on previous experiences and learning. Prefrontal areas that are also involved in cognitive and motor control, including DL-PFC (selective attention, working memory), and VL-PFC (inhibition) (Ochsner, Silvers, & Buhle, 2012), subserve the deployment of emotion-regulation strategies such as reappraisal or attention switching (Coombes et al. 2012; Goldin, McRae, Ramel, & Gross, 2008; Kanske, Heissler, Schönfelder, Bongers, & Wessa, 2011; McRae et al., 2009). In the case of DCD, however, reduced coupling of cognitive control and motor planning networks (Zwicker, Missiuna, Harris, & Boyd, 2011) along with reduced WM capacity may undermine the ability to utilise reappraisal techniques. More specifically, cognitive control deficits in DCD may also manifest in a reduced ability to utilise emotion-regulation strategies in order to delay gratification. Problems with inhibitory control, as seen in DCD, may impair the ability to modulate activation of emotion centres in the brain. However, to the best of my knowledge, this hypothesis has not been explored in children with motor coordination problems.

1.6.2.4 The effect of preterm birth. One of the risk factors for DCD is preterm birth, in that between 12.5% to 50% of children born preterm suffer from motor coordination difficulties that are consistent with DCD (Davis, Ford, Anderson, & Doyle, 2007; Goyen &

Lui, 2009; Holsti, Grunau, & Whitfield, 2002). Recent literature also links preterm birth to deficits of hot EF. For example, children born moderate to late preterm (32-36 weeks gestation) performed worse than full-term children at age 4 on a delay discounting task – even after controlling for processing speed and IQ (Hodel et al., 2015). However, the preterm children performed similarly to the full-term group on cool EF tasks. This finding indicates that it may be particularly taxing for preterm children to enlist EF in emotionally significant contexts. Deficits of hot EF in preterm children have been linked to a differential maturation of neural networks underlying affective decision-making (e.g., low volume of OFC) (Ball et al., 2012; Gimenez et al., 2006; Nagy et al., 2009; Thompson et al., 2007). Thus, preterm birth and its associated disruptions in neural development could be another risk factor for deficits of hot EF in DCD.

1.7 Future Directions

Both cool and hot EF have been examined in a number of developmental disorders, particularly ADHD (e.g., cool EF: Pennington & Ozonoff, 1996; hot EF: Geurts, van der Oord, & Crone, 2006); however, little work has been done on the possible link between DCD and deficits of hot EF. Indeed, developmental disorders may vary in their patterns of EF deficits. Autism, for instance, has been mainly linked to reduced hot EF (Zelazo & Müller, 2002; cf. Dawson, Meltzoff, Osterling, & Rinaldi, 1998), while ADHD has been associated with both cool and hot EF deficits (Castellanos, Sonuga-Barke, Milham, & Tannock, 2006; Dinn, Robbins, & Harris, 2001; Toplak, Jain, & Tannock, 2005). To date, only one study investigated what appears to be ‘hot’ dimensions of EF in DCD: Tal Saban and colleagues (2014) reported that young adults with DCD performed worse than their otherwise normal peers on both indices of performance on the Behavior Rating Inventory of Executive Function – Adult version (BRIEF-A; Roth, Isquith, & Gioia, 2005). Tal Saban and others did not use the term ‘hot EF’ in their report; however, the Behavior Regulation Index of the

BRIEF-A includes scores from the four subscales of the test that overlap with the description of hot EF. These are inhibition, shifting, self-monitor, and emotional control. The DCD group performed significantly worse than the controls on each of these subscales. The BRIEF-A, however, is a self-report measure, and is bound by the limitation of this particular method of data collection. More importantly, the adults with DCD were not assessed on common measures of hot EF in this study, and their performance could be due to co-existing attention problems (i.e., ADHD symptoms).

Although hot EF has been shown to predict real-world functioning and developmental outcomes (Brock et al., 2009; Hongwanishkul et al., 2005; Willoughby et al., 2011), two key questions remain in relation to DCD: First, do cold EF deficits in DCD extend to hot EF, and second, does the severity of motor impairment moderate the likely deficits of hot EF in DCD? We now have access to experimentally-validated measures of hot EF such as the child-friendly variant of the IGT, called the Hungry Donkey Task (HDT; Crone & van der Molen, 2004), that can be administered to various paediatric populations. The evidence provided in section 1.6.2 suggests that children with DCD are likely to perform poorly on hot EF tasks like the HDT. Moreover, the question of how EF develops in children with DCD across the lifespan is unknown. This developmental question is important because reduced EF is known to disrupt psychological adjustment and self-regulation skills (Casey et al., 2011). Longitudinal studies have the potential to add knowledge about the causal connections between EF deficits and their downstream effects on performance patterns in DCD. The investigation of a possible interaction between deficits at the level of motor control and EF in emotionally-laden situations has the potential to complement existing neurocognitive accounts of the disorder, and guide theory development and intervention programs in DCD.

1.8 Conclusion

Converging evidence from experimental and neuroimaging studies support a bidirectional relationship between motor and cognitive development. Atypical development in each of these domains has been linked to deficits in the other. DCD is a neurodevelopmental disorder mainly characterised by difficulties with motor control and learning. However, what is intriguing is the presence of a wide range of co-occurring cognitive deficits in this cohort that disrupt *both* motor and cognitive control. Although different lines of research have explored the aetiology of DCD, neurocognitive approaches have provided the most promising account of DCD; and suggested that the range of deficits in DCD tends to be related to several aspects of control: among these, predictive control, the ability to develop stable coordination patterns, and EF have been dominant themes over the past couple of decades. However, none of these approaches have provided a comprehensive account of DCD. In the case of EF, for instance, the exact role of affective involvement on performance of children on EF tasks is not clear.

In a similar vein, developmental theories of EF have failed to take into account the effects of emotionally-significant stimuli on executive processes. However, a more recent conceptualisation of EF distinguishes between executive processes involved in emotionally-neutral contexts ('cool' EF) and those implicated in affectively-laden situations ('hot' EF). This approach to the conceptualisation of EF enables researchers to investigate those aspects of EF in DCD that represent more ecologically-valid manifestations of EF deficits in DCD. In other words, given that motor activities are rarely conducted in abstract, decontextualised lab-based environments, a more valid assessment of EF in DCD would incorporate motivationally-significant stimuli in EF tasks. Although age-appropriate measures of hot EF have been available for quite some time, research in DCD has not yet studied this vital aspect of cognitive control in children with motor impairments. Thus, the broad aim of my research

is to investigate hot EF in school-aged children with DCD, determine whether the disorder can be characterised by deficits in both aspects of EF, and identify the neurocognitive mechanisms involved in possible impairments of hot EF in DCD. In chapter two, I describe the experimentally-validated measures of hot EF that I use in my research, and provide detailed information about the design and data analysis of each of the three studies included in this thesis.

Chapter 2: Methodology

CHAPTER 2: METHODOLOGY

2.1 Overview

Chapter 1 reviews the interaction of motor and cognitive development, describes the characteristics of Developmental Coordination Disorder (DCD), and provides a detailed review of different categories of research in DCD. One of the main neurocognitive accounts of DCD attributes symptoms associated with this disorder to deficits of executive function (EF). However, contemporary conceptualisations of EF – discussed in chapter 1 – show that the investigation of EF in DCD has been limited to 'cool' aspects of cognitive control. Given the significance of 'hot' EF in predicting various developmental outcomes, and facilitating psychosocial adjustment, it was imperative to investigate this aspect of EF in children with DCD.

Chapter 2 aims to describe the underlying methodology used in each study of my thesis to investigate hot EF in DCD. This chapter starts with an outline of the cognitive neuroscience approach, adopted in each study. This is followed by the research questions of Study 1, and a description of the characteristics of participants in this study. I then discuss the logic in choosing the Hungry Donkey Task (HDT) – as a measure of hot EF – in the first study; and review the other tasks used in Study 1. The chapter then turns to the discussion of design and data analysis of the first study. Next, I present details of the methodology of Studies 2 and 3, respectively. I start with the research questions and/or hypotheses of each study, then describe participants, and focus on the underlying reasons for choosing two different versions of emotional go/no-go tasks in the second and third studies. I then discuss data analytic approaches in each of these studies, and show how particular outcome measures were analysed to answer the research questions. Table 2.1 presents a summary of the research questions and hypotheses, materials, main outcome measures, and data analytic approaches of each of the three studies of my thesis. Finally, I present an overview of the general procedure of the three studies.

Table 2.1

Summary of the Methodologies of Studies 1-3

Study	Research Question/Hypothesis	Participants	Materials	Outcome Measures	Design and Data Analysis
Study 1	*Question:* Are EF deficits in DCD confined to 'cool' cognitive control? *Hypothesis:* Children with DCD would perform significantly worse than TD children on the HDT, as operationalized by lower net score and a sensitivity to options with high immediate reward on the HDT.	14 children with DCD, and 22 TD children	Hungry Donkey Task; McCarron Assessment of Neuromuscular Development; One-Back Task	Total net score; Net score per each block of 20 trials; Frequency of selections from each option; Reaction time to each option	Mixed-model ANOVA; repeated measures MANOVA; effect sizes and their 95% confidence interval on all possible comparisons
Study 2	*Question:* Is reduced hot EF in DCD due to a generalized deficit of inhibitory control, or specific sensitivity to rewarding stimuli? *Hypothesis:* The prediction was that children with DCD would show significantly higher commission errors than the controls *only* in response to positively-valenced stimuli.	12 children with DCD, and 28 TD children	Emotional go/no-go task ['cool' version: Neutral facial expressions; 'hot' version: Happy and fearful faces]; McCarron Assessment of Neuromuscular Development	Commission and omission errors; Reaction time to go targets; d'	Mixed-model ANOVA; effect sizes and their 95% confidence interval on all possible comparisons
Study 3	*Question:* Do children with DCD also show difficulty approaching negatively-valenced stimuli? Or are their deficits of cognitive control in affectively-laden contexts specific to rewarding stimuli? *Hypothesis:* The DCD group would show stimulus-specific reduced inhibitory control only in response to positively-valenced stimuli.	12 children with DCD, and 24 TD children	Emotional go/no-go task [two versions: Happy and neutral, sad and neutral]; McCarron Assessment of Neuromuscular Development	Commission and omission errors; Reaction time to go targets; d'	Planned contrasts on specific predictions; effect sizes and their 95% confidence interval on all possible comparisons

2.2 Neurocognitive Approach in the Assessment of Hot EF in DCD

Chapter 1 shows there are different approaches in research on DCD and its associated cognitive and behavioural deficits. These include descriptive research, intervention research, and different approaches in the study of the underlying causes of DCD. The neurocognitive approach has arguably been the dominant one informing experimental work, focusing on the neurocognitive mechanisms underlying action and behaviour (Wilson, 2015). This approach has become a unifying force in that it adopts a set of converging methods to explain motor and cognitive impairments. The cognitive neuroscience account integrates brain and behaviour under a single conceptual scheme, and uses a combination of experimental methods and neuroimaging techniques or neuropsychological comparisons to study the effects of the interaction of neural systems on cognition and action.

Even though different studies have examined the underlying neurocognitive mechanisms of DCD, very few explanatory models have been put forward in this field. The range of deficits shown by children with DCD tends to be related to several aspects of control – among these, predictive control, the ability to develop stable coordination patterns, and EF have been the dominant accounts (Wilson, Ruddock, Smits-Engelsman, Polatajko, & Blank, 2013). Importantly, we see both motor control and cognitive deficits in the work conducted from this perspective over the last 20 years. As mentioned in chapter 1, deficits of EF in DCD have been shown in studies that utilised ‘cool’ measures of EF – for example, tests of visuospatial WM, response inhibition, and mental flexibility in abstract, decontextualised contexts (Wilson et al., 2013). The study of hot EF in DCD, from the perspective of cognitive neuroscience, enhances our understanding of the disorder, and leads to a more comprehensive characterisation of the cognitive, motor and behavioural deficits in DCD.

To address the specific focus of my thesis on hot EF in children with DCD, I used state-of-the-art experimentally-validated measures of hot cognition, and compared their

performance with typically-developing (TD) peers. These tasks included the Hungry Donkey Task (HDT; Crone & van der Molen, 2004), which is based on a delay of gratification paradigm, and two different emotional go/no-go tasks, which measure the ability to inhibit prepotent responses in affectively-laden contexts (Tottenham, Hare, & Casey, 2011). The measures of choice had been validated in experimental work on mainstream cognitive development in children (e.g., Crone & van der Molen, 2007; Garon & Moore 2007) and in disorders of development including attention deficit/hyperactivity disorder (ADHD), Autism Spectrum Disorders (ASD), and those with acquired brain injury (e.g., Ernst et al., 2003; Yerys, Kenworthy, Jankowski, Strang, & Wallace, 2013). The measures are particularly sensitive to the neural systems involved in the delay of gratification, particularly inhibitory control networks that modulate tendencies to respond automatically to compelling/rewarding cues. The studies reported in my thesis combine behavioural data from the performance of children with DCD on the HDT and the two go/no-go tasks with the existing literature on the neural systems implicated in delay of gratification to infer the underlying mechanisms of hot EF deficits in DCD.

2.3 Study One

The aim of the first study was to investigate hot EF in children with DCD, using a developmentally appropriate measure of hot EF. The logic behind the choice of a delay of gratification paradigm to measure hot EF is explained under the 'materials' section for Study 1 (Section 2.3.2). Given the deficits of EF in DCD, and the fact that emotionally-significant stimuli – as in hot EF task – increase task difficulty and require greater cognitive control, it was hypothesised that the DCD group would perform worse than their same-aged TD peers on the hot EF task.

2.3.1 Participants. The DCD group included 14 children (8 boys) aged between 7-12 years (M = 9.0, SD = 1.6), and the control group 22 TD children (7 boys) with the same age

range as the DCD group (M = 9.0, SD = 1.7). G*Power software, version 3.1.3 (Faul, Erdfelder, Buchner, & Lang, 2009), was used to conduct a-priori power analysis. Two separate analyses were conducted here: One using 't tests' and the other using 'F tests' under 'Test Family' option. For t-test, the '*Statistical test*' option was '*Means: Difference between two independent means (two groups)*', and for the F-test it was '*ANOVA: Repeated measures, within-between interaction*'. Both analyses were based on an expectation of moderate to strong effects on EF tasks (e.g., Cohen's $d > 1$, Wilson et al., 2013), $\alpha = .05$, and a recommended statistical power ($1 - \beta$) of .8. For the 'F tests' category, Cohen's d of 1 was transformed to the effect size of .5 for this family of tests. The rest of the options for the 't tests' were as follows: '*Tail(s)*' = one; '*Allocation ratio (N2/N1)*' = 0.50, 0.75, and 1. The use of different allocation ratios in three separate power analyses was based on the expectation of having more children in the TD (control) group. The remaining options for the 'F tests' analysis were: '*Number of groups*' = 2, '*Number of measurements*' = 4 (see the description of the HDT under 2.3.2.1.2 – there are 4 options to choose from in this task), and '*Nonsphericity correction* ε' = 1. The power analyses suggested that 14 participants in each group would satisfy the power requirement of .8.

Both the diagnostic criteria in the latest edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; APA, 2013) and the research guidelines (Blank et al., 2012; Geuze, Jongmans, Schoemaker, & Smits-Engelsman, 2001) were used to screen children for DCD. First, all children in the DCD group scored 85 or below on the Neurodevelopmental Index (NDI) of the McCarron Assessment of Neuromuscular Development (MAND; McCarron, 1997) (M [SD]$_{NDI}$ = 75.18 [8.64], range: 51–85). The level of movement skill in the DCD group, which met criterion A of the DCD diagnosis in DSM-5, is almost the same as the 15th percentile on the MAND (NDI = 84; Tan, Parker, & Larkin, 2001). The use of this cut-off point, recommended by research guidelines (Blank et al., 2012;

Geuze et al. 2001), increases the likelihood of identifying children at risk for DCD. An NDI of at least 100 was the cut-off point for the control group (M [SD]$_{NDI}$ = 108.63 [7.04], range: 100-130). A brief developmental questionnaire completed by parents/guardians of children helped to determine if the other criteria for the DCD diagnosis in DSM-5 were also met; more specifically, whether deficits of motor coordination interfered with activities of daily living and/or academic achievement in the DCD group (Criterion B), *and* whether parents reported that difficulties with motor control and learning in these children were evident by school age (Criterion C). Children with past or current diagnosis of any other developmental (e.g., ADHD, autism), neurological or physical disorders were also excluded from the analysis. The decision to exclude children from analyses, due to other diagnoses, was based on parents' responses to the brief developmental questionnaire. Moreover, given that the participants were recruited from mainstream primary schools, and none were participating in remedial classes, intelligence levels were assumed to be within the normal range (Criterion D).

2.3.2 Materials. The HDT (Crone & van der Molen, 2004) is described below, along with its validity and the reason for choosing this task as the measure of hot EF in Study 1. Also described are the motor screening device (MAND) and measure of working memory (WM) in children, a possible correlate of hot EF.

2.3.2.1 Hot EF tasks. Hot EF is mainly assessed by tasks that are based on the delay of gratification paradigm, in which success depends on the ability to forgo small immediate rewards in order to get larger delayed rewards (Müller & Kerns, 2015). Performance here is subserved by a network of structures including ventromedial prefrontal cortex (VM-PFC), dorsolateral prefrontal cortex (DL-PFC), anterior cingulate, the cerebellum, the insula, and the inferior parietal cortex (Ernst et al., 2002). The HDT is based on one of the most widely used tests of hot EF—the Iowa Gambling Task (IGT), designed by Bechara and colleagues (Bechara, Damasio, Damasio, & Anderson 1994). I describe each in more detail, in turn.

2.3.2.1.1 Iowa Gambling Task (IGT). The IGT is the prototypical hot EF task (Bechara et al., 1994) that simulates uncertainties in real-life decision-making by manipulating reward and punishment using a hidden gain-loss schedule (Cassotti, Aïte, Osmont, Houde, & Borst, 2014). Individuals are given $2000 play money and are asked to increase it – over 100 trials – through selection of cards from four different decks. Two decks (A & B) present high immediate rewards in each trial ($100), but are also associated with occasional high loss. These are called 'disadvantageous' decks because the net result of choosing cards from them is a loss of $250 after every 10 trials. The other two decks (C & D) give lower immediate rewards in each trial ($50), but lower occasional loss; these decks are known as 'advantageous' decks because they lead to an overall gain of $250 after every 10 selections. Individuals need to use feedback from earlier trials in order to perform advantageously on the IGT. Total net score, calculated by subtracting the total number of disadvantageous choices from the advantageous ones, is the most commonly used index of performance on the HDT. I discuss the importance of other outcome measures in the IGT/HDT under the 'design and data analysis' section of Study 1.

The IGT is very sensitive to VM-PFC lesions. Lesion studies show that patients with VM-PFC damage perform poorly on the IGT in that they opt for disadvantageous decks, and show 'myopia for future' (Bechara et al., 1994). A neural dissociation is evident in that patients with lesions of temporal or occipital lobe (Bechara, 2004), or damage to dorsolateral prefrontal cortex (DL-PFC) perform as well as otherwise normal adults on the IGT (Bechara, Tranel, & Damasio, 2000; Fellows, 2004). Some of the other groups in which the IGT has been used to assess hot EF include individuals with ADHD (e.g., Ernst et al., 2003; Toplak, Jain, & Tannock, 2005), schizophrenia (e.g., Kester et al., 2006; Nakamura et al., 2008), and substance abuse problems (e.g., Barry & Petry, 2008; Bechara & Martin, 2004).

2.3.2.1.2 Hungry Donkey Task (HDT). In order to assess children's hot EF in Study 1, I used the HDT (Crone & van der Molen, 2004), which is a computerised child-friendly variant of the IGT. The task is presented as a computer game in which children are asked to help a donkey win as many apples as possible. Changing the IGT into a pro-social game makes the task more meaningful and appropriate for the children and increases their engagement in the task. Apart from this change, the HDT uses the same format and similar schedule of rewards and losses as the IGT. The task ran on a laptop computer using E-Prime™ (Schneider, Eschman, & Zuccolotto, 2002).

The stimulus display included the pictures of four doors and a donkey sitting in front of them. Each door was mapped to a key on the keyboard – from left to right, the doors opened with keys A, S, K, and L. Children were informed of the response keys and that they could start a new trial by pressing the spacebar on the laptop keyboard. Upon pressing one of the keys, the outcome display replaced the stimulus one, and children saw the number of green apples they won. In some trials, green apples were accompanied by red, crossed apples. These red apples represent the number of apples participants lose in each trial. The vertical bar on the right side of the outcome display provided a schematic representation of win/loss in each particular trial. On the outcome display, the total number of apples won across the trials, up until a specific trial, also replaced the picture of the donkey at the bottom of the doors (see Figure 2.1).

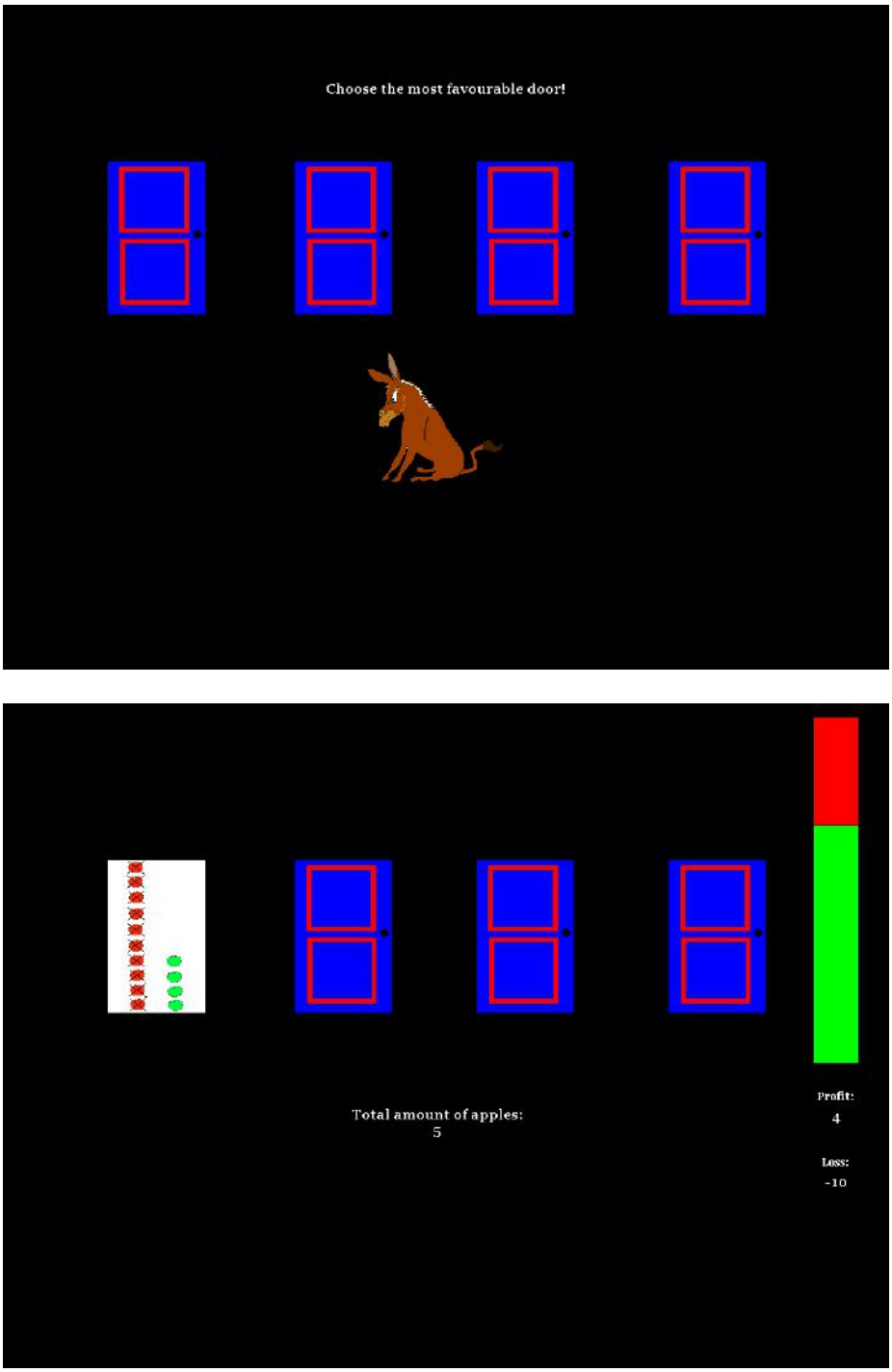

Figure 2.1. Stimulus and outcome displays in the Hungry Donkey Task (HDT)

Just like the IGT, two of the options on the HDT are considered ‘disadvantageous’. These are the two on the left of the stimulus display (doors A & B), which are associated with high immediate reward, but an overall loss. More specifically, both doors give 4 green apples in each trial; however, every 10 trials of options A is associated with five losses of 8, 10, 10,

10, and 12 apples, while the same number of trials for B is linked to the loss of 50 red apples in one trial. The pattern of these losses on *all* the doors is random and unpredictable. In contrast, the other two doors (C & D) give only 2 apples per trial, but are considered 'advantageous' because of their gains in the long run. Door C presents 1, 2, 2, 2, and 3 red apples in every 10 trials of this door, while door D penalises once over the same number of trials with 10 red apples. Therefore, the net result in every 10 trials of a specific door is the loss of 10 apples on doors A and B, and a gain of 10 on doors C and D. The characteristics of the doors (i.e., the win-loss schedule) were not disclosed to children; instead, they needed to infer this using feedback from earlier trials. Children were not told that they had to complete 100 trials; however, they were informed that they needed to select the doors 'many times', and that they could switch between the options as often as they liked.

Behavioural data from both the IGT and the HDT support the validity of these tasks as measures of EF by showing that performance on these tasks parallels the developmental trajectory of EF across lifespan. A number of studies have shown a direct relationship between age and the IGT/HDT performance (e.g., Crone & van der Molen, 2007; Garon & Moore 2007; Hooper, Luciana, Conklin, & Yarger, 2004; Kerr & Zelazo 2004). With increasing age – from childhood to adulthood – individuals are more likely to opt for the advantageous options. For instance, in the case of the HDT, children aged 6-12 years are often tempted by the disadvantageous options, whereas 13-15 year olds gradually learn the benefits of the advantageous ones. Older adolescents (15-18 years) and adults take fewer trials to opt for the advantageous options (Crone & van der Molen, 2004, 2007).

Neuroimaging data from the studies of otherwise normal adults are in line with the findings of lesions studies in that they show an increased activity in the orbitofrontal cortex (OFC) when completing the IGT (Grant, Bonson, Contoreggi, & London, 1999; Windmann et al., 2006). Some have shown a direct link between the activation level of the OFC and

performance on the IGT (e.g., Bolla et al., 2003). In a similar vein, gradual age-related improvement of performance on the HDT has been linked to the protracted development of VM-PFC (Crone & van der Molen, 2004, 2007). Thus, both behavioural and neuroimaging data support the validity of the IGT/HDT as measures of hot EF.

Importantly, the setup of both the IGT and the HDT show these tasks make few demands on motor performance (Crone & van der Molen, 2004). Children only interact with the task by pressing four keys on a laptop keyboard, two of which may be pressed with their left hand (i.e., keys A and S), while the other two may be pressed with the right (i.e., keys K and L). However, children may only use the dominant hand to press any of the keys as there is no speed requirement in these tasks. Therefore, the design of the IGT/HDT minimises the potential confounding effects of motor control deficits – as in children with DCD – on performance on a hot EF task, which enhances construct validity.

2.3.2.2 McCarron Assessment of Neuromuscular Development (MAND). The MAND (McCarron, 1997) is a standardised test of motor skill that can be administered to individuals from 3.5 to 18 years of age. The MAND includes 10 short tests of fine and gross motor skills – five of each – and provides an index of motor functioning (NDI) derived from comparing the sum of scaled scores on the 10 tests to the age-appropriate norms. Unlike the Movement Assessment Battery for Children-2 (MABC-2; Henderson, Sugden, & Barnett, 2007), the MAND uses a common set of items across ages, which is ideal when assessing performance longitudinally or when comparing sub-groups based on age. The NDI has a normal distribution with a mean of 100 and standard deviation of 15. An NDI less than 55 denotes *severe* motor impairment, between 55-70 *moderate* impairment, and between 70-85 *mild* impairment (Piek et al., 2004). Several studies have supported the validity and reliability of the MAND as an assessment tool for motor impairment, and have shown that this test has good specificity and sensitivity. For instance, test-retest reliability of the MAND over a 1-

month period is between .67 and .98 (McCarron, 1997). The content, construct, predictive and concurrent validity of this test have also been established in different populations (e.g., Australia: Hands, Larkin, & Rose, 2013; Tan et al., 2001; USA: McCarron, 1997). To illustrate, there is a high correlation between scores on the MAND and other motor screening tests such as the Movement Assessment Battery for Children (MABC; Henderson & Sugden, 1992) (r = .86) and the Bruininks-Oseretsky Test of Motor Proficiency (Bruininks, 1978) (r = .83) (Tan et al., 2001). The MAND also has a similar factor structure to the 2nd version of Bruininks-Oseretsky Test of Motor Proficiency (Hands et al., 2013). The MAND also has a good level of predictive ability (r = .7) for work-related behaviours (McCarron, 1997).

2.3.2.3 One-Back Task. This task was used as a covariate given the ongoing debate on whether performance on the IGT/HDT relies on WM. Individuals need to keep track of wins and losses associated with each option on these tasks, with some studies finding a covariate effect for WM (e.g., Hinson, Jameson, & Whitney, 2002), but others not (e.g., Crone & van der Molen, 2007). To control for possible covariation between performance on the HDT and WM in children, I used the one-back task from the CogState brief battery to measure WM (Collie, Maruff, Falleti, Silbert, & Darby, 2002). Even though one-back is the simplest of the *n*-back design tasks, children with DCD still perform poorly on the task, enabling researchers to examine the covariation between WM and the HDT performance. The one-back task is a computerised game using playing cards as stimuli, presented on a 12-inch tablet PC. Children must decide whether the playing card presented at the centre of screen is the same as the last card or not. Two keys (e.g., D and K) on a keyboard correspond to ‘yes’ and ‘no’ responses. As soon as children respond, the card goes to the back of the deck, and the next one appears, asking whether it is the same as the one they just saw (See Figure 2.2 for a schematic presentation of the task). The task, which starts with a short practice session, presents 42 cards in the test phase, and terminates after all the trials or after 3 minutes has

elapsed. The correct response in half of the trials is ‘yes’. The higher the accuracy of response, the better is the WM. According to Maruff and colleagues (2009), the One-Back Task has good construct and criterion validity. The task has strong correlations (r > .8) with ‘span’ tasks that require visual scanning and WM.

a) First trial

b) Same card as before

c) Different card to the last one

Figure 2.2. Schematic presentation of the One-Back Task

2.3.3 Procedure. Following the approval of this project at the Human Research Ethics Committee of the Australian Catholic University, the parents and guardians of children in three primary schools across Melbourne received plain language statements about the

whole project, and their consent as well as that of their children were sought before the testing began. Children were only tested during their class time, and in a quiet environment. To control for order effects, the tasks (the HDT, the MAND, and the One-Back Task) were counterbalanced across the participants. The testing took between 20-25 minutes for each child.

2.3.4 Design and data analysis. Both groups of children (DCD and control) completed the 100-trial version of the HDT. The most commonly used dependent measure on this task is the total net score, which is calculated by subtracting the total number of disadvantageous choices from the total advantageous choices [(C+D) – (A+B)]. Higher scores indicate better ability to delay gratification. The other outcome variable, which indicates whether the children utilised feedback from earlier trials in their choice of doors, is the net score per each block of 20 trials. Those who perform well on the task gradually learn to avoid the disadvantageous options, and their highest gains occur in the last blocks of trials (Crone & van der Molen, 2007).

In addition, breaking down the frequency of selections from advantageous (C & D) and disadvantageous (A & B) options into the number of selections per *each* option (A, B, C, D) provides more information about response tendencies of participants. Developmental studies (e.g., Carlson, Zayas, & Guthormsen, 2009; Cassotti, Houde, & Moutier, 2011; Crone & van der Molen, 2004; Huizinga, Crone, & Jansen, 2007) show that children often prefer low-frequency loss options (i.e., B & D) on the HDT, regardless of whether the option is associated with high or low immediate reward. This bias in door selection, which is not observed in patients with VM-PFC lesions, decreases with age, with adults gradually shifting from low-frequency loss options to the advantageous ones (Cassotti et al., 2014). This suggests similar performance of children and VM-PFC patients on the gambling tasks may be due to different factors. To illustrate, impaired use of somatic markers (viz. somatic marker

hypothesis; Damasio, 1994) in VM-PFC patients increases the likelihood of opting for *both* disadvantageous options (A & B); however, in the case of TD children, reduced complexity of rule use – compared to adults – make them consider only the frequency of loss, and opt for options B and D. Adults, on the other hand, consider both the frequency of loss and the amount of loss, and generally opt for option D.

Finally, the speed of responses to each option on the gambling task is another outcome measure, which has only recently been considered in the studies of IGT/HDT. For instance, Smith, Xiao, and Bechara (2012) found that reduced net score on the IGT is associated with faster responses to the disadvantageous options. This finding suggests that high sensitivity to immediate reward affects response latency on the gambling task, and impairs performance. For each child, the RT values that were not within 3 SD of the average RT of that child to a particular door were excluded from the analyses.

In analysing children's performance on the HDT, I used all of the aforementioned metrics. The total net score was compared between the DCD and control groups, and the net score per each block of 20 trials was submitted to a 2 (DCD vs. control) × 5 (blocks 1-5) mixed-model analysis of variance (ANOVA). The source of interaction effects was analysed using tests of simple effects. The simple main effect of block was analysed separately for each group, and indicated whether children utilised feedback from earlier trials. The average frequency of advantageous and disadvantageous choices was submitted to a 2 (groups) × 2 (type of option) mixed-model ANOVA to identify whether children with DCD opted for the disadvantageous options. More importantly, two separate repeated measures MANOVAs – one for each group – were run on the average frequency of selecting the four options on the HDT. This analysis helped identify whether any option was approached below or above the chance levels; therefore, showing whether children had any particular choice strategy. The same analysis was done for each block of 20 trials to investigate the effect of feedback

utilisation in each group. Finally, two separate mixed-model ANOVAs were carried out for the reaction time (RT) data. Within-subjects factor on the first ANOVA had 2 levels (option type: advantageous vs. disadvantageous), and on the second one had 4 (option: A, B, C, D). Effect sizes and the 95% confidence interval (CI) on each outcome measure were computed to temper the use of significance tests and the probability of Type I and II errors.

2.4 Study Two

In Study 1, children with DCD had a significantly lower total net score than the controls on the HDT; they opted for the high immediate reward option that was also associated with infrequent loss (i.e., door B on the HDT), and showed preference for high immediate reward options. What remained unclear was whether deficits of hot EF in DCD – as operationalised by their performance on the HDT – were due to a generalised deficit of inhibitory control *or* to a specific (heightened) sensitivity to emotionally-significant stimuli. The broad goal of Study 2 was to extend our understanding of hot EF deficits in DCD by examining the effects of affectively-relevant stimuli on the response tendencies of children with DCD. To address this aim, I used an emotional go/no-go task to better isolate inhibitory control differences both in the presence and absence of motivationally-significant stimuli (Tottenham et al., 2011). It was hypothesised that children with DCD would show significantly higher sensitivity than the controls to rewarding stimuli, as operationalised by their commission errors in response to positively-valenced targets on the go/no-go task.

2.4.1 Participants. The DCD group included 12 children (6 boys; M_{age} = 9.3, SD_{age} = 1.5 years), and the control group 28 (10 boys; M_{age} = 9.7, SD_{age} = 1.6 years). The age range for both groups was between 7-12 years. The power analysis in this study was based on the moderate-to-large effect sizes observed on key measures of *both* cool EF (Wilson et al., 2013) and hot EF (Study 1: Rahimi-Golkhandan, Piek, Steenbergen, & Wilson, 2014). G*Power 3.1.3 (Faul et al., 2009) was once again used to run the analysis. Given that the

potential participants for Study 2 were students of the same schools who partook in Study 1, the expectation was to recruit more children in the Control group than the DCD. Thus, the '*Allocation ratio (N2/N1)*' used in the a-priori power analysis of Study 2 were 0.50 and 0.75. The rest of the settings in the G*Power (for both 't tests' and 'F tests') were the same as the ones used in Study 1. The minimum sample size for a recommended power of .8 was 12.

Children in the DCD group had an NDI score of 80 or less on the MAND (M [SD]$_{NDI}$ = 77.82 [5.73]). The main reason that Study 1 used a larger cut-off point of 85 ('*Mild motor impairment*') on the MAND was that the use of a stricter criterion would not yield the minimum sample size needed to achieve a statistical power of .8 in that study. The use of an NDI of 80 in Study 2 conforms to the recommendation of Geuze and others (2001) in that it is a stricter criterion than the 15th percentile (score of 84 on the MAND; Tan et al., 2001) – this reduced the likelihood the false positives could bias the results.

The research guidelines (Blank et al., 2012; Geuze et al., 2001) and the DCD diagnostic criteria in DSM-5 (APA, 2013) were used again to identify children with DCD. The level of movement skills in the DCD group was in line with the description of criterion A in the diagnostic criteria: Four of the children in DCD group had *moderate* motor difficulties (55 < NDI < 70), while the rest (N = 8) had *mild* impairments (70 < NDI < 80) (Piek et al., 2004). The exclusion criteria were the same as Study 1. As confirmed by parent questionnaire, motor impairment was of a severity sufficient to disrupt activities of daily living and/or academic achievement in the DCD group (criterion B), and was first evident at an early developmental stage (criterion C). Criterion D is also met for the same reasons as those discussed under section 2.3.1. Similar to the first study, the control group included children with an NDI of at least 100 (M [SD]$_{NDI}$ = 109.3 [6.2]).

2.4.2 Materials.

2.4.2.1 Go/no-go paradigm. The logic behind choosing this particular paradigm and its validity in the assessment of inhibitory control are described in this section. Evidence for age-related changes in the development of *cognitive control* comes from a variety of paradigms that are designed to measure key transitions over the childhood period. Key examples are the 'A not B' task in infants (Diamond, 1985), motor and patience tasks – like walking a line as slowly as possible – in toddlers (Kochanska, Murray, & Harlan, 2000), card sorting tasks – like the simplified version of the Dimensional Change Card Sort (DCCS) – in pre-school children (Munakata & Yerys, 2001), and computerised tasks such as the Stop Signal task in school-aged children (Oosterlaan, Logan, & Sergeant, 1998). In a similar vein, age-appropriate tasks have been used to study *emotion-regulation* over childhood and into adolescence; for example, delay of gratification using marshmallows during the pre-school years (Mischel & Underwood, 1974), emotional Stroop-like tasks in school children (Kindt, Bierman, & Brosschot, 1997), and simulated driving in adolescence – used to measure impulsivity and risk-taking (Steinberg et al., 2008). A major limitation of many of these tasks is the adult ceiling levels (Lagattuta, Sayfan, & Monsour, 2011). Even though EF develops well into adulthood, the relative ease of some of the aforementioned tasks means adults often obtain the maximum possible score on these tasks. Thus, ceiling effects mask the real difference between the cognitive abilities of adults and children by narrowing down the gap in their performance. This issue has prompted many researchers to use different tasks in each age group. However, task specificity can render between-age comparison more problematic and create a methodological divide in the study of EF.

An important advantage of the go/no-go paradigm in the study of hot EF is that this task is capable of bridging child-oriented and adult-oriented methods in research on EF in that it is not affected by floor or ceiling effects (Lagattuta et al., 2011). Tasks designed within this paradigm present a sequence of stimuli (e.g., letters, pictorial cues) and ask participants

to respond to designated ('go') targets and prevent responses to the other ('no-go') targets. The 'go' targets appear frequently – often in about 70-75% of trials – and create a prepotent tendency to respond. Success on the task relies on the ability to inhibit these prepotent responses. The go/no-go paradigm is considered an attractive neuropsychological tool (Tottenham et al., 2011) because it provides a reliable assessment of impulse control ability (Aron, Robbins, & Poldrack, 2004; Dalley, Everitt, & Robbins, 2011) – an important construct underlying hot EF (Eigsti et al., 2006) – across multiple age groups (Casey et al. 1997; Durston et al., 2002, 2006).

2.4.2.1.1 Emotional go/no-go task. This adaptation of the classic go/no-go paradigm (Schulz et al., 2007) was first described in the study of hemispheric specialisation for face recognition (Reynolds & Jeeves, 1978). The task measures impulse control in response to emotionally-significant stimuli, most often different facial expressions (e.g., neutral, happy, sad, fearful, etc.) which are used as both 'go' and 'no-go' cues (Ladouceur et al., 2006). The paradigm assesses *both* response inhibition – as in the traditional go/no-go paradigm – as well as the emotional modulation of this inhibition (Drevets & Raichle, 1998) – i.e., the ability to modulate approach and/or avoidance of emotional stimuli, depending on the task context. For this reason, this type of go/no-go task provides a powerful tool to examine the effects of inhibitory control on heightened sensitivity (of children with DCD) to emotionally-significant stimuli.

Different variations of the emotional go/no-go task have been "validated in neuroimaging studies to dissociate top down prefrontal cognitive systems from subcortical limbic regions for both negative and positive emotions" (Tottenham et al., 2011, p.2). Moderate correlations between the number of commission errors (i.e., failure to resist no-go targets) on the emotional and classic go/no-go tasks suggest that the underlying construct of impulse control is preserved in the emotional go/no-go task (Schulz et al., 2007). This type of

go/no-go task has been used across a wide age range (e.g., Hare et al., 2005) as well as in developmental disorders like ADHD (Köchel, Leutgeb, & Schienle, 2012) and autism (Yerys et al., 2013).

The go/no-go task used in Study 2 comprised two different versions. The 'cool' version used neutral facial expressions of a selection of men and women. For the 'hot' task, the faces of the same individuals were presented with happy and fearful expressions. Converging evidence indicates happy faces are perceived as social rewards (Chakrabarti, Bullmore & Baron-Cohen, 2006; O'Doherty et al., 2003), and can bias behaviour just like primary reinforcers (e.g., marshmallow on a delay of gratification task) (Hare et al., 2005; Somerville, Hare, & Casey, 2011). These positive social cues induce hedonic feelings in individuals and are shown to positively reinforce behaviour (Kohls, Peltzer, Herpertz-Dahlmann, & Konrad, 2009). Since positive faces induce approach behaviour, individuals have more difficulty inhibiting responses to the happy faces than any other facial expression (Schulz et al., 2007). In contrast, fearful faces are exemplars of negative stimuli associated with some unknown danger in the environment; and as a result induce withdrawal (Whalen et al., 2001).

Each of the two versions of the go/no-go task in Study 2 had two different runs. The go stimulus in one run (e.g., happy faces) served as the no-go target in the second. Each run presented 40 pictures (28 'go') in a pseudo-randomised order in order to create a prepotent tendency to respond. The order of presenting the two versions as well as the runs within each version was counterbalanced across the participants. The tasks were run on a 13-inch laptop computer running E-Prime^{TM}. At the start of each task, children received a short written description informing them which gender (on the cool task) or facial expression (hot task) would serve as the go target. They were asked to press the spacebar as fast as they could only in response to the go targets. To make sure children understood the task, they were asked to

take part in 12 practice trials before the onset of each run. Each picture was presented for 500ms, followed by a 1500ms inter-stimulus interval (ISI) during which a white fixation cross appeared at the centre of the screen but which children were still able to respond. The stimuli were grey-scaled pictures of six men and six women – models 6, 8, 11, 14, 15, 16, 27, 28, 36, 39, 44, and 45 from the NimStim^{TM} collection, available at www.macbrain.org. All the images had the same size and luminance. A schematic presentation of the task is provided in Figure 2.3.

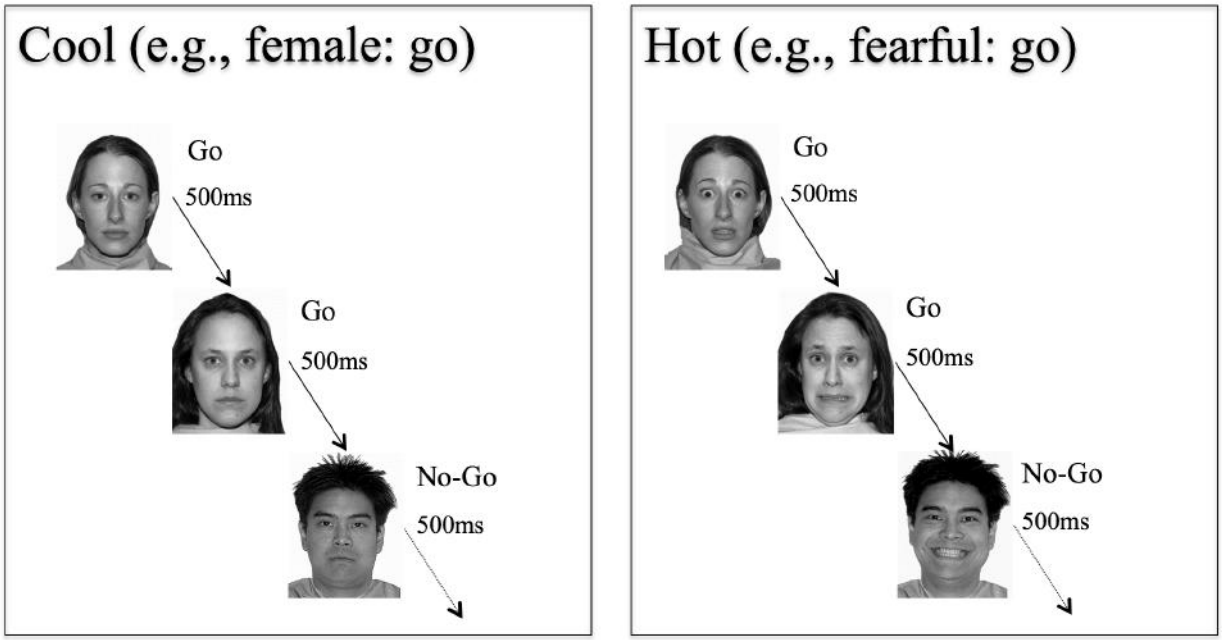

Figure 2.3. Stimulus presentations in emotional go/no-go task of study 2

2.4.2.2 McCarron Assessment of Neuromuscular Development (MAND). (See section 2.3.2.2 for details).

2.4.3 Procedure. A minor amendment to the ethics approval for Study 1 was submitted and accepted to cover the procedures of Study 2 and 3. Similar to Study 1, children took part in the testing during their class time, in an uncluttered, quiet and distraction-free test room. The go/no-go and the MAND were counterbalanced across participants. Testing for this study took approximately 15 minutes for each child.

2.4.4 Design and data analysis. For both the cool and hot go/no-go task, a 2 (block: go vs. no-go) × 2 (stimulus: either male vs. female, or happy vs. fearful) factorial design was used. Children in DCD and control group completed both versions of the go/no-go task. The main outcome measure of interest was failure to withhold responses to no-go targets – defined as commission error. This measure was used to assess whether response inhibition varied as a function of the type of no-go stimulus (i.e., positive, neutral, negative), and whether the response pattern differed between groups; i.e., whether deficits of inhibitory control in the DCD group were generalised or stimulus-specific. The proportion of commission errors (as a function of the total number of no-go trials presented for each stimulus type) was submitted to a 2 (group: DCD and control) × 4 (no-go stimulus: male, female, happy, fearful) mixed-model ANOVA. Each mixed-model ANOVA in Study 2 was followed by tests of simple main effects, as well as the report of effect sizes and the 95% CI around them. An analysis of individual differences was also conducted. Here commission errors to happy faces by each child were presented graphically, for each group.

Omission errors were defined as a failure to approach go targets; this provides a measure of attention in a go/no-go task. This measure indicated whether attentional problems in children with DCD contributed to their performance on the emotional go/no-go task, specifically to go trials. The proportion of omission errors was calculated as a function of the total number of go trials for each stimulus type. Omission errors were also submitted to a 2 × 4 mixed-model ANOVA – with 'group' as between-subjects, and the type of 'go' stimulus as within-subjects factor. Tests of simple main effects were used to isolate the locus of interaction effects.

Response time to go targets is a measure of behavioural execution and an index of the tendency to approach each stimulus. Happy faces are often associated with the fastest responses, while the negative expressions (e.g., fearful, sad, angry) are often approached

more slowly compared to the neutral ones (Hare et al., 2005; Schulz et al., 2007; Tottenham et al., 2011). RT was calculated only for correct 'hits'; for each child, outliers (± 3 SD from the child's average RT to the particular stimulus) were removed from the analysis. RT was also analysed using a 2 (DCD vs. control) × 4 (go stimulus: male, female, happy, fearful) mixed-model ANOVA. This analysis not only provided a measure of approach tendencies to different stimuli, but also showed whether the emotional valence of stimuli was apparent to both groups; this reduces the possibility that performance of any group on the emotional go/no-go task was affected by a failure to recognise stimuli.

Another measure of perceptual sensitivity to different stimuli (i.e., facial expressions) was d'. Low omission errors (i.e., high approach rate) do not necessarily reflect a more developed ability to recognise the stimuli. To illustrate, if a child constantly presses the spacebar in response to both go and no-go faces, omission errors would be minimal; however, commission errors would be maximal. This indicates poor perceptual sensitivity to the target. The d' index provides a highly valid measure of sensitivity by combining the likelihood of correctly detecting go stimuli with the likelihood of commission errors (aka false alarms). The formula used to calculate d' is as follows: d' = z(H) - z(F), where z(H) is the standardised score for correct hits, and z(F) is the standardised score for false alarms. The larger the value of d', the better is the child's ability to discriminate go and no-go stimuli. This variable was also subjected to a 2 × 4 mixed-model ANOVA.

2.5 Study Three

Study 2 showed that reduced inhibitory control of children with DCD on the emotional go/no-go task was stimulus-specific (i.e., only in response to salient positive cues). However, *both* the avoidance of positive stimuli and the ability to approach negatively-valenced stimuli are vital aspects of emotion-regulation which needs to be modulated according to the environmental and/or task context. An important limitation of Study 2 was

the pairing of two affect stimuli (i.e., happy and fearful) together such that sensitivity to one stimulus (e.g., happy face) could prime approach to or avoidance of the other (e.g., fearful face). Study 3 optimised the assessment of sensitivity to reward by modifying *both* the stimuli and the design of the emotional go/no-go task. Emotional stimuli were no longer paired together; instead, they were paired with neutral facial expressions. Fearful faces in Study 2 were also replaced by sad ones, which are more easily discriminable as 'negative' targets by children. Thus, the aim of Study 3 was twofold: First, to investigate whether there would be differences between children with DCD and their TD peers in the ability to approach negatively valenced stimuli. In line with the findings of Study 2, it was predicted that there would be no group differences in approach tendencies toward 'negative' targets. The second aim was to examine sensitivity to reward in DCD and control groups using a more sensitive metric, one controlling for the effects of sensitivity to a specific emotional valence on approach/avoidance of a different emotional expression. It was hypothesised that the DCD group would find it more difficult than the controls to inhibit responses to rewarding stimuli, as operationalised by higher commission errors in response to the happy faces.

2.5.1 Participants. Study 3 included a group of 12 children with DCD (4 boys; M_{age} = 9.8, SD_{age} = 1.4 years), and 24 TD controls (10 boys; M_{age} = 10.2, SD_{age} = 1.6 years). Minimum age was 7 and maximum was 12 years. The participants were students of the three primary schools that also took part in the first two Studies. The power analysis in Study 3 was based on the same assumptions regarding anticipated effect size and the same G*Power settings as Study 2; this yielded an estimate of at least 12 children in each group in order to achieve a statistical power of .8. Similar to the second study, all children with an NDI of 80 or less on the MAND (criterion A) were allocated to the DCD group (M [SD]$_{NDI}$ = 77.6 [7.6]), while the control group included children with an NDI of at least 100 (M [SD]$_{NDI}$ =

109.2 [9.7]). The other criteria for DCD diagnosis (criteria B, C, and D in DSM-5 (APA, 2013)) were also met for the same reasons as those in Studies 1 and 2. The exclusion criteria were the same as those in the first two Studies.

2.5.2 Materials.

2.5.2.1 Emotional go/no-go task. Innovations in the design of the emotional go/no-go task used in Study 3, compared with Study 2, are described in this section. Stimuli were modified to enhance the sensitivity of the paradigm to selective deficits in hot EF. Sad faces replaced fearful ones and all pictures were cropped to exclude hair. Han and others (2012) suggest this modification makes facial expressions more salient. Stimuli were taken from the widely used battery of emotion pictures by Ekman and Friesen (1976), borrowed with permission from the Sackler Institute for Developmental Psychology (*sacklerinstitute.org*). The set of pictures comprised black and white images of 10 adults (five men, five women) on a grey background, each of the same size and luminance. Another important change in the design of the go/no-go task in Study 3 was that emotional faces (i.e., happy and sad) were paired with neutral expressions, not each other. This modification reduces the priming effect of sensitivity to one emotional stimulus on approach to or avoidance of the other, and provides a more sensitive metric of inhibitory control in response to emotionally-significant stimuli.

The task had two blocks (happy and neutral, sad and neutral) each divided into two runs. The ‘go’ target in one run became the ‘no-go’ in the other. Each picture was presented for 500ms, with an ISI of 1500ms, during which a fixation cross replaced the stimuli at the centre of the screen. Therefore, from the onset of each trial, children had 2000ms to make a response. Just like Study 2, children were asked to respond as quickly and as accurately as possible to only the relevant ‘go’ targets in each run by pressing the spacebar on the keyboard of a 13-inch laptop. The number of trials in each run, and the proportion of no-go trials were

the same as those in Study 2 (40 trials: 28 ‘go’, 12 ‘no-go’). Before the onset of each run, 12 practice trials were presented to make sure children understood the specific requirements of that run. Trials were presented in a pseudorandom order. The runs within each block, as well as the blocks themselves were counterbalanced across the participants. The emotional go/no-go task was presented using the E-Prime^{TM} software. Figure 2.4 provides a schematic representation of the task.

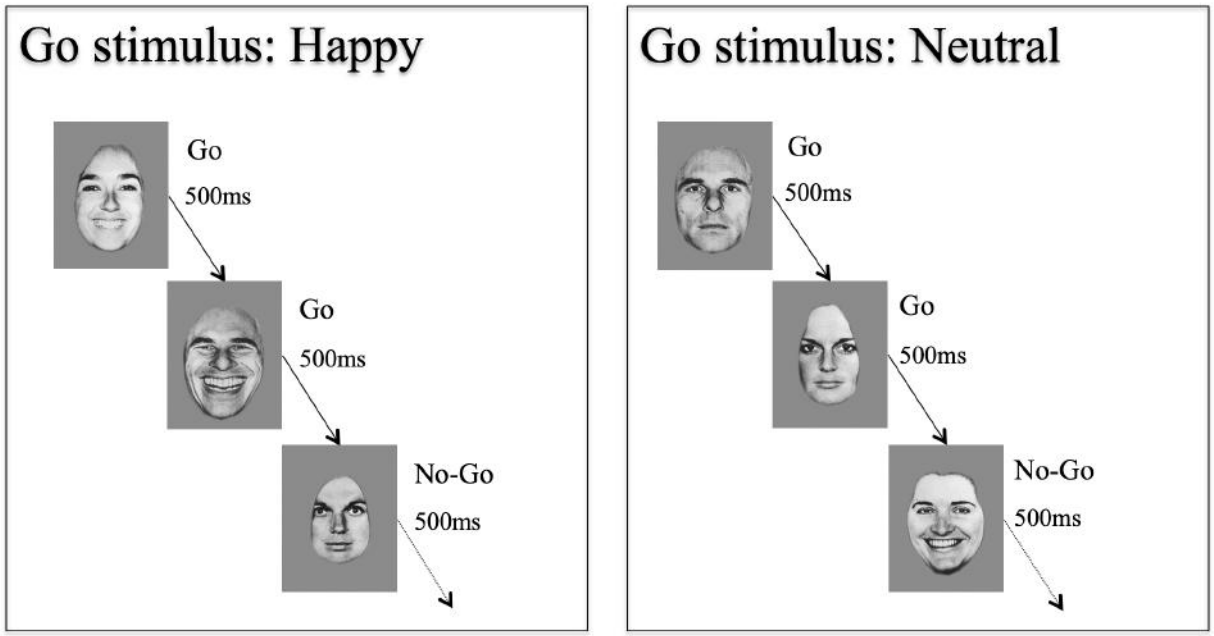

Figure 2.4. Stimulus presentations in emotional go/no-go task of study 3

2.5.2.2 McCarron Assessment of Neuromuscular Development (MAND). Motor skills were assessed using the MAND (McCarron, 1997) – see section 2.3.2.2 for a detailed description of the task.

2.5.3 Procedure. An amendment to the ethics clearance for Study 1 and 2 was processed, enabling Study 3. Children completed the emotional go/no-go task and the MAND in a counterbalanced order. The test environment was as described in section 2.3.3. Testing took 15-20 minutes to complete. At completion, children were presented with a certificate of appreciation for their contribution to the three studies.

2.5.4 Design and data analysis. The go/no-go task had two blocks in that neutral faces were paired with either happy or sad expressions. Each block had two runs; the go stimulus in one run became the no-go target in the second; therefore, each block of the task had a 2 (go vs. no-go) × 2 (stimulus: either happy vs. neutral, or sad vs. neutral) factorial design. A set of parametric planned contrasts was conducted. The decision to use planned contrasts was guided by the specific predictions of Study 3 regarding groups differences in (a) sensitivity to rewarding stimuli, and (b) the ability to approach negatively-valenced stimuli, as well as the pattern of group effects observed in Studies 1 and 2. Planned contrasts are more statistically powerful than mixed-model ANOVA, particularly in small-n designs, and reduce the likelihood of making Type-1 error (Field, 2013), relative to factorial ANOVA. For commission errors, planned comparisons were conducted for happy and sad faces. These analyses addressed the question of whether reduced inhibitory control affects the performance of children with DCD in response to *both* positive and negative no-go cues *or* is specific to rewarding stimuli (i.e., positive facial cues). Individual differences in commission errors were also analysed within each group. Like Study 2, a graphical presentation of the number of commission errors for each child was used to show the heterogeneity of performance in the DCD group. Omission errors and RT to go stimuli were also compared between groups using planned contrasts to better understand approach responses to 'go' stimuli, particularly negatively-valenced ones (hypothesis one). To temper the interpretation of significance tests, effect sizes and their 95% CI were calculated for all group comparisons on the above outcome variables. The d' measure was compared between the groups to determine whether differences were present on the ability to recognise facial expressions.

2.6 Summary

A compelling fact is that children with DCD show a cluster of deficits in both motor control and EF (Wilson et al., 2013). What is particularly intriguing in the case of EF is the

question of whether dysfunction extends to contexts or stimuli that have an emotional reward value—so called 'hot EF'. Empirical work that forms the basis of this thesis was designed within a cognitive neuroscience framework, and adopted a series of cross-sectional, experimental designs. The issue of hot EF in children with and without motor coordination problems (DCD) was examined using two highly validated paradigms: Study 1 used the HDT, which is a child-friendly computerised variant of the IGT. The IGT is considered the prototypical measure of hot EF, and is based on a delay of gratification paradigm. Hot EF is operationalised using net score on the HDT. RT to each option (i.e., advantageous and disadvantageous) on the task, as well as the analysis of choice strategy provided measures of sensitivity to reward. In order to investigate whether reduced hot EF in DCD, as operationalised by the HDT, was due to a generalised deficit of inhibitory control, or specific sensitivity to rewarding stimuli, Study 2 utilised an emotional go/no-go task in which inhibitory control was assessed in response to both neutral ('cool' task) and emotional ('hot' task) faces. Sensitivity to reward was operationalised using commission error on the go/no-go task – in response to positive facial expressions (i.e., happy faces). Omission error reflected the level of attention on the task, while RT to each go stimulus provided a measure of behavioural execution. More importantly, the analysis of d' values was used to determine whether both groups of children were equally adept at recognising facial expressions. Study 3 optimised the assessment of sensitivity to rewarding stimuli by pairing emotional faces with affectively-neutral expressions, and using stimuli that were more easily discriminable by children. Outcome measures were the same as Study 2. The sample size in each study was determined by a power analysis that was based on earlier research showing moderate to strong effects between the DCD and TD groups. Group differences were analysed using a combination of parametric tests (e.g., mixed-model ANOVA, tests of simple main effects, planned contrasts) and effect sizes. Study 1, which was the first attempt to investigate hot EF

in DCD, is presented in the next chapter. The performance of school-aged children with DCD on the HDT was compared to that of their TD peers to determine whether EF deficits in DCD also extended to affectively-laden contexts.

Notes: Chapters 3-5 cover journal publications that emanated from my thesis. Upon the request by the thesis examiners to choose pronouns consistently across all of the chapters, I changed 'we' in chapters 3-5 to 'I'; however, the original versions of these papers appear in appendices I-K.

Chapter 3: Hot executive function in children with developmental coordination disorder: Evidence for heightened sensitivity to immediate reward

CHAPTER 3: STUDY ONE

3.1 Introduction

Developmental Coordination Disorder (DCD) is a neurodevelopmental disorder characterised by problems with motor coordination and skill acquisition that significantly disrupt the daily living activities and/or academic achievement of children (APA, 2013; DSM-V). The clinical diagnosis of DCD is motor coordination significantly lower than expected from chronological age and intellectual ability, and also not due to a pervasive developmental delay or medical conditions such as cerebral palsy or muscular dystrophy. DCD is not a trivial disorder. It affects about 5–6% of school-aged children and is associated with a range of psychosocial and behavioural problems, such as low academic achievement, poor social interaction, poor self-concept, and higher incidence of psychological disorders (Missiuna, Moll, King, Stewart, & McDonald, 2008; Rigoli, Piek, & Kane, 2012). Numerous studies exist of the underlying motor control and learning issues that may explain the disorder, but very few unifying accounts have been put forward. However, a recent quantitative review of the literature by Wilson, Ruddock, Smits-Engelsman, Polatajko, and Blank (2013), compared the performances of children with DCD and typically developing (TD) children on behavioural measures, and revealed that deficits in DCD tend to coalesce around several aspects of control, including predictive control, ability to develop stable coordination patterns, and executive function (EF), the last of these the focus of the present study.

3.1.1 Executive function deficits in DCD. Given that the control of action is supported by a complex and interactive network of neural structures, motor coordination problems are likely to be constrained by not only motor processes but also cognitive and affective ones (Alloway, 2007; Rigoli, Piek, Kane, & Oosterlaan, 2012a; Rigoli, Piek, Kane, & Oosterlaan, 2012b), for example visual attention and EF (Dewey, Kaplan, Crawford, & Wilson, 2002; Green, Baird, & Sugden, 2006), consistent with both modern information

processing theory and recent advances in the cognitive neuroscience of action. For example, in Sergeant's (2000) cognitive-energetic model, EF refers to the complex array of neurocognitive processes that support the conscious and goal-directed control of thought, emotion, and action (Zelazo & Carlson, 2012). In this model, cognitive control and motor behaviour are intertwined. From the perspective of interactive specialisation (Johnson & Munakata, 2005), cognition and action become increasingly coupled over the course of development. Initially distinct systems interact according to the timescales of neural maturation and the moderating effect of experience. From this perspective, there are two prime hypotheses about EF deficits in DCD. The first is that the biological process by which specific neurocognitive and neuromotor systems mature is impaired, and the second is that the emerging neural systems are not stimulated (via appropriate learning experiences) in a way that promotes coupling between specialised sub-systems (e.g., the modulating effect of frontal planning on more primitive limbic structures that resolve stimulus reward).

Studies of clinical populations, such as children with DCD (Piek, Dyck, Francis, & Conwell, 2007) or ADHD (Barkley, 1997; Sergeant, 2000), and non-clinical TD children (Pennequin, Sorel, & Fontaine, 2010) support the interactive relation between cognition and action. We see ample indication that poor coordination, such as in children with DCD, is associated with deficits in EF. Wilson et al. (2013) identified very large effect sizes across core domains of EF, including working memory (WM) (d = 1.07, averaged over visuospatial and verbal), inhibitory control (1.03), and executive attention, which includes deficits in set-shifting and planning (1.46). Most striking was degree of generalised executive dysfunction (operationalised by performance deficits on different EF tasks), in excess of that reported in children with ADHD (Piek, Dyck, et al., 2007).

3.1.2 Are deficits of EF in DCD confined to 'cool' EF? The executive functions studied in children with DCD are generally grouped under the label 'cool' EF. Cool EF is

associated with lateral prefrontal cortex (L-PFC) and is required in situations characterised by abstract, decontextualised stimuli with no affective or motivational component (Zelazo & Carlson, 2012). Recent research has broadened the conceptualisation of EF to include neurocognitive processes that operate in emotion-laden or motivation-laden situations. These contexts are likely to elicit 'hot' affective aspects of EF, mainly associated with ventromedial prefrontal cortex (VM-PFC) (Zelazo & Müller, 2011). Hot EF facilitates flexible reappraisal of emotionally significant stimuli, and assists decision-making when a task involves some affective or motivational component (Prencipe et al., 2011).

Deficits of hot EF are linked to poor ability in anticipating future consequences of actions, poor impulse- and self-control, and, consequently, poor affective decision-making, and even low academic achievement (Casey et al., 2011). Patterns of deficit in different aspects of EF differ across disorders. Autism may be considered primarily as a disorder of hot EF with secondary deficits of cool EF (Zelazo & Müller, 2002; cf. Dawson, Meltzoff, Osterling, & Rinaldi, 1998), while ADHD may be associated with cool EF deficits (Zelazo & Carlson, 2012). However, this variability will also depend on the comorbidity of the disorder (see Dinn, Robbins, & Harris, 2001). There is a lack of evidence as to whether EF problems in DCD are confined to cool EF or extend to both cool and hot EF. Accordingly, current interventions for DCD do not address potential deficits in affective decision-making. Given that different behavioural and psychological problems (e.g., internalising and externalising disorders) that are associated with DCD are also linked to the deficits of hot EF (Casey et al., 2011; Dolan & Lennox, 2013; Must et al., 2006), and considering the important contribution of hot EF to functioning in daily life activities, there is a need to better understand hot EF in DCD in order to inform interventions and consequently improve functioning and quality of life for those diagnosed with DCD.

3.1.3 Assessment of hot EF. Measures of hot EF (e.g., delay discounting or gambling/card tasks) generally involve some obvious rewards and losses (Zelazo & Carlson, 2012), unlike the cool EF tasks where there is little to be gained or lost (e.g., Wisconsin Card Sorting Test; Grant & Berg, 1948). Evidence from neuroscientific research supports the construct of hot EF and shows that hot and cool EF are dissociable. For example, patients with lesions to orbitofrontal cortex perform poorly on the Iowa Gambling Task (IGT; Bechara, Damasio, Damasio, & Anderson, 1994), but not the Wisconsin Card Sorting Test (Bechara, 2004; Eslinger, Flaherty-Craig, & Benton, 2004).

Indeed, the IGT is one of the most widely used hot EF tasks that simulates uncertainties of decision-making in real life by necessitating the weighing of potential rewards and losses. Participants are required to repeatedly select cards from four different card decks. Two decks (A & B) are characterised by high constant gain; however, deck A has a high loss in 50%, and B a very high loss in 10% of the trials (disadvantageous options). The other two (C & D) are characterised by low immediate gain. Deck C has a low loss in 50%, and D a high loss in 10% of the trials (advantageous options). Therefore, amount of constant gain, frequency of loss, and amount of unpredictable loss are the three properties of each deck. Although many healthy adults gradually opt for the advantageous options, patients with lesions of VM-PFC opt for disadvantageous options (Bechara et al., 1994). Given the protracted development of PFC, the failure to anticipate future consequences of actions is also observed in children and adolescents (Smith, Xiao, & Bechara, 2012).

An age-appropriate analogue of the IGT for children and adolescents is the Hungry Donkey Task (HDT; Crone & van der Molen, 2004), in which participants help a donkey win as many apples as possible. Older children and adolescents up to age 15 often opt for options with low frequency loss (Huizinga, Crone, & Jansen, 2007; van Duijvenvoorde, Jansen, Visser, & Huizinga, 2010). Children younger than 12, however, either choose randomly or

prefer options with high immediate reward (A & B). These patterns of performance differentiate hot EF abilities not only in TD children, but also in children with developmental disorders (Geurts, van der Oord, & Crone, 2006). Moreover, WM has also been linked to IGT/HDT performance in some studies (Hinson, Jameson, & Whitney, 2002); therefore, investigation of hot EF in children with and without DCD needs to control for WM.

3.1.4 Deficits of hot EF in children with DCD. Inhibitory control, which is disrupted in children with DCD, is a significant predictor of affective decision-making (van Duijvenvoorde, Jansen, Bredman, & Huizinga, 2012). A deficit of response inhibition can exacerbate the consequences of hot EF deficits, reflected in poor self-control (Casey et al., 2011) and self-regulation (Zelazo & Carlson, 2012). Indeed, children with DCD are at risk for a range of emotional and behavioural problems associated with poor impulse control (Dewey et al., 2002) such as aggression (Tseng, Howe, Chuang, & Hsieh, 2007), conduct problems generally (Kanioglou, Tsorbatzoudis, & Barkoukis, 2005), and poor (motor) self-regulation (Sangster Jokic & Whitebread, 2011).

Moreover, the involvement of particular brain structures in both motor coordination and hot EF may underlie possible deficits of hot EF in DCD. The high incidence of internalising disorders and emotional problems in DCD may be attributed to common neurodevelopmental causes, such as cerebellar dysfunction (Cairney, Veldhuizen, & Szatmari, 2010). Cerebellar activity does not only facilitate motor coordination, but also appears to be involved in the regulation of emotion and mood (Schmahmann & Caplan, 2006; Schutter & van Honk, 2009) and is linked to better performance on the IGT (Ernst et al., 2002). Reciprocal connections between the cerebellum and both the limbic system structures, such as the amygdala, and the prefrontal areas of the cerebral cortex provide some neuroanatomical evidence for the involvement of the cerebellum in the control of mood and emotion (Schutter & van Honk, 2009). Therefore, the high incidence of social and emotional

problems, along with the involvement of the cerebellum in emotion regulation, suggest that children with DCD might show deficits of hot EF compared to their same-age TD peers.

3.1.5 Aim and Hypothesis. The aim of this study was to investigate hot EF in children with and without DCD, using an age-appropriate test of hot EF. I predicted that children with DCD would show an impaired pattern of affective decision-making and perform less well than their same-age TD peers on the HDT.

3.2 Method

3.2.1 Participants. The sample consisted of 14 children (6 girls) in the DCD group (*M* = 9.03, *SD* = 1.59, range 6.7–11.9) and 22 TD children (15 girls) in the control group (*M* = 9.02, *SD* = 1.73, range 6.6–11.7). The DCD group included children who scored at or below 85 on the Neurodevelopmental Index (NDI) of the McCarron Assessment of Neuromuscular Development (MAND; McCarron, 1997). These children are identified as being at risk for DCD. The average NDI in the DCD group was 75.18 (*SD* = 8.64, range: 51–85). The control group consisted of those who had an NDI of 100 or above (*M* = 108.63, *SD* = 7.04, range: 100–130). Given that sometimes up to 50% of children with DCD also meet the diagnostic criteria for ADHD, and links exist between DCD and learning disabilities (Zwicker, Missiuna, Harris, & Boyd, 2012), children with a current or past diagnosis of ADHD, learning, neurological, or any other physical disorder were excluded. All children were recruited from two mainstream primary schools in Australia. Their IQ levels were assumed to be within the normal range, eliminating any need to conduct neurological or medical examination to ensure the criterion C of the DCD diagnosis in DSM-IV-TR (APA, 2000) was met (Geuze, Jongmans, Schoemaker, & Smits-Engelsman, 2001).

3.2.2 Materials.

3.2.2.1 Hungry Donkey Task. The HDT (Crone & van der Molen, 2004) is a computerised, developmentally appropriate analogue of the IGT in which children are asked

to help a hungry donkey win as many apples as possible. Each trial consists of a stimulus and an outcome display. The stimulus display shows a donkey sitting in front of four doors. Each door corresponds to a key on the keyboard (door A: A, door B: S, door C: K, and door D: L). Upon pressing one of the keys, participants see the outcome display showing the number of intact, green apples won and – in some trials – crossed, red apples lost. A vertical bar on the side of the screen presents a graphical index of performance while the amount of overall gain is displayed under the doors. The task includes 100 trials and retains the basic format of the IGT in that two options (A & B) are characterised by high immediate gain (4 apples) but also high loss (disadvantageous), while the other two (C & D) are characterised by low immediate gain (2 apples), but low loss (advantageous). In every 10 trials, door A presents five unpredictable losses of 8, 10, 10, 10, and 12 apples, while door B present one unpredictable loss of 50 apples, leading to an overall loss of 10 apples for each of these doors. Door C, however, leads to five unpredictable losses of 1, 2, 2, 2, and 3 apples in every 10 trials, while door D has one unpredictable loss of 10 apples. Therefore, the net gain on every 10 trials of doors C and D is also equal – 10 apples. Similar to the IGT, net score is the main outcome measure. Participants are not told the properties of each door or the number of trials. They are, however, informed that they have to play many times and that they can switch doors as often as they like.

3.2.2.2 McCarron Assessment of Neuromuscular Development. The MAND (McCarron, 1997) is a standardised test of motor skills comprising five tests of fine motor skills and five of gross motor skills. The sum of the scaled scores on each of these 10 tests is compared to age-appropriate norms (available for individuals aged between 3.5 and 18 years) to determine the NDI score. The test-retest reliability of the MAND (over a 1-month period) ranges between .67 and .98 (McCarron, 1997). The MAND has acceptable criterion validity

and concurrent validity (McCarron, 1997), as well as good specificity and sensitivity (Tan, Parker, & Larkin, 2001).

3.2.2.3 One-Back Task. The One-Back Task is part of the CogState brief battery (Collie, Maruff, Falleti, Silbert, & Darby, 2002) and provides a measure of WM. The participant is instructed to press K (Yes) if the card presented in the centre of the laptop screen is exactly the same as the previous card, and to press D (No) if the card is different. A total of 42 cards are presented, with the task terminating after all the trials or after 3 min has elapsed. The yes or no response is correct in half of the 42 trials. Accuracy of responses is the main outcome measure, with a higher score indicating better WM. The One-Back Task demonstrates good construct validity and criterion validity (Maruff et al., 2009).

3.2.3 Procedure. The individual session was conducted in a quiet environment. It took approximately 10–15 min to administer the MAND, five min to complete the HDT, and five min to finish the One-Back Task. The tasks were administered in varied order across children. Some completed the HDT first and others the One-Back Task or the MAND.

3.2.4 Data analysis. Separate mixed-factorial analysis of variance (ANOVA) was used to compare groups on net score (per block & total) and reaction time (RT) to each response option. Although the control group had significantly better WM than the DCD (M (SD)$_{DCD}$ = 1.04 (0.20), M (SD)Control = 1.24 (0.15), p = .002, d = 1.12; r (NDI & WM) = .56, p < 001), WM was not considered a covariate because there was no significant correlation between net score and WM (r = .13, p = .46). Although males often outperform females on tests of hot EF like the IGT (van den Bos, Homberg, & de Visser, 2013) and the HDT (Crone, Bunge, Latenstein, & van der Molen, 2005), a comparison using gender as a covariate revealed no covariate effects and as gender was not considered further. Moreover, neither gender nor age was significantly correlated with total net score, WM, or MAND NDI.

For a recommended power of .80, and based on the expectation of at least a moderate effect size, I calculated a minimum needed sample size of 14–16 per group.

3.3 Results

3.3.1 Net score. Mean (±SE) net scores for each of the five blocks of 20 trials of the HDT are presented in Figure 3.1, which shows that the control group had higher overall gain on all of the blocks.

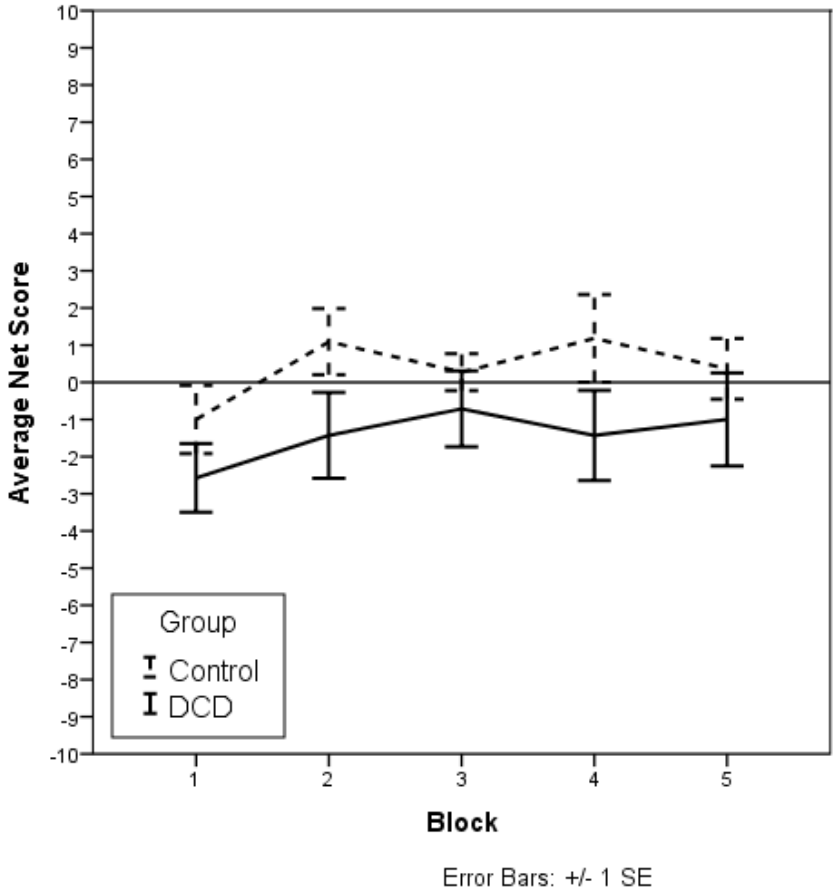

Figure 3.1. Mean net scores of DCD and control groups in each block of the HDT

A 2 x 5 ANOVA – group (DCD vs. control) x block (blocks 1-5) – was conducted to assess whether the pattern of performance across the HDT was different between the two groups. I found no significant interaction between group and block, Wilks' $\Lambda = .96$, F (4, 31) = 0.35, $p = .84$, $\eta^2 = .04$. The simple main effect of block was also not significant ($p > .10$,

.03 < η^2 < .08). However, the total net score was significantly lower for the DCD group, (*M* (*SD*)$_{DCD}$ = -7.14 (11.55), *M* (*SD*)$_{Control}$ = 1.91 (10.41), *p* = .020, *d* = 0.82 [95% CI = 0.12, 1.51]), and there was a significant positive relationship between NDI and total net score (*r* = .42, *p* = .01).

3.3.2 Choice strategy. To investigate whether preference for particular doors/options was responsible for the difference in net scores of DCD and control groups, I first calculated group averages (and *SD*) for the frequency of selecting each type of option: advantageous (low immediate reward): *M* (*SD*)$_{DCD}$ = 46.43 (5.77), *M* (*SD*)$_{Control}$ = 50.95 (5.20); disadvantageous (high immediate reward): *M* (*SD*)$_{DCD}$ = 53.57 (5.77), *M* (*SD*)$_{Control}$ = 49.05 (5.50). The interaction between group and option type (advantageous vs. disadvantageous) was significant, Wilks' Λ = .85, *F* (1, 34) = 5.95, *p* = .020, η^2 = .15. The DCD group had a significantly higher number of selections from the disadvantageous options than the advantageous ones (*p* = .019).

Figure 3.2 shows the average (±SE) number of times each option was approached. Both groups tended to select options with infrequent loss (i.e., B & D) more often; however, compared to the controls, the DCD group showed a higher tendency to approach the disadvantageous option B, and a lower preference for the advantageous option D. In the control group, the number of choices for all doors deviated significantly from the number that would be expected if choice were random, that is 100/4 = 25. The control group preferred options B (*p* = .007) and D (*p* = .006), and avoided options A (*p* < .001) and C (*p* = .007). In contrast, the DCD group only opted for B (*p* = .001). The advantageous option C was approached below the chance level (*p* = .011), while selections from A (*p* = .12) and D (*p* = .32) did not deviate from the chance level.

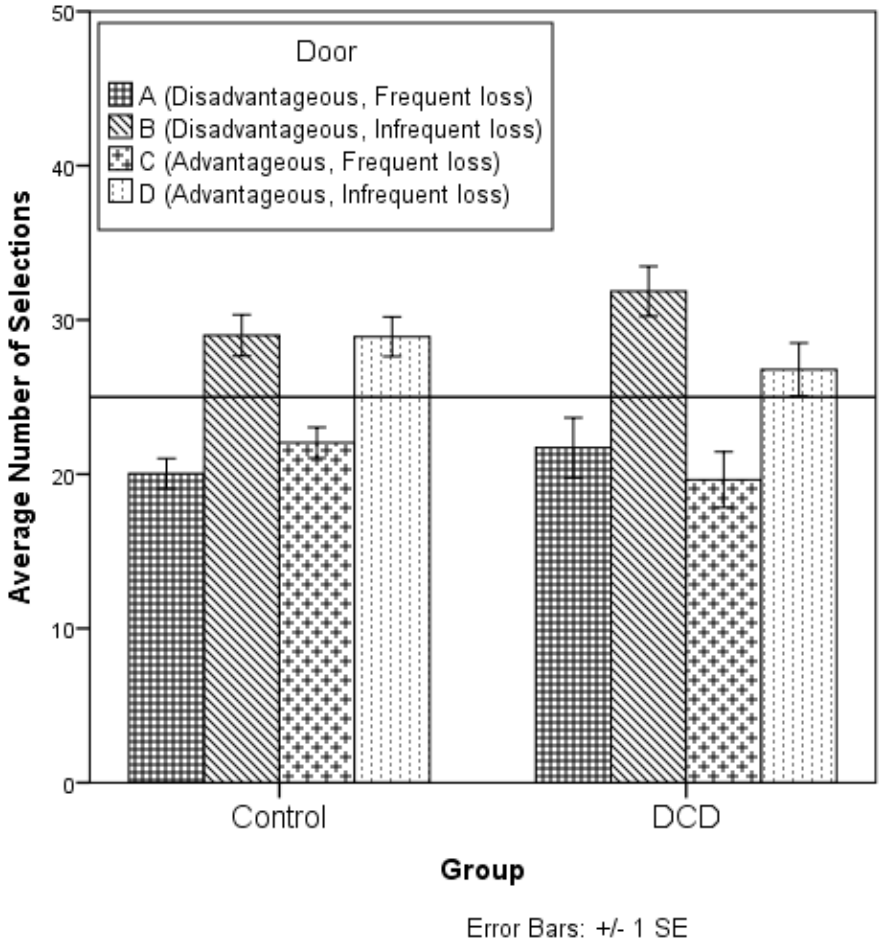

Figure 3.2. Mean number of choices for each option as a function of group. The horizontal line represents chance performance, when each option would be chosen equally (M = 25).

Figure 3.3 depicts the average number of times each option was approached in each block of 20 trials. While the DCD group showed more tendency to approach disadvantageous options (A & B) throughout, the controls almost always approached the advantageous options (C & D) at a higher frequency than did the children with DCD. Moreover, feedback from earlier trials did not affect choice strategy. The DCD group, for example, had their highest number of selections from option B in block 4, which deviated significantly from the chance level (i.e., 20/4 = 5); p = .009). Likewise, while the controls approached option D at a significantly higher than chance level from block 2 onwards (.005 < p < .047), the frequency

of selecting this option was never significantly different from chance level in the DCD group ($p > .10$).

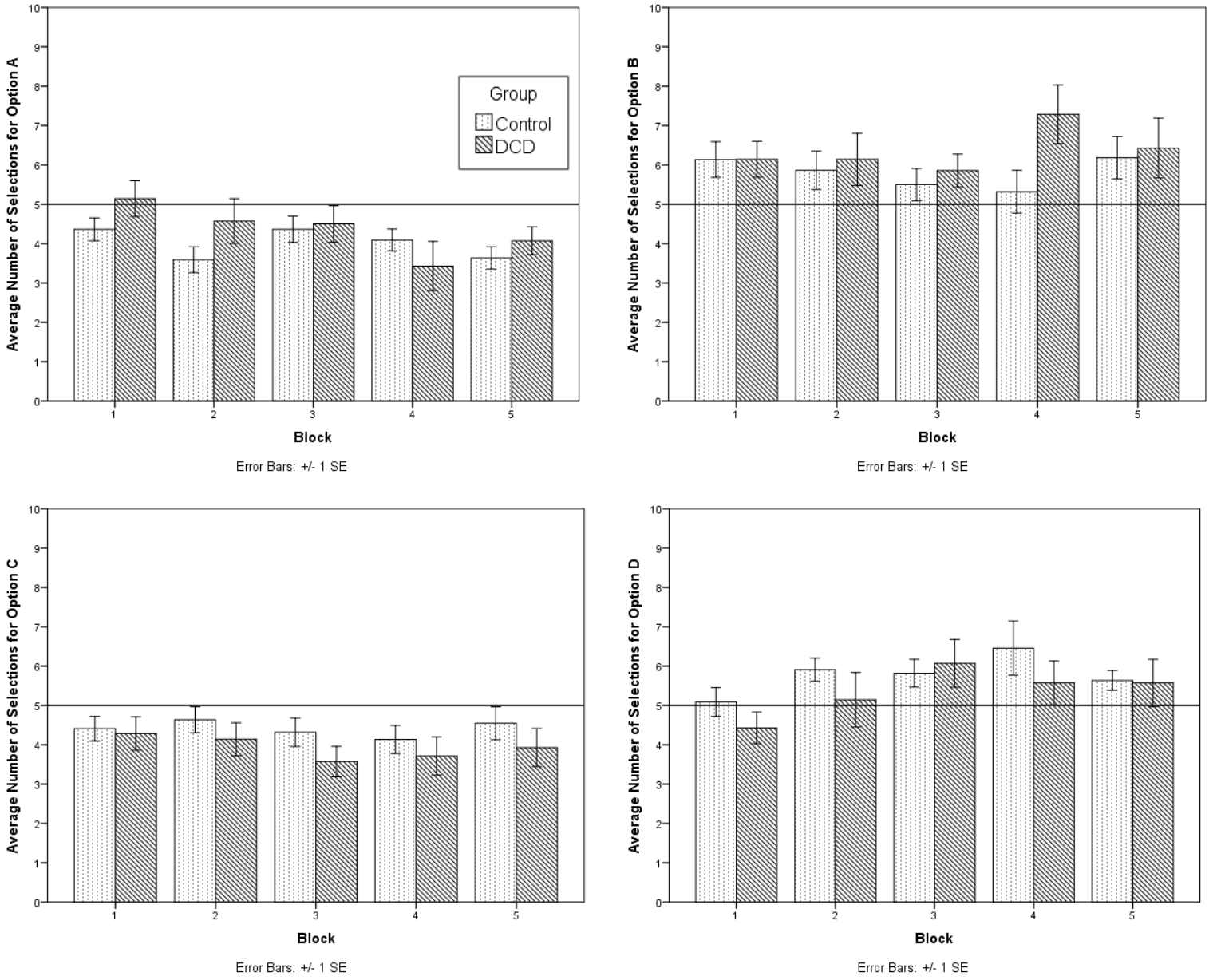

Figure 3.3. Mean number of selections from each option in each block of 20 trials. The horizontal line represents chance performance ($M = 20/4 = 5$).

3.3.3 Reaction Time (RT). I calculated the average RT of DCD and control groups to each door. For each child, the RTs that did not fall within 3*SD* of the child's mean RT to the

particular door were not included in analyses. The average RT of each group (in milliseconds) for each type of door/option was as follows: advantageous (low immediate reward): M $(SD)_{DCD}$ = 1179.69 (368.77), M $(SD)_{Control}$ = 859.59 (281.46); disadvantageous (high immediate reward): M $(SD)_{DCD}$ = 1019.04 (335.49), M $(SD)_{Control}$ = 873.52 (275.13).

There was a significant interaction between group and option type (advantageous vs. disadvantageous), Wilks' Λ = .62, F (1, 29) = 18.17, p < .001, η^2 = .39. Although the RT of control group did not depend on type of option (p = .57, η^2 = .01), the DCD group had significantly faster reactions to disadvantageous, high immediate reward options (p < .001, η^2 = .45). In a similar vein, the DCD group had a significantly longer RT than controls only for approaching advantageous, low immediate reward options (p = .01; door C: p = .022, η^2 = .17; door D: p = .011, η^2 = .20). I observed no changes in this pattern of group differences in RT across the 5 blocks.

The interaction between group and door (A, B, C, & D) was also significant, Wilks' Λ = .59, F (3, 27) = 6.32, p = .002, η^2 = .41. There was a significant difference (p < .001) between the fastest (disadvantageous option B: M = 980ms, SD = 406ms) and the slowest RT (advantageous option C: M = 1233ms, SD = 396ms) in the DCD group. In the control group, however, the fastest RT was to the advantageous option D (M = 792ms, SD = 289ms), significantly different (p = .012) from the slowest RT (option C: M = 927ms, SD = 302ms). Figure 3.4, which presents means (±SE) of each group RT to the four options, also shows that the RT of DCD group to both disadvantageous, high immediate reward options was faster than their RT to the advantageous ones.

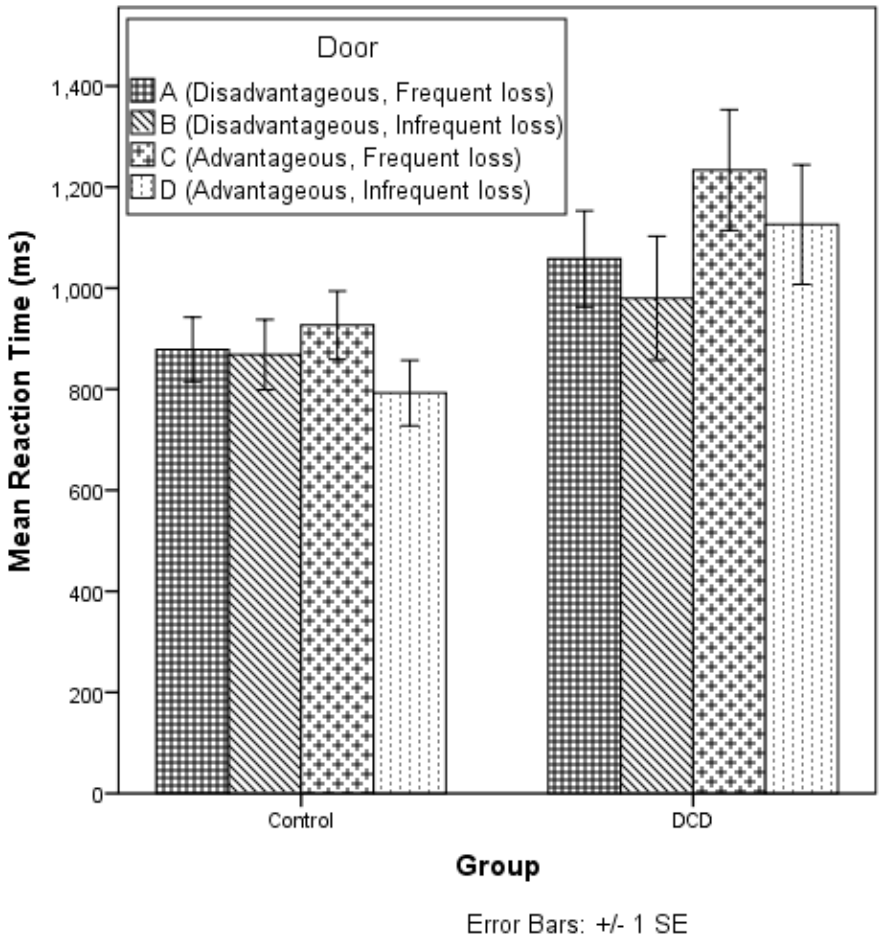

Figure 3.4. Mean reaction time to each option in the DCD and control groups

3.4 Discussion

The results supported the hypothesis that children with DCD would perform more poorly than controls on the HDT. Overall, the DCD group had significantly lower total net scores than controls, across blocks. In addition, children with DCD had a higher tendency to approach the disadvantageous options (high immediate reward) and a lower tendency to approach the advantageous ones (low immediate reward). In particular, the DCD group made less optimal choices by selecting the disadvantageous option B more often, but refraining from choosing the advantageous option D at a rate similar to controls. In contrast, the control group opted for both B and D – the options associated with low frequency loss. I discuss

these group differences by conceptualising the HDT within a proportional reasoning paradigm.

3.4.1 Choice strategy on the HDT. Bechara et al. (1994) argued that given the complexity of weighing all three dimensions of options on the IGT/HDT while making decisions, individuals rely on somatic markers (viz. somatic marker hypothesis; Damasio, 1994), and attempt the task using more intuitive decision processes. Dunn, Dalgleish, and Lawrence (2006), however, suggested that optimal outcomes on the IGT – or in this case the HDT – can be achieved if one simply considers the frequency and amount of loss, and ignores the constant gain. This means that the HDT can be framed as a proportional reasoning task, in which each option is characterised by two dimensions – a dominant (loss frequency) and a subordinate (amount of loss) – which must then be integrated to guide an appropriate response (Huizinga et al., 2007). Individuals progress through a series of developmental levels prior to using the appropriate strategy on a proportional reasoning paradigm (Siegler, 1981). This is shown developmentally by increasingly sophisticated rule use on the IGT/HDT. Young children often switch randomly between the four options, and attempt the task by guessing (‘rule 0’). Hence, the probability of selecting any of the four options is equal. Later, children may consider the dominant dimension (i.e., frequency of loss). This improves the chances of selecting options B and D (‘rule 1’: B & D > 100/4). According to Huizinga et al. (2007), older children (10–12) and adolescents up to age 15 often use ‘rule 1’ to attempt the task. However, the frequency of loss is equal between options B and D. A small group of adolescents, and about a third of adults, will then focus on the subordinate dimension (i.e., amount of loss) and opt for D, which leads to a positive overall outcome (‘rule 2’: D > 100/4). These observations suggest developmental changes in the complexity of rule use not only predict performance in contexts that are relatively devoid of emotional content or valence (“cool contexts”), but also contribute to advantageous decision-making

about motivationally salient stimuli (such as the HDT), and afford better control over thoughts, impulses, and actions.

In this study, the frequency of loss was the dominant dimension for both DCD and control groups. More specifically, the opting of the control group for options B and D is similar to that shown in earlier studies of the IGT/HDT among same-age or older typical children (Huizinga et al., 2007; Prencipe et al., 2011). The DCD group, however, selected only option B above chance level (and not D as well); the disadvantageous option B yields infrequent loss, but also high immediate reward.

Although the loss frequency was the dominant dimension of choice for both groups, the amount of constant gain, instead of the unpredictable loss, became the subordinate dimension of choice for children with DCD. In other words, the choices made by these children were driven not only by a need to avoid frequent loss but also the desire to reap immediate rewards. This shows how the reward component of stimuli in motivationally salient contexts may influence cognitive processes such as proportional reasoning and response inhibition, both of which feed into cool and hot EF. That children with DCD were faster to respond to the two high immediate reward options (A & B) compared with low highlights the impact of emotionally salient stimuli in their affective decision-making, and suggests a heightened sensitivity to immediate reward. The important question here is whether the performance of the DCD group is due to some of their already acknowledged deficits of EF, such as inhibitory control (Wilson et al., 2013), or whether their performance represents an additional/distinct impairment.

3.4.2 High sensitivity to reward. The DCD group opted only for the disadvantageous, high immediate reward option B, and responded significantly faster to the disadvantageous options. For most chronometric tasks, children with DCD are slower than typical children (Piek & Skinner, 1999). We observed the same pattern of difference when

the DCD group approached advantageous, low immediate reward options (C & D); however, it seems the propensity of the DCD group to approach high immediate reward (relative to controls) reduced the gap between the groups' RT to disadvantageous options (A & B). If the DCD group had not shown a preference for high immediate reward, we would have observed similar levels of group difference in RT in response to both types of choices. This differential pattern of performance suggests that children with DCD may have a heightened sensitivity to immediate reward and greater impulsivity. Indeed, disadvantageous performance on the IGT has been linked to faster RT to options A and B (Smith et al., 2012).

Both motor coordination problems and their psychosocial consequences reduce social interaction in children with DCD (Poulsen, Ziviani, Cuskelly, & Smith, 2007) and minimise their involvement in activities such as organised and unorganised play (Bouffard, Watkinson, Thompson, Causgrove Dunn, & Romanow, 1996), which are associated with meaningful reward. One hypothesis is that fewer chances to receive reward in real-life settings may then increase the sensitivity of children with DCD to rewarding stimuli. Reduced feelings of self-worth, social isolation, and poor social support may also contribute to a heightened sensitivity to positive cues. Depression, for instance, has been linked to higher sensitivity to reward in the IGT (Must et al., 2006), and children and adolescents with DCD are at greater risk for depression (Piek, Rigoli, et al., 2007; Rigoli, Piek, & Kane, 2012).

Deficits of hot EF are attributed to either an overactive emotional system or poor inhibitory skills (Bechara & van der Linden, 2005). Indeed, the processes of WM, response inhibition, and even set-shifting are all involved in affective decision-making, which could explain conflicting findings about the factor structure of EF (see Welsh & Peterson, 2014 for a review). These processes would still underpin the management and allocation of attention and memory when the performer must respond to emotionally salient stimuli; weighing the reward component of these stimuli may be the critical function of hot EF.

This conceptualisation of EF in general may offer a better understanding of behaviour in context, compared with the traditional EF framework. To illustrate, immaturities of hot EF (e.g., poor self-control) during childhood or adolescence are explained by the interaction of cognitive control and emotion processing networks whereby the activity of emotion circuitry is not properly controlled by a developing PFC and its associated 'cool' processes, such as inhibitory control (Somerville, Hare, & Casey, 2011). Moreover, the degree to which one must draw on either aspect of EF, and the relative 'heat' of a task are largely determined by the type of stimulus and the behavioural context, moderated by individual differences in the perception of stimuli as motivationally salient (Welsh & Peterson, 2014). For instance, one aspect of hot EF that has been linked to delay of gratification has been the ability to use various cognitive reappraisal strategies to alter how the affective component of a stimulus is perceived or interpreted (e.g., envisioning an attractive marshmallow as a cloud or a cotton ball; Mischel, Shoda, & Rodriguez, 1989).

High sensitivity to rewarding stimuli in DCD and a focus on immediate reward may therefore reflect deficits of inhibitory/impulse control and a reduced ability to delay gratification; associated with this has been low self-control and impulsive behaviours (Riggs, Blair, & Greenberg, 2004). A constellation of cognitive and behavioural problems consistent with this profile appears in DCD: poor inhibitory control, impulsiveness, and a higher incidence of externalising problems. This cluster could be explained, in part, by high sensitivity to immediate reward and poor affective decision-making associated with DCD.

One way to determine the contribution of inhibitory control to poor affective decision-making in DCD is to compare the ability of children with poor coordination to inhibit prepotent responses to neutral and emotionally significant stimuli. This will indicate whether high sensitivity to reward, and consequently deficits of hot EF, is a distinct impairment in DCD or whether it is due to poor inhibition and high impulsivity within this cohort.

3.4.3 Are deficits in hot EF in DCD due to impairments of learning and planning? An alternative hypothesis is that performance of the DCD group on the HDT is due to deficits in cognitive planning (Asonitou, Koutsouki, & Charitou, 2010). Planning involves using feedback from earlier responses (learning) and anticipating the consequences of the same responses (forward thinking and modelling). Although both groups showed impaired performance on the HDT, feedback utilisation and response anticipation seemed to be disrupted more severely in children with DCD.

Throughout the task, the DCD group approached both of the disadvantageous, high immediate reward options (A & B) more often than the controls and showed a reduced ability to integrate feedback – i.e., high loss of apples – in their decisions. Moreover, they had slower responses than controls to only advantageous, low immediate reward options across all 5 blocks of the HDT. These performance patterns suggest impairment of the ability to learn from previous disadvantageous decisions and a deficit of response reversal – an ability closely associated with set-shifting. Indeed, another 'executive' component of hot EF could be the ability to integrate the emotional representation of stimuli based on previous experience, which informs adaptive decisions about emotionally significant stimuli (Welsh & Peterson, 2014). Although DCD has been linked to some learning problems (see Zwicker et al., 2012, for a review), similar patterns of performance (i.e., inability to use and integrate feedback in decisions) has been observed among typical school-aged children (Cassotti, Houde, & Moutier, 2011; Cauffman et al., 2010; Prencipe et al., 2011) who often persist in using a particular choice strategy (e.g., 'rule 1') on either the IGT or the HDT, and fail to integrate feedback into their choices.

Developmental studies (Prencipe et al., 2011; Smith et al., 2012) have shown that with increasing age, but particularly from young adulthood, otherwise normal individuals are more likely to perform response reversal, in that they gradually learn to avoid

disadvantageous decks on the IGT, and select more cards from the advantageous options. However, DCD and its associated learning problems persist into adulthood for a significant proportion of individuals diagnosed with this disorder at childhood (Zwicker et al., 2012). Therefore, if learning problems contribute to the poor performance of the DCD group on tests of hot EF, differences between DCD and control groups may become clearer with age. The assessment of hot EF in young adults with and without motor coordination problems may help us determine the exact role of learning difficulties in affective decision-making.

In addition to using feedback, anticipating the consequences of action (in real time) is another aspect of planning hypothesised to be disrupted in DCD (Williams, Thomas, Maruff, Butson, & Wilson, 2006). The Internal Modelling Deficit (IMD) hypothesis (Wilson et al., 2004) suggests that an impaired ability to internally represent actions and mentally simulate movement leads to problems with predicting the consequences of motor behaviour, organising motor responses, and monitoring/modifying action. This leads to problems with learning and predicting motor behaviour. Indeed, Wilson et al. (2013) argued that predictive control of action is a core deficit in DCD. Although speculative, the impaired ability to generate and/or utilise a forward model of (motor) action could affect performance and decision-making in other domains as well. Thus, poor HDT performance of the DCD group might be due to poor ability in anticipating the outcome of selecting disadvantageous, high immediate reward options. In other words, the prediction deficit may be more generalised than just real-time motor control.

Forward modelling deficits in DCD diminish the ability to modify existing internal models and notably increase the time required to build an adequate model for action (Wilson et al., 2013). If a similar or related mechanism affects performance of children with DCD on a non-motor EF task such as the HDT, they may take significantly longer to reach advantageous levels of decision-making, relative to their peers. However, if high sensitivity

to reward underlies their performance, they might not be able to easily change their decision-making strategy. My study was limited to 100 trials in the HDT. The inclusion of more trials could determine the impact of forward thinking on hot EF deficits of the DCD group.

3.4.4 Implications of hot EF deficits in DCD. In most instances, decision-making depends on a combination of both cool and hot EF. DCD has been linked to cool EF deficits; however, deficits in hot EF and an impaired ability to exercise inhibitory control in motivationally relevant settings, may compromise the control of emotional responses and further disrupt decision-making and behaviour regulation. Generally, hot EF deficits have been linked to poor ability to anticipate future consequences of actions, deficits in impulse- and self-control, and consequently, poor decisions in situations with high emotional or motivational significance (Crone & van der Molen, 2004; Toplak, Jain, & Tannock, 2005; van Duijvenvoorde et al., 2010).

Hot EF in childhood is an important predictor of self-control, academic achievement, interpersonal skills, and frustration tolerance (Casey et al., 2011), highlighting the need for early interventions targeting problems in affective decision-making. Indeed, there is evidence that both cool and hot EF are malleable, particularly during 'sensitive periods' of preschool years and transition to adolescence when there is either a rapid growth or reorganisation of prefrontal systems (Zelazo & Carlson, 2012). Thus, intervention programmes may ameliorate deficits of hot EF in children with DCD.

The current study is the first to show deficits of hot EF in children with DCD. Given the suggested plasticity of hot EF, if future studies corroborate that DCD is indeed linked to heightened sensitivity and lowered resistance to emotionally significant stimuli, interventions should be designed to address deficits of hot EF in children with DCD. Such interventions could be highly effective in improving their functioning, facilitating their learning and social

interactions, as well as reducing their problem behaviours, and consequently improving their quality of life.

Chapter 4: Deficits of Hot Executive Function in Developmental Coordination Disorder: Sensitivity to Positive Social Cues

CHAPTER 4: STUDY TWO

4.1 Introduction

Neurodevelopmental disruptions in one aspect of functioning (e.g., motor) can have far-reaching consequences beyond the primary domain (Leonard & Hill, 2014). In the particular case of poor motor coordination in children (or Developmental Coordination Disorder—DCD), aspects of psychosocial functioning, cognition, and academic performance can also be disrupted (Zwicker, Harris, & Klassen, 2012). In this paper I explore issues of cognition in DCD using an experimental approach, specifically the nature of *executive function* (EF) in these children.

Under DSM-V, DCD is conceptualised as a neurodevelopmental disorder that is marked by motor coordination problems that negatively affect one's daily living activities and/or academic achievement (Zwicker, Missiuna, Harris, & Boyd, 2012), and is generally diagnosed in 5-6% of school-aged children (APA, 2013). The disorder is a distinct diagnostic entity, but often co-occurs with other conditions like Attention Deficit/Hyperactivity Disorder (ADHD) and Specific Language Impairment (SLI) (APA, 2013; DSM-V). Importantly, DCD has been linked to underlying difficulties in not only motor control (Wilson, Ruddock, Smits-Engelsman, Polatajko, & Blank, 2013), but also psycho-social adjustment (e.g., poor self-worth, self-esteem, feelings of loneliness, depression and anxiety, as well as externalising problems) and cognitive control (Cairney, Rigoli, & Piek, 2013; Cummins, Piek, & Dyck, 2005; Schmahmann & Caplan, 2006; Skinner & Piek, 2001; Zwicker, Harris, et al., 2012). More specifically, the recent review of Wilson and colleagues (2013) shows a quite pervasive pattern of dysfunction across (predictive) motor control, all major aspects of EF (i.e., inhibition, working memory (WM) and executive attention—Diamond, 2013), and the self-regulation of movement (e.g., Sangster Jokic & Whitebread, 2011). What remains unclear is the role of affect in the expression of these deficits, or indeed, whether certain types of problems exist only when the child’s emotional investment in the task is heightened.

4.1.1 Cool and hot EF. EF is an umbrella term that refers to a set of neurocognitive processes involved in conscious and effortful control of thought, emotion, and behaviour. Broadly, it can be divided into cool and hot EF. Cool EF is mainly subserved by lateral prefrontal cortex (L-PFC), enlisted when one deals with abstract and decontextualised stimuli. In contrast, hot EF is linked to ventromedial prefrontal cortex (VM-PFC), active in many real-life situations that are characterised by high affective involvement; here, one needs to consider or reappraise the emotional/motivational significance of stimuli and refrain from impulsive actions (Zelazo & Müller, 2011).

EF has been traditionally assessed using 'cool' tasks (e.g., WM, inhibition, and set-shifting), which include decontextualised stimuli (Miyake et al., 2000; Zelazo & Carlson, 2012). There is strong evidence of cool EF deficits in DCD. The recent meta-analysis by Wilson and colleagues (2013) showed very large effect sizes ($d > 1$) on tasks that assess WM, inhibitory control, and executive attention. The stimuli in cool EF tasks, however, often bear little resemblance to everyday situations where one interacts with emotionally and motivationally meaningful stimuli. By comparison, measures of hot EF aim to mimic aspects of real-life decision-making through use of reward and losses, as in delay of gratification and gambling tasks (e.g. Iowa Gambling Task (IGT); Bechara, Damasio, Damasio, & Anderson, 1994).

The studies that compared the performance of typically-developing (TD) children and adolescents on hot and cool EF tasks report that cool EF may mature earlier since adult-like levels of performance are reached later for hot EF. This fits with the view that VM-PFC or its connections might follow a protracted trajectory of development relative to more dorsal aspects of PFC (Hooper, Luciana, Conklin, & Yarger, 2004; Prencipe et al., 2011). However, it has also been suggested that regions associated with hot EF (i.e., orbitofrontal cortex) may develop earlier than those recruited in 'cool' tasks of EF (e.g., DL-PFC) (Orzhekhovskaya,

1981). The fact is that the neurocognitive networks involved in hot and cool EF overlap and form part of a larger interactive functional system. As such, it remains challenging to design 'pure' measures of each of the major two domains of EF (Hongwanishkul, Happaney, Lee, & Zelazo, 2005). Deficits of hot EF, for instance, have been linked to inadequate response inhibition which results in reduced modulation of what is otherwise a relatively mature affective system (Bechara & van der Linden, 2005). Therefore, disruptions in the development of 'cool' regions may also impact deficits of hot EF. Data on children with ADHD – a disorder with high comorbidity for DCD – support this argument: while considered mainly as a disorder of cool EF (Zelazo & Müller, 2011), a number of studies also report deficits of hot EF, particularly the hyperactive subtype (Castellanos, Sonuga-Barke, Milham, & Tannock, 2006; Dinn, Robbins, & Harris, 2001; Toplak, Jain, & Tannock, 2005).

Both aspects of EF are vital determinants of behaviour and adjustment over the lifespan. They predict important developmental outcomes and underlie age-appropriate cognitive and social functioning (Prencipe et al., 2011). EF deficits not only contribute to poor mental and physical health (e.g., higher rates of obesity, overeating, substance abuse) but are linked to reduced academic success and problems in other aspects of adaptive function (e.g., finding and keeping a job, marital satisfaction, public safety, and general quality of life) (Diamond, 2013). While deficits of hot EF have been linked to internalising and externalising problems and poor academic achievement (Casey et al., 2011), little evidence exists about hot EF in DCD.

4.1.2 Preliminary evidence for hot EF deficits in DCD. In an earlier study (Rahimi-Golkhandan, Piek, Steenbergen, & Wilson, in press), I investigated hot EF in children with DCD using the Hungry Donkey Task (HDT; Crone & van der Molen, 2004). The HDT is an age appropriate variant of the IGT in which children are required to win as many apples as possible for a donkey. Children need to maximise their win by choosing among four options

presented to them over 100 trials; they are expected to use feedback from earlier trials to identify the most profitable option(s). In each trial, children can select only one of the options. Each of these options is characterised by three dimensions: amount of win, amount of loss, and frequency of loss. Two options that are disadvantageous in the long run (A and B) are characterised by higher immediate reward (4 apples per trial) but also high loss overall. The other two (C and D) are associated with lower immediate rewards (2 apples) on each trial but are advantageous in the long run because of their low overall loss. In every 10 trials, frequency of loss is high for A (five unpredictable losses of 8, 10, 10, 10, and 12 apples) and C (five unpredictable losses of 1, 2, 2, 2, and 3 apples), and low for B (one unpredictable loss of 50 apples) and D (one unpredictable loss of 10 apples); leading to equal net gain after every 10 trials: Loss of 10 apples for the disadvantageous options, and win of 10 for the advantageous ones.

TD children up to age 12 often fail to integrate feedback in their decision-making and, rather than opting for the advantageous options (C and D), simply prefer to avoid frequent loss, selecting options B and D at a significantly higher level than chance (Huizinga, Crone, & Jansen, 2007; van Duijvenvoorde, Jansen, Visser, & Huizinga, 2010). The TD children (control group) in Rahimi-Golkhandan et al. study showed the same pattern of performance; however, children with DCD showed a preference for option B, characterised by infrequent loss but also high immediate reward. As well, the DCD group selected option D at chance level (i.e., 100/4) and had a significantly lower total net score than the controls. In terms of response speed, these children had significantly faster responses to the disadvantageous (high reward) options, while responses of the control group were similar for both types of options. Finally, the DCD group was slower than the controls when they approached advantageous (low reward) options, only. Taken together, this pattern of performance by the DCD group, which persisted over 100 trials, suggests heightened sensitivity to rewarding, emotionally

significant stimuli (Toplak et al., 2005), which underlies poor affective decision-making. However, other hypotheses could explain the performance of children with DCD on the HDT, notably impaired use of somatic markers and poor inhibitory control.

4.1.3 Predictors of poor performance on the HDT.

4.1.3.1 Somatic Markers. The IGT and the HDT attempt to mimic real-life decision-making in which the logical cost-benefit analysis of available response options is difficult (Bechara et al., 1994). The somatic marker hypothesis (SMH; Damasio, 1994) suggests that under complex task conditions, individuals rely on more intuitive decision processes; in order to solve the problem, use is made of emotion-based biasing signals and sensations (i.e., *somatic markers*) that arise from the body (e.g., viscera, skeletal and smooth muscles) and/or the central representation of the body (Damasio, 2004). These markers are integrated in the emotion circuitry of the brain and signal the likely outcome of each single action (i.e., reward or punishment) and its associated emotional outcomes. The brain then uses these signals to create a *forward model* of the changes expected to happen in the body, which enables one to respond rapidly to the stimuli (Dunn, Dalgleish, & Lawrence, 2006). For instance, somatic markers indicate whether an action is going to be rewarding or punishing, and assist decision-making before the activity emerges in the periphery. Indeed, dysfunction of the brain regions involved in the representation and regulation of the body-state (e.g., basal ganglia, insula, somatosensory cortices) impairs the ability to use somatic markers and leads to poor affective decision-making (Damasio, 1998). Intriguingly, DCD has been linked to deficits in the predictive control of action (Wilson et al., 2013) as well as dysfunction of the basal ganglia and insula in possible sub-groups of DCD (Lundy-Ekman, Ivry, Keele, & Woollacott, 1991; Zwicker, Missiuna, Harris, & Boyd, 2011). In short, it is possible that hot EF deficits in DCD are at least partly due to an impaired ability to effectively use somatic markers.

Another important issue is that performance on many EF tasks is likely to be supported by a combination of cool and hot EF (Hongwanishkul et al., 2005). For instance, individuals need to keep track of wins and losses associated with each option on the IGT or the HDT, with some studies showing significant covariation between performance on these tasks and WM (e.g., Hinson, Jameson, & Whitney, 2002), with others not (e.g., Crone & van der Molen, 2007; Rahimi-Golkhandan et al., in press).

4.1.3.2 Inhibition. Inhibitory control is thought to not only affect other cool aspects of EF but also play a crucial role in hot EF (e.g., the ability to resist temptation and delay gratification) (Diamond, 2013; van Duijvenvoorde, Jansen, Bredman, & Huizinga, 2012). For instance, successful performance on the IGT relies not only on the activation of emotion circuitry, but also other regions known to be active during inhibitory control tasks like anterior cingulate (Braver, Barch, Gray, Molfese, & Snyder, 2001), cerebellum, insula, and inferior parietal cortex (Ernst et al., 2002). Additionally, children need to perform a response reversal on the HDT: optimally, they should avoid options that are immediately appealing, use this feedback, and instead approach options that benefit them in the long run (Dunn et al., 2006). Difficulties in reversal learning and inhibiting such a prepotent response inevitably lead to low net scores on the HDT (e.g. Crone, Vendel, & van der Molen, 2003). It is possible, therefore, that poor affective decision-making could be due to a combination of an overactive emotional system and underdeveloped inhibitory control processes (Bechara & van der Linden, 2005), which results in an ‘emotional overshoot’ (van Duijvenvoorde et al., 2010) whereby impulsive reward-driven performance is not paralleled by effective control systems (Smith, Xiao, & Bechara, 2012).

DCD has been linked repeatedly to poor response inhibition in different settings using a variety of tasks (see Piek et al., 2004; Wilson et al., 2013). Poor inhibitory control and high incidence of externalising problems in children with DCD suggest an overall degree of

impulsivity within this cohort that could be linked to heightened sensitivity to immediate rewards and poor affective decision-making. To further dissect this hypothesis, it is important to test whether the performance on the HDT is a reflection of a distinct deficit in regulating responses to emotionally salient stimuli or is more attributable to a problem of inhibitory control. To this end, I compared the performance of DCD and non-DCD groups when required to inhibit prepotent responses to either neutral or emotionally significant stimuli on two versions of a go/no-go task.

4.1.4 Cool and hot go/no-go tasks. To tease out the contribution of inhibitory control per se, I compared two versions of the go/no-go task: the 'cool' task used neutral facial expressions of men and women, while the 'hot' version presented happy and fearful expressions of the same individuals (Casey et al., 2011). Given the attraction of a happy facial expression, people tend to associate it with positive affect and reward. Indeed, happy facial expressions activate the same brain areas (e.g., ventral striatum) involved in the processing of rewards, and like other rewarding stimuli are approached instinctively, and can provoke impulsive behaviour (Hare, Tottenham, Davidson, Glover, & Casey, 2005). For this reason, it is more difficult to avoid happy faces because one must resist the natural tendency to approach a rewarding stimulus (Hare et al., 2005). The ability to withhold responses to a happy face reflects one's level of self-control and sensitivity to alluring cues, and has been linked to the ability to delay gratification (Casey et al., 2011). No such compulsion is involved when responding to neutral or negative facial expressions.

4.1.5 Aim and hypothesis. The broad aim of this study was to examine whether heightened sensitivity to rewarding stimuli is a distinct deficit in DCD, associated with hot EF deficits in this cohort. More specifically, I sought to determine whether apparent deficit of hot EF is explained selectively by a heightened sensitivity to rewarding stimuli, or is due to a general deficit of inhibitory control. Results have implications for understanding other

aspects of cognitive, motor and emotional functioning in DCD. I predicted that children with DCD would show significantly higher sensitivity to positive stimuli on the go/no-go task, operationalised by commission errors to happy faces, than their TD peers.

4.2 Method

4.2.1 Participants. The sample included 12 (6 boys, 6 girls) children with DCD (*M* (*SD*) $_{age}$ = 9.31 (1.47)), and 28 (10 boys, 18 girls) TD children (*M* (*SD*) $_{age}$ = 9.72 (1.61)). The age range for both groups was between 7 and 12 years, and there was no significant difference between the average ages of the groups (p = .45). Children who had a Neurodevelopmental Index (NDI) score of 80 or less on the McCarron Assessment of Neuromuscular Development (MAND; McCarron, 1997) are considered *at risk* for DCD (Piek et al., 2004), and were allocated to the DCD group (*M* (*SD*) $_{NDI}$ = 77.82 (5.73)). Four children had an NDI of between 55 and 70 (indicating moderate motor difficulties (Piek et al., 2004) while the rest had NDIs between 70 and 80. The control group included children with an NDI of 100 or above (*M* (*SD*) $_{NDI}$ = 109.29 (6.24)). The exclusion criteria were the diagnosis of other developmental disorders, such as ADHD, or any other neurological, learning, or physical disorder. I did not conduct any neurological or medical examination, which is used to ensure the criterion C of the DCD diagnosis in DSM-IV-TR is met (Geuze, Jongmans, Schoemaker, & Smits-Engelsman, 2001), mainly because children were all enrolled in three mainstream primary schools in Melbourne, and their intelligence levels were inferred to be within the normal range; no children were attending remedial classes for literacy or mathematics.

4.2.2 Materials.

4.2.2.1 Go/No-Go task. The two versions ('cool' and 'hot') of the go/no-go task used grayscale pictures of 12 individuals (6 males, 6 females) from the NimStim collection, downloaded from www.macbrain.org. The size and luminance were the same for all the

images. The faces used were numbers 6, 8, 11, 14, 15, 16, 27, 28, 36, 39, 44, and 45. Neutral facial expressions were used for the cool task, while the stimuli for the hot task were happy and fearful faces of the same individuals. Each task consisted of two runs in which the go target for the first run was the no-go target in the second run. Therefore, both cool and hot tasks used a 2 (either male, female or happy, fearful) x 2 (go, no-go) factorial design. Before the start of each run, which presented 40 pictures in a pseudorandom order (28 go, 12 no-go), children received a short notification on the monitor informing them of which sex (for the cool task) or facial expression (for the hot task) served as the go target. Each picture was presented for 500ms followed by a 1500ms inter-stimulus interval, during which children saw a fixation cross, but were still able to respond. Children were instructed to respond as quickly and as accurately as possible, pressing the spacebar for the go trials, and refraining from responding to no-go targets. Accuracy (of responding to go stimuli, and withholding a response to no-go targets) and reaction time (RT) to go stimuli were the main indices of performance in each task.

4.2.2.2 McCarron Assessment of Neuromuscular Development (MAND). The MAND (McCarron, 1997), which includes 10 short tests of fine and gross motor skills (5 each), provides a standardised index of motor skills for 3.5 to 18 year old individuals. The NDI score is calculated by comparing the sum of scaled scores (on the 10 tests) to age-appropriate norms for each child. The MAND is recognised as a valid and reliable assessment tool for motor impairment (Piek et al., 2004). It has a good test-retest reliability (.67 to .98) over a 1-month period, acceptable criterion and concurrent validity (McCarron, 1997), and good specificity and sensitivity (Tan, Parker, & Larkin, 2001).

4.2.3 Procedure. After the approval of this experiment by the Human Research Ethics Committee of the Australian Catholic University, different primary schools across Melbourne, Australia, were contacted, and the parents and guardians of children in the three

schools that expressed their interest in this project received plain language statements about the study. Informed consents were obtained from the parents and guardians before the start of the experiments. The testing was conducted in a quiet environment, and during children's class time. Half of the children first completed the go/no-go task, while the other half were first tested on the MAND. I also counterbalanced the two versions of the go/no-go task as well as the two different runs within each task. The go/no-go task was administered on a laptop computer running the E-Prime version 1.1 (Schneider, Eschman, & Zuccolotto, 2002), and the testing session of each child took approximately 20 minutes.

4.2.4 Data analysis. The most important index of performance, which provided a direct test of my hypothesis, was the commission error. This outcome variable measured the ability to withhold a response to no-go targets: low commission errors represent better performance. Commission errors were submitted to a 2-way mixed-factorial ANOVA with group (DCD vs. control) the between-subjects factor, and the type of no-go stimulus (male, female, happy, and fearful) the repeated factor. The number of consecutive go trials prior to a no-go target has been linked to a higher rate of commission errors (Durston, Thomas, Worden, Yang, & Casey, 2002). The highest number of continuous go trials, in each run of the hot task in the current study, was four. To assess the impact of task difficulty on performance, I compared the groups on the proportion of commission errors after four consecutive go trials. Omission errors were operationalised as the percentage of trials in which children did not approach go targets. Omission errors were also submitted to a 2-way mixed-factorial ANOVA: group (DCD, control) and type of go stimulus (male, female, happy, and fearful). RT to go targets was compared between groups, reflecting whether the emotional significance of stimuli had any impact on the latency of responses. Finally, to investigate the potential impact of emotion discrimination on the task performance, and to determine how successfully children were able to discriminate between various facial

expressions, I used d-prime (d') which provides an index of sensitivity to each stimulus in a go/no-go task. d', which takes into account the respondent's bias, can be used as an index of emotion recognition (Tottenham, Hare, & Casey, 2011). The following formula was used to calculate this index: [d' = *z*(H) - *z*(F)]. For each child, *z*(H) is the standardized rate of approaching the relevant go target ('hits'), while *z*(F) is the standardized score for commission errors or false alarms. The higher the d' value, the more capable is the child to discriminate go and no-go targets.

4.3 Results

4.3.1 Commission errors. Figure 4.1 shows the proportion of commission errors on no-go trials for each group. As shown in this Figure, the groups had similar accuracies during all the no-go trials, except when the no-go target was a happy face. The average commission error was notably higher for the DCD group who failed to suppress their responses to happy faces on more than half of the trials.

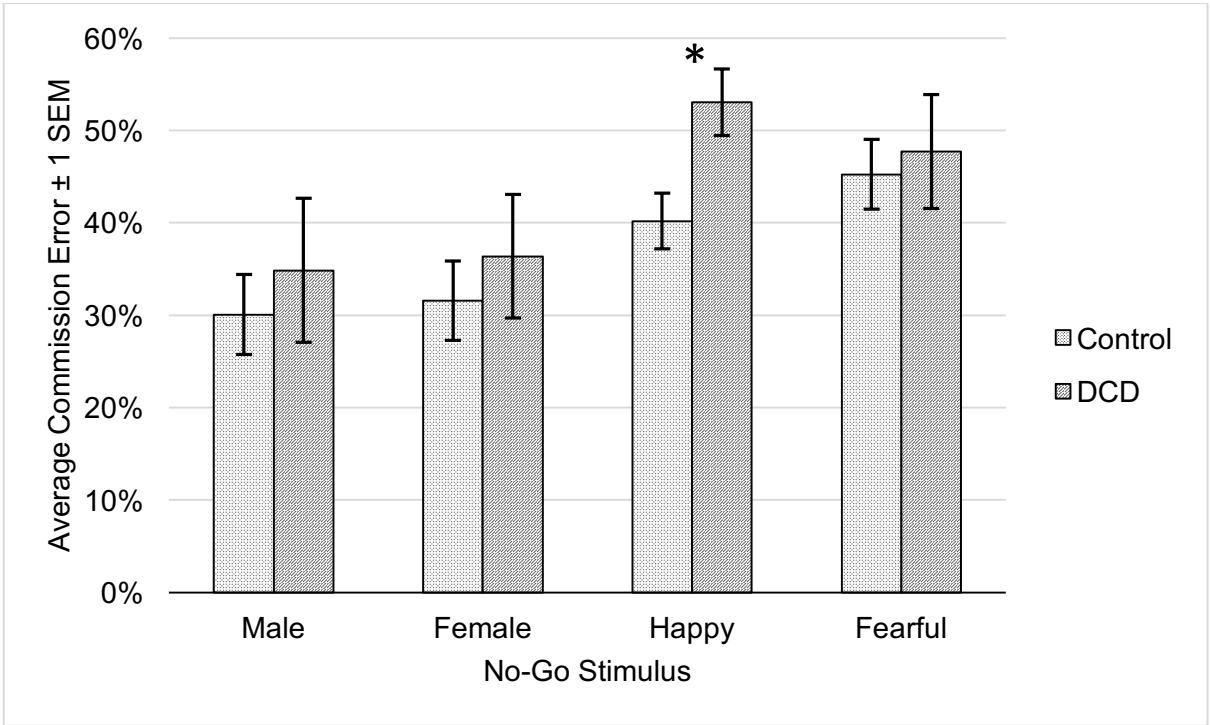

Figure 4.1. Mean (±SE) commission errors to no-go targets in the DCD and control groups (*$p < .05$)

Interestingly, the 2 x 4 mixed-factorial analysis of variance (ANOVA) did not show a significant interaction between group and stimulus, Wilks' $\Lambda = .92$, F (3, 35) = 1.05, $p = .38$, $\eta^2 = .10$. The low observed power for this particular analysis (.26) possibly accounts for the lack of a significant interaction. The analysis of simple main effects, however, provided a better reflection of group differences. There was no significant difference in commission errors on both versions of the cool task: female no-go: $p = .55$, $d = 0.21$ (95% CI [- 0.49, 0.91]); male no-go: $p = .57$, $d = 0.20$ (95% CI [- 0.50, 0.90]). In contrast, I found significant differences in commission errors on the hot task. While the groups showed no difference in commission errors in response to fearful no-go targets: $p = .73$, $d = 0.12$ (95% CI [- 0.58, 0.82]), the DCD group produced more commission errors when the no-go stimulus was a happy face, $p = .02$, $d = 0.86$ (95% CI [0.14, 1.58]). The bootstrapped analysis (using 5000 samples, and 95% CI) produced a p value of .009 for the group differences in commission errors to happy faces. None of the other bootstrapped analyses for commission errors reached significant levels – smallest p was for female commissions (.53). Moreover, within-group analyses (of the hot task) showed that while controls had less error in response to the happy faces ($d = 0.27$), the DCD group were better able to withhold their responses to the fearful faces ($d = 0.29$).

There were three instances in each run of the 'hot' task where a no-go target was preceded by four go trials. Therefore, the maximum number of commission errors after these 'difficult' trials was 3. However, the error rate of each group to either happy or fearful non-targets was less than half of this [no-go stimulus: (a) fearful: M (SE) $_{DCD} = 1.27$ (0.36), M (SE) $_{Control} = 1.04$ (0.17); (b) happy: M (SE) $_{DCD} = 1.18$ (0.32), M (SE) $_{Control} = 1.11$ (0.19)], with no significant differences between DCD and control groups ($p > .5$). I did not run the same analysis on the 'cool' task because its two runs did not have equal number of 'difficult' trials.

With respect to individual differences, Figure 4.2 presents the number of commission errors to happy no-go targets by each child in the DCD and control groups. There were six children in the DCD group who made commission errors on more than half of the trials (7 or more errors out of 12 trials), 3 of which had NDIs less than 70; as well, the remaining child with moderate motor difficulties made five errors. The difference between the commission error rates of children with NDI <70 (58.3%) and the rest of the DCD group (50.0%) was about 0.7 of the *SD* of the total group average (53%).

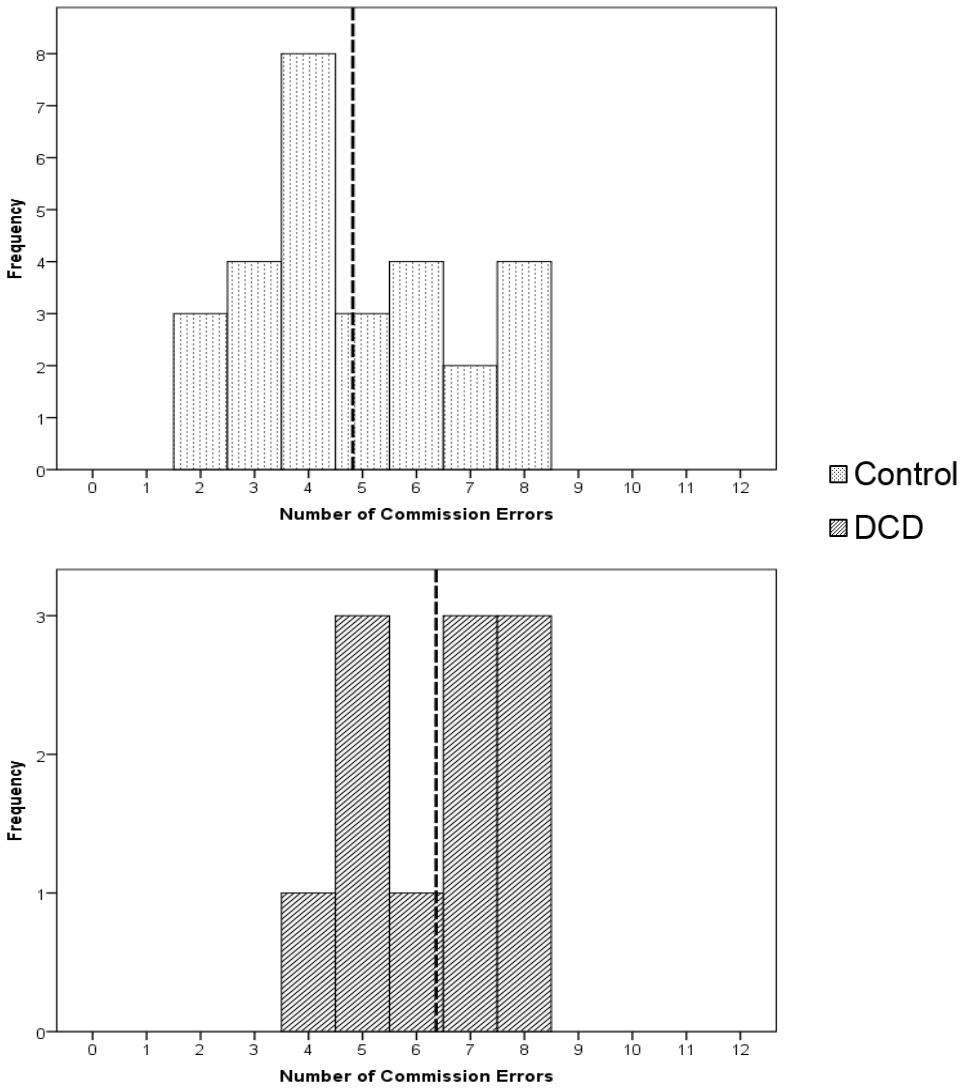

Figure 4.2. Frequency distribution of commission errors to happy no-go targets. The dotted line represents group average. Maximum possible number of commission errors was 12 (trials).

4.3.2 Omission errors. Figure 4.3 presents the average (±SE) percentage of omission errors in each group. Both DCD and control groups produced few errors, showing that each were able to correctly identify and approach the relevant go stimulus in each task.

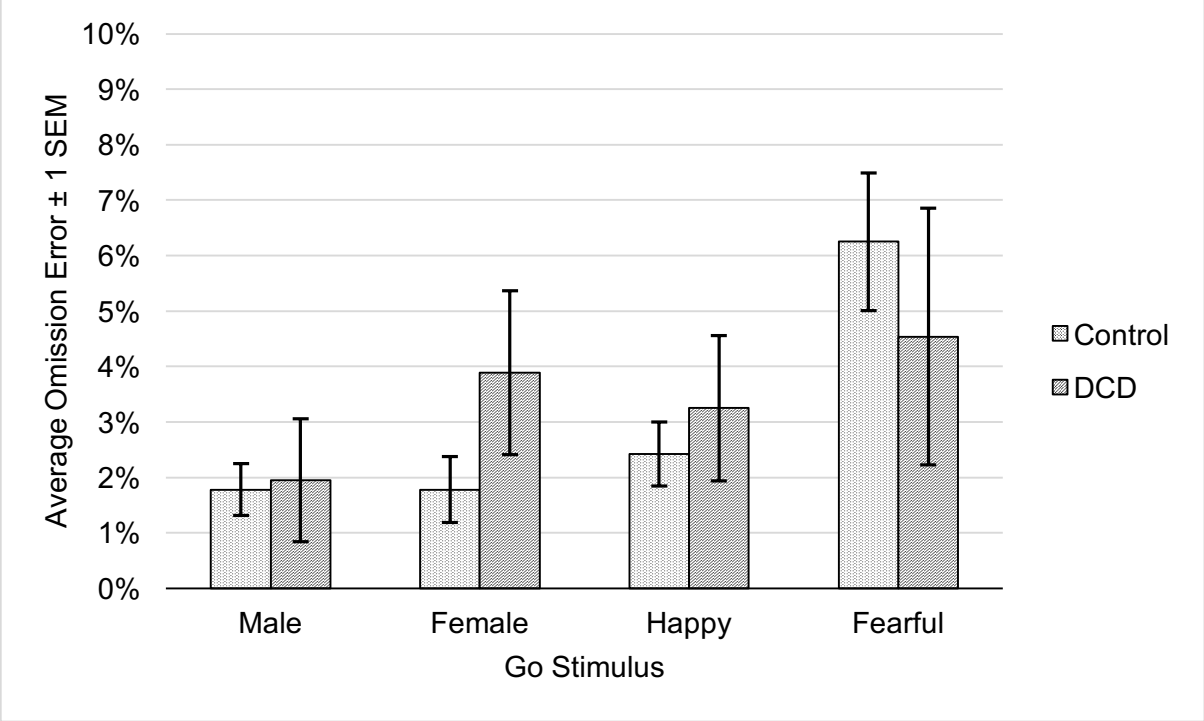

Figure 4.3. Mean (±SE) omission errors to go targets in the DCD and control groups

The 2 x 4 mixed-factorial ANOVA did not reveal any significant interaction between group (DCD, control) and the go stimulus (male, female, happy, and fearful), Wilks' $\Lambda = .90$, F (3, 35) = 1.25, $p = .31$, $\eta^2 = .10$. Although the DCD group made more errors than controls in response to male faces ($p = .87$, $d = 0.06$ (95% CI [- 0.64, 0.75]), female ($p = .12$, $d = 0.57$ (95% CI [- 0.14, 1.28]), and happy ($p = .51$, $d = 0.24$ (95% CI [- 0.46, 0.94]), they performed better than controls in response to fearful targets ($p = .49$, , $d = - 0.25$ (95% CI [- 0.95, 0.45]). Within-group analyses showed no significant differences in the omission errors of the DCD group to different stimuli. However, the controls had significantly fewer omission errors in response to fearful faces (p for all pairwise comparison was less than .003).

4.3.3 Reaction time. Table 4.1 present the average (and SD) RT of each group to the four possible go stimuli. According to this table, children with DCD always had a slower response to different go targets. More importantly, the slowest RT for both groups was to the fearful faces.

Table 4.1

Mean (SD) RT of DCD and Control Groups to Different Go Targets

	Go Stimulus			
	Male	Female	Happy	Fearful
DCD	528.53 (47.37)	543.14 (57.50)	545.16 (88.22)	567.85 (59.34)
Control	501.77 (64.15)	496.47 (75.01)	516.75 (80.43)	545.75 (95.37)

Note. RT is in milliseconds.

A 2 x 4 mixed-factorial ANOVA found no significant interaction between group and go stimulus, Wilks' $\Lambda = .94$, $F\ (3, 35) = 0.69$, $p = .56$, $\eta^2 = .06$, suggesting that the pattern of RT was similar for both groups. Tests of simple main effects showed no significant difference between RTs to the go targets of the cool task (i.e., male and female faces) in each of the two groups ($p > .35$). On the hot task, however, both groups responded faster to the happy faces ($d_{\text{control}} = 0.32$, $p = .001$, $d_{\text{DCD}} = 0.28$, $p = .08$). Moreover, group differences in RT did not reach significance level for any of the go targets ($p > .07$).

Children with DCD are often significantly slower than their TD peers on most chronometric tasks (e.g. Piek & Skinner, 1999). However, their RT to different go stimuli was not significantly different to that of controls. This prompted us to correlate RT (to go targets) to commission errors in each group to investigate the possibility of a speed-accuracy trade-off in the DCD group (Table 4.2). Interestingly, however, it was the control group whose performance suggested a possibility of speed-accuracy trade-off in both runs of the

'hot' task. The correlations for the DCD group, on the other hand, were small and non-significant for both happy and fearful go targets. Even though this pattern of results is reversed on the 'cool' task, the DCD group did not have significantly more commission errors than the controls on this version of the go/no-go task.

Table 4.2

Pearson's r Correlations between RT to Go Targets and Commission Errors to No-Go Stimuli

	No-Go Stimulus			
	Male[1]	Female[2]	Happy[3]	Fearful[4]
DCD	.58^	-.71*	-.03	-.22
Control	-.26	-.16	-.33^	-.49*

Note. Go stimulus: 1 = female, 2 = male, 3 = fearful, 4 = happy.

*$p < .05$. ^$p < .10$

4.3.4 D-prime. Figure 4.4 depicts d' for each of the go targets. The DCD group had a lower sensitivity to each of the face stimuli. However, the group (DCD, control) by stimulus (male, female, happy, fearful) interaction was not significant, Wilks' $\Lambda = .98$, F (3, 35) = 0.19, $p = .90$, $\eta^2 = .02$. Tests of simple main effects also revealed no significant differences in d' between the two groups ($.32 < p < .58$).

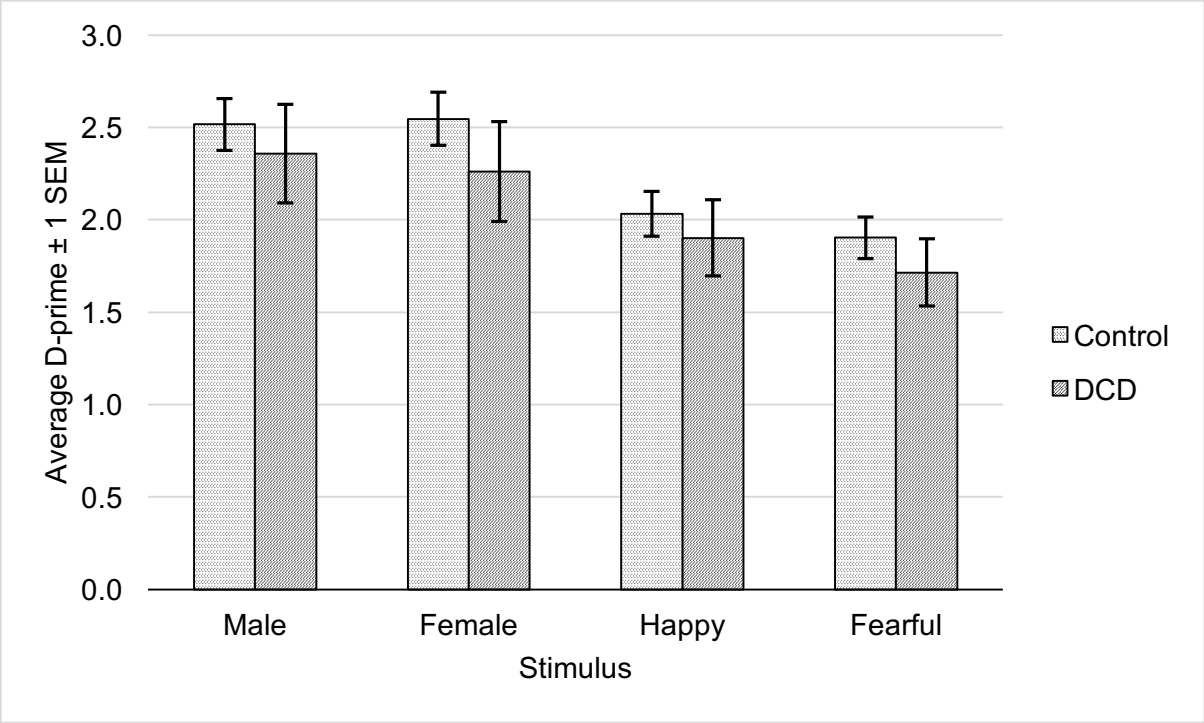

Figure 4.4. Mean (±SE) d' for DCD and control groups in response to the go stimuli

Finally, it should be noted that different ratio of boys and girls in the two groups could have confounded the current findings. However, the inclusion of gender as a covariate in the analyses of outcome measures did not lead to any notable change in *p* values or effect sizes. For instance, the *p* value for the significantly higher commission errors of the DCD group in response to happy non-targets changed from .020 to .026, and the effect size remained the same.

4.4 Discussion

The aim of this study was to investigate whether children with DCD show heightened sensitivity to emotionally significant stimuli. The prediction was that the DCD group would have more difficulty than their TD peers in suppressing responses to positive social cues, namely happy non-targets in a go/no-go task. The findings supported this prediction, and suggest that poor affective decision-making in DCD on hot EF tasks can be explained in part by a high sensitivity to rewarding stimuli, and that this effect is not attributable to a

generalised deficit of inhibitory control. In the discussion that follows, I explore factors underlying this pattern of performance in DCD and suggest interactions between emotion processing and action control centres. I also review the implications of these findings for understanding self- and emotion-regulation in children with motor coordination problems.

4.4.1 Why do children with DCD show greater sensitivity to rewarding stimuli?

Successful performance on go/no-go tasks that use emotionally salient stimuli requires a combination of emotion discrimination, cognitive control, and affect regulation (Tottenham et al., 2011).

4.4.1.1 Emotion Discrimination. Deficits in recognising and responding to static and changing facial expressions of emotion have been reported in DCD (Cairney et al., 2013; Cummins et al., 2005). However, poor emotion discrimination does not explain the pattern of performance observed in DCD. Both DCD and control groups in the current study responded faster to happy than fearful faces suggesting adequate facial discrimination and in-built approach behaviour in both groups. Put simply, the emotional valence of the stimuli was apparent to and discriminative in both groups. This is similar to the previous reports of TD children, healthy adolescents and adults who generally show a bias for positive stimuli on emotional go/no-go tasks (Hare et al., 2005; Schulz et al., 2007; Tottenham et al., 2011; Urben, van der Linden, & Barisnikov, 2012). In contrast, affective disorders (e.g., major depressive disorder) have been linked to faster responses to negative stimuli (Murphy et al., 1999), while autism characterised by the absence of a preference to either stimuli (Duerden et al., 2013). There is still insufficient evidence about the performance of children with ADHD on the emotional go/no-go task, particularly in response to the positive stimuli.

Although all children responded with a high level of accuracy to go targets, and no group difference was evident, few omission errors do not necessarily mean good discrimination ability. To illustrate, selecting both go and no-go targets would result in high

approach levels, but also high commission errors. The analyses of d', however, showed that DCD and control groups were equally sensitive to the go targets; indicating that differences in commission errors could not be due to poor emotion discrimination of children with DCD. Finally, my findings suggest that speed-accuracy trade-off, which was previously observed in the other developmental disorders such as autism (Yerys, Kenworthy, Jankowski, Strang, & Wallace, 2013), can also be ruled out as the contributing factor to the performance of the DCD group on the 'hot' go/no-go task.

4.4.1.2 Cognitive Control. Apart from the need to recognise the emotional significance of stimuli, behavioural and neuroimaging data suggest emotion regulation relies on the functioning of control networks in the context of emotional information. To illustrate, an increased false alarm rate to emotional stimuli in TD children compared with otherwise normal adolescents and adults has been linked to the protracted development of cognitive control (Tottenham et al., 2011). In a similar vein, Yerys and others (2013) reported that commission error rate was positively correlated with ADHD symptoms of hyperactivity and impulsivity among a group of children with autism, who made more impulsive responses to emotional stimuli than their TD peers. Neurophysiological evidence also highlights the impact of control processing centres in modulating sensitivity to appetitive cues. For example, adults who at age 4 performed poorly on the seminal Stanford marshmallow experiment (Mischel, Ebbesen, & Zeiss, 1972) had more difficulty avoiding happy non-target faces in a go/no-go task, and showed exaggerated recruitment of ventral striatum, and hypoactivity in the inferior frontal gyrus – indicating that resistance to temptation relies on fronto-striatal regions involved in emotion regulation (Casey et al., 2011). Likewise, immature prefrontal activity has been linked to poor response inhibition in emotional contexts (Hare et al., 2008). Therefore, an alternative hypothesis is that a generalised deficit of

inhibitory control may underlie heightened sensitivity of the DCD group in the current study to positive stimuli.

However, this suggestion can also be ruled out as an alternative hypothesis. The higher incidence of commission errors in the DCD group was stimulus specific—it occurred when the no-go stimulus was a happy face but not for fearful or neutral faces. The absence of a significant group difference on commission errors has been previously reported in studies of DCD that used a similar go/no-go paradigm as ours (Piek et al., 2004; Querne et al., 2008). A pure deficit of cognitive control would manifest itself in a general increase in commission errors regardless of the type of stimulus. For example, children show more difficulty – than adolescents or adults – inhibiting responses to no-go targets, regardless of the emotional valence of the stimulus (Tottenham et al., 2011). Indeed, the performance of the DCD group is in line with the evidence that behavioural correlates of the ability to withhold prepotent responses are a function of not only inhibitory control, but also the salience of the stimulus to each person (Casey et al., 2011). One way to enhance the salience of stimuli, and increase false alarm rate to the targets one must resist is to manipulate task difficulty by increasing the number of consecutive go trials preceding a no-go trial. This method, which does not depend on the emotional valence of the stimulus, increases cognitive demands, and improves the chances of detecting differences in cognitive control (Durston et al., 2002; Eigsti et al., 2006). The finding that children with DCD performed comparably to controls on difficult trials of the 'hot' go/no-go task further suggests that stimulus specific deficit of response inhibition in the DCD group is probably not solely due to poor impulse and attention control.

4.4.1.3 The Interaction of Cognitive Control and Emotion Processing Networks. Alternatively, the inherent salience of a stimulus, which is independent of task design, can disrupt response inhibition by itself. To illustrate, appetitive cues are more difficult to avoid, and require greater impulse control because of the natural tendency to approach them. The

ability to invoke greater cognitive control in emotional contexts (e.g., using reappraisal strategies to cool the appealing aspects of tempting stimuli) enhances delay of gratification (Mischel et al., 1972), and improves the chances of withholding a response to positive social stimuli (Casey et al., 2011). Therefore, a more likely interpretation is that emotional stimuli constitute a higher load on inhibitory control than neutral stimuli, and that children with DCD have more difficulty controlling their responses to the former. To illustrate this point, Lagattuta and colleagues examined inhibitory control using two versions of a Stroop-like card task: the neutral version used pictures of "day" and "night" as the competing stimuli, while the emotionally-laden version used happy and sad faces. It was shown that inhibition was more difficult for faces, with no ceiling effects, even for adults (Lagattuta, Sayfan, & Monsour, 2011). In DCD, heightened sensitivity to positive social cues may reflect a reduced level of coupling between emotion processing and cognitive control centres.

Differential rates of development in cognitive and affective systems can determine the drive or tendency to approach emotionally significant stimuli (Hare & Casey, 2005). For example, increased risk-taking and heightened sensitivity to appetitive cues in adolescence is linked to early maturation of brain regions (like the ventral striatum) that are involved in the representation of potential rewards compared with later developing systems that support controlled/planned behaviour like fronto-striatal networks (Somerville, Hare, & Casey, 2011). Immaturities in the development of cognitive control centres in children with DCD may reduce the ability to modulate the activation of emotion processing centres, resulting in more approach-oriented behaviour toward positive stimuli. Children with DCD did not differ to TD children when inhibiting responses to less emotionally-rewarding stimuli (like neutral or fearful faces); higher commission errors were only evident when required to enlist inhibitory control in response to positive social cues which are known to increase the activation of emotion processing networks (Hare et al., 2005). Maturation of EF networks and

their reciprocal connections with limbic and frontal motivation systems unfolds gradually over childhood and adolescence (Dennis, Malone, & Chen, 2009). Some delay in maturation could disrupt affect-regulation in children with DCD, with implications for self-regulation.

There is only scant evidence in DCD regarding the function of brain regions that support the ability to suppress responses to compelling (positive) stimuli or inhibition of impulsive behaviour more generally. Activation of frontro-striatal regions (Somerville et al., 2011), caudate nucleus (Hare et al., 2005), and parts of basal ganglia such as subthalamic nucleus (Frank, 2006) are known to subserve inhibitory function, particularly in the face of emotionally salient stimuli. There had been a handful of studies on the neural correlates of DCD (see Kashiwagi & Tamai, 2013 for a review). There is some debate about the involvement of basal ganglia in DCD (Groenewegen, 2003; Wilson, Maruff, & Lum, 2003), and it is possible that dysfunction within this structure and/or fronto-striatal regions contribute to a heightened sensitivity of children with DCD to rewarding stimuli.

It is also noteworthy that the performance of the children with moderate motor difficulties in the DCD group raises the possibility of a *dose-response relationship* (see Cairney & Veldhuizen, 2013) between the level of motor functioning and performance on the go/no-go task; however, numbers were small for the DCD group in the current study. Moreover, the analysis of commission errors at the individual level shows that some children in the DCD group perform equally to or even better than their TD peers. This finding supports the view that DCD is indeed a heterogeneous developmental disorder (Green, Chambers, & Sugden, 2008).

4.4.2 Self-Regulation. Exerting cognitive control in the face of salient, appetitive cues can be challenging for children and, in the case of DCD, may pose particular difficulty; for instance, affecting the ability to suppress competing thoughts and actions, and undermining self-regulation. Poor self-regulation has been likened to control systems being

'hijacked' by the primitive limbic system, disrupting the neural modulation of behaviour (Somerville et al., 2011). At a functional level, poor self-regulation can lead to the disturbances of goal-directed action, concentration, and academic achievement.

Self-regulation, however, is not a well-articulated construct. Various definitions of self-regulation have been proposed and with them a variety of tests to assess the broad construct (see Hoyle & Davisson, 2011 for a review). One important point of consensus is that different aspects of self-regulation (i.e., the ability to plan, monitor, and modify attention, feelings, and behaviour) are significant predictors of success in everyday life. For example, high impulsivity, and an impaired ability to control thoughts and actions during childhood have been linked to poor academic achievement (e.g., leaving school), health problems (e.g., smoking, taking drugs), and risky decision making during adolescence, as well as poor physical and mental health and lower quality of life in adulthood, even after controlling for IQ, gender, and socioeconomic status (Moffitt et al., 2011).

Diagnostic criteria for DCD suggest that the motor disturbance impacts academic achievement, participation, and everyday skills. The current study data accord with the view that selective deficits of inhibitory control might be a common underlying issue explaining both poor self-regulation and motor control in DCD, with the functional outcomes being reduced success at school and psychosocial issues.

The ability to successfully regulate one's thoughts and feelings in the service of goal-directed action relies on a combination of both cool and hot EF. For example, cool EF is a significant predictor of school readiness and classroom functioning, while hot EF has been implicated in the development of early literacy and math skills (Wyatt, 2013). My data and earlier studies of cool EF in DCD suggest that it is prudent to consider how interventions might be modified to accommodate the reduced capacity of children with DCD on EF, which we know has implications for self-regulation. Indeed, interventions that target EF have been

shown to enhance psychosocial and physical functioning in children (see Diamond & Lee, 2011). At present, our direct knowledge of self-regulation in children with DCD is limited to the motor domain (Sangster Jokic & Whitebread, 2011). There is a dire need for research in the broader domain of self-regulation.

4.4.3 Emotion-Regulation. Emotion regulation, which can be viewed as a subset of self-regulation, involves monitoring, evaluating, and modifying the intensity and temporal dynamics of emotional responses (Thompson, 1994). Underlying it is the ability to suppress responses to distracting stimuli and control attention in emotionally demanding contexts (Dennis et al., 2009)—modifying responses to both the pleasurable and the aversive (Tottenham et al., 2011). For instance, in a hot go/no-go task, the average false alarm rate to both emotional no-go stimuli is considered as the index of emotion regulation. It has been shown that children's ability to shift attention from an appetitive cue in a delay of gratification task predicts the ability to resist temptation on the task (Cole, 1986) and later ability to manage negative emotions during adolescence (Shoda, Mischel, & Peake, 1990). The current study showed that children with DCD might have higher sensitivity to positive social cues, and lower resistance to temptation than their TD peers. Whether this has direct implications for coping and psychological adjustment in DCD remains to be investigated.

In contrast, the DCD group approached negative stimuli (i.e., fearful faces) on go trials at a comparable rate and speed to controls. The absence of significant group differences here could reflect the balance between positive and negatively-valenced stimuli task protocol. The presence of happy no-go faces (positive, rewarding stimuli) on 30% of the trials could have created a prepotent tendency for the children to respond. This suggests that on go trials, if the negative stimulus was paired with a neutral stimulus, we could have seen lower response rates. Moreover, commission errors in response to fearful no-go targets could be partly due to a build-up of the tendency to approach happy faces, and not the tendency to

approach negative stimuli per se. This limitation is particularly relevant to the performance of the DCD group who showed a heightened sensitivity to positive stimuli. Therefore, it is recommended that follow-up studies pair emotional targets (e.g., happy, fearful, or sad faces) with neutral facial expressions in order to obtain a more accurate measure of emotion regulation.

Results from the go/no-go task that bear on approach behaviour to negatively valenced stimuli stand in contrast to the effects we would expect to see in the real world. Generally, a reduced ability to approach negatively valenced stimuli may have important repercussions for the efficacy of action, and psychological well-being of individuals. To illustrate, children with DCD often refrain from partaking in physical/sport activities mainly due to their fear of being criticised, ridiculed, and bullied by their peers. This fear of repeated failure not only reduces the desire to practice skills (Cairney et al., 2013), but also makes participation in skilled motor activities an unpleasant experience (Cairney & Veldhuizen, 2013). The prior experience of physical activity as aversive can engender negative attitudes and feelings about participation per se; that is, the negative emotion/attitude can further reduce the tendency to approach physical play, and create the self-belief that physical competence and self-efficacy is limited. An unfortunate outcome, therefore, is anxiety and social isolation. In sum, this avoidance of ‘negative’ stimuli creates a vicious cycle (Skinner & Piek, 2001) which can lead to more physical and psychological problems.

4.4.4 Conclusion. In sum, I established, using a go/no-go task, that a small group of children with DCD showed higher sensitivity and lower resistance to positive social cues than their TD peers; no abnormality in response was seen for both neutral and negative cues. The main implication of this pattern of results is that children with DCD might experience particular difficulty enlisting inhibitory control when presented with emotionally and motivationally salient stimuli, particularly for strong appetitive cues. The ability to couple

emotion processing and cognitive control networks in the service of action may be compromised. I recommend use of neuroimaging techniques, using larger samples of children with DCD, to examine more closely the action of neural systems that support emotion regulation and the coordination of goal-directed action. High sensitivity to appetitive, rewarding cues in DCD and reduced ability to enlist inhibitory control in response to them, may impair both the coordination of motor skill and self-regulation. The deficits of hot EF that I have identified may involve a constellation of processes that impact motor control, coordination, and functional behaviour.

Chapter 5: Revealing Hot Executive Function in Children with Motor Coordination Problems: What's the Go?

5.1 Introduction

Recent experimental work has raised several viable hypotheses about the neurocognitive underpinnings of atypical motor development (or Developmental Coordination Disorder—DCD). In a recent meta-analysis of the literature (Wilson, Ruddock, Smits-Engelsman, Polatajko, & Blank, 2013), the pattern of motor and cognitive performance in children with and without DCD was compared across 129 studies between 1998 and 2011. In addition to a broad cluster of motor control and learning issues, what was striking about this work was the consistent pattern of executive dysfunction (Cohen's d >1) across inhibitory control, working memory (WM) and executive attention tasks. Executive function (EF) refers to a group of neurocognitive processes involved in conscious and effortful control of thought, emotion, and behaviour. More specifically, EF encompasses WM, executive attention, mental flexibility, and inhibition (Diamond, 2013). In recent studies, I have shown executive dysfunction in DCD extends to tasks that require so called 'hot EF'. These tasks have superimposed the requirement that emotional cues (positive and negative) be processed in order to achieve a task goal—hence the term hot EF. The issue of hot EF in children with DCD is particularly salient given other work showing that these children have difficulty with self-regulation (Sangster Jokic & Whitebread, 2011) and a higher incidence of anxiety associated with their motor problems (Zwicker, Harris, & Klassen, 2012). In the study reported here, I investigated hot EF using a go/no-go paradigm that used facial stimuli that were readily discriminable by children. Critically, I tested the specificity of the putative deficit in DCD that relates to the ability to inhibit responses to salient no-go stimuli.

DCD occurs in about 5-6% of children, and is characterised by problems in learning fine and/or gross motor skills, with resultant disruptions in daily living activities and/or academic achievement (DSM-5; APA, 2013). Also associated with the disorder are a range of psychosocial issues including poor self-esteem, low self-efficacy (particularly for physical

tasks), and impaired social relations. A bidirectional relationship between motor and cognitive development in DCD is supported by data showing concurrent deficits in each domain. EF deficits in DCD have led some to postulate that DCD is more than a purely motor disorder, but rather a broader neurodevelopmental syndrome (Wilson et al., 2013), one also associated with poor social-emotional adjustment (Zwicker et al., 2012). As a result, efforts to understand the underlying basis of DCD have centred on examination of motor control and EF.

While the classification of EF varies from one theorist to another (see Welsh & Peterson, 2014), two points have been brought into clear focus by recent work: first, the emotional valence of stimuli is critical in determining what nodes in a neural network are enlisted when performing an EF task, and second, component processes like inhibition are shared between 'cold' and 'hot' EF. Cold EF, subserved by dorsolateral prefrontal cortex (DL-PFC), is enlisted when one interacts with abstract, decontextualised stimuli, such as in the traditional lab-based tests of EF used to assess WM, mental flexibility, and inhibition (Zelazo & Carlson, 2012). Hot EF, associated with ventromedial prefrontal cortex (VM-PFC), is more relevant to everyday decision-making, and incorporates the ability to reappraise the emotional-motivational significance of stimuli in order to voluntarily inhibit or activate a particular behaviour. The hot EF tasks, such as the delay of gratification and gambling tasks (e.g., Iowa Gambling Task (IGT); Bechara, Damasio, Damasio, & Anderson, 1994), mimic aspects of real-life decision-making through the use of rewards and losses. I recently showed that in the case of DCD, atypical patterns of performance were evident not only for traditional (cold) EF tasks but also so-called hot EF. Intriguingly, there may be a reduced ability in DCD to resist stimuli that offer high immediate reward, but longer term loss (Rahimi-Golkhandan, Piek, Steenbergen, & Wilson, 2014). Impaired inhibitory control may contribute to this.

Response inhibition is believed to be an important determinant of not only cold EFs (e.g., WM and set-shifting), but also the ability to resist temptation (Aron, Robbins, & Poldrack, 2004; Diamond, 2013; van Duijvenvoorde, Jansen, Bredman, & Huizenga, 2012). For instance, some regions (e.g., anterior cingulate) that are active during inhibitory control tasks (e.g., go/no-go; Braver, Barch, Gray, Molfese, & Snyder, 2001) also predict optimal performance (i.e., higher net scores) on the IGT and its variants which tap into both the reward and inhibitory circuitry of the brain (Ernst et al., 2002; Smith, Xiao, & Bechara, 2012). Poor response inhibition prevents the contemplation and implementation of other response options, and eventually leads to low self-control and impulsive behaviours in children (Riggs, Blair, & Greenberg, 2004).

Recent fMRI data suggest that hypoactivity of DL-PFC in children with DCD may explain their reduced ability to switch (i.e., mentally shift) between go and no-go motor responses (Querne et al., 2008). Moreover, given the extensive connections between VM-PFC and the emotion circuitry of the brain, disruption of prefrontal regulation could underlie reduced emotional regulation that has been observed in DCD (Deng et al., 2014). Although cognitive inhibition – integral to interference control and selective attention – has been tested extensively in DCD, there is a dire need to investigate behavioural inhibition (i.e., self-control, resistance to temptation) (Diamond, 2013) in this cohort. Understanding the mutually interactive relationship between motor, cognitive and affective processes is a critical issue in both typical and atypical child development (Zelazo & Müller, 2011) and, in the case of DCD, holds significant implications for the design of interventions that target motor and/or behavioural issues, e.g. how training tasks can accommodate EF deficits in children.

5.1.1 Deficits of hot EF in DCD. In an earlier study (Rahimi-Golkhandan, Piek et al., 2014), I investigated EF in DCD using a child-friendly variant of the IGT, called the Hungry Donkey Task (HDT; Crone & van der Molen, 2004). The optimal performance on the HDT

relies on ignoring options that are initially rewarding but lead to an overall loss, and instead choose those associated with lower immediate reward (see Crone & van der Molen, 2004 for a description of the HDT). Children with DCD had a significantly lower total net score than typically developing (TD) children, and opted for the disadvantageous (high immediate reward) options. Moreover, even though the reaction time (RT) of the TD group did not depend on the type of option, the DCD group responded significantly faster to the disadvantageous options. One of the possible reasons for this pattern of performance in the DCD group is a deficit of inhibitory control (Rahimi-Golkhandan, Steenbergen, Piek, & Wilson, 2014).

The follow-up study (Rahimi-Golkhandan, Steenbergen et al., 2014), with the same groups of children, used an emotional go/no-go task to investigate specifically the role of inhibition in the heightened sensitivity of the DCD cohort to rewarding stimuli. Children completed both 'cold' (neutral facial expressions) and 'hot' (happy and fearful faces) versions of the task. There were no significant group differences in omission errors, a measure reflecting attention (Schulz et al., 2007). As well, analysis of d' – a measure of perceptual sensitivity – did not reveal any significant group differences, indicating that the emotional valence of the stimuli was apparent to both groups, and that both children with DCD and the controls were equally adept at recognising facial expressions. However, commission error rate, a measure of behavioural inhibition, was similar between the two groups for all the no-go stimuli except when the target was a happy face. The DCD group made significantly more errors, and failed to withhold responses to happy no-go faces on more than half of the trials. This result suggests that poor affective decision-making of children with DCD on a hot EF task (i.e., the IGT) could be attributed to their heightened sensitivity to emotionally and motivationally significant stimuli, and their reduced inhibitory control in emotionally rewarding contexts. I suggest the interaction between emotion

processing and cognitive control networks underlies this deficit (Rahimi-Golkhandan, Steenbergen et al., 2014). More generally, these results suggest that what characterises the performance pattern of children with DCD may be a deficit of emotion regulation.

The effects I observed, however, may have been moderated by the choice of no-go stimuli—happy faces were used as no-go stimuli in one condition, with fearful faces as the go stimuli. Being intrinsically rewarding, the presentation of happy no-go faces on 30% of the trials may have created an approach bias (or tendency to respond) that also influenced responses to go stimuli (Hare, Tottenham, Davidson, Glover, & Casey, 2005). For instance, RT to a fearful go face might be quicker immediately after exposure to a happy no-go face. Because sensitivity to rewarding stimuli was shown to be heightened in children with DCD, this "priming effect" could be enhanced, masking real differences between groups in their response to 'negative' stimuli. Moreover, commission errors of the DCD group to fearful faces could be partly due to a built-up tendency to respond to happy go targets, and not necessarily be an indication of incorrectly approaching 'negative' stimuli. Therefore, a more systematic method to investigate approach (or appetitive) tendencies is to pair emotional facial expressions with neutral stimuli.

As well, children younger than 10 years of age often fail to identify a fearful face, even at peak intensity (Gao & Maurer, 2009), while adult-like levels are reached by 5 years of age for happy and sad faces. Intensity here is defined as the degree of displacement of facial muscles from a neutral state (Hess, Blairy, & Kleck, 1997). While children may identify 'fearful' as an expression different to 'neutral', they may not perceive it as a negative expression. Moreover, a fearful expression is often perceived as more positive than a sad face (Dennis, Malone, & Chen, 2009). Tottenham, Hare, and Casey (2011) reported similar findings in that the RT to fearful faces was only higher than RT to happy faces. Fearful faces were approached faster than angry faces, and significantly faster than sad faces. Generally,

the more positive a facial expression, the faster is the RT to it. Taken together, sad faces are a better representation of a negative stimulus in a go/no-go task for children, particularly for those with DCD who have associated emotion recognition deficits (Cummins, Piek, & Dyck, 2005).

In the study presented here, I optimised the assessment of sensitivity to reward by pairing emotional expressions with neutral ones, and enlisting negative stimuli (i.e., sad faces) that were clearly discriminable by children. Therefore, the first of my two aims was to investigate potential differences between TD and DCD groups in the tendency to respond to negatively valenced stimuli. I predicted no group difference in line with earlier behavioural data which showed that the deficit in inhibition related specifically to rewarding (no-go) stimuli. My second aim was to measure sensitivity to reward in DCD using a more sensitive metric. In line with earlier findings, I predicted that children with DCD would show higher commission errors than the controls in response to happy no-go faces, specifically. This would provide evidence that these children show a heightened sensitivity to reward, which suggests reduced inhibitory control in response to emotionally-laden stimulus events.

5.2 Method

5.2.1 Participants. The total sample comprised 36 children, 12 with DCD (4 boys, 8 girls; *M* [*SD*] age = 9.82 [1.44]) and 24 TD (10 boys, 14 girls; *M* [*SD*] age = 10.25 [1.62]). The age range of children was between 7 and 12 years, and there was no significant difference between the groups' average age ($p = .44$). All participants were selected from a group of primary school students who took part in my earlier studies of hot EF in DCD. Data for the current study were collected 6 months after the first go/no-go study (Rahimi-Golkhandan, Piek et al., 2014). Children with DCD were identified by parents and teachers as having reduced levels of movement skill that interfered with day-to-day functional activities (Criterion B). Parents confirmed that these motor problems were evident before school age

(Criterion C). Motor difficulties, reported earlier by parents and/or teachers, were confirmed by a score of 80 or below on the Neurodevelopmental Index (NDI) of the McCarron Assessment of Neuromuscular Development (MAND; McCarron, 1997) (Criterion A); *M* [*SD*] NDI = 77.58 [7.64]. No child with a diagnosed intellectual disability, visual impairment, or neurological condition affecting movement was included in this study (Criterion D). Given that all children were attending mainstream primary schools and none were attending remedial classes for literacy or mathematics, I considered that children were sufficiently matched on intelligence at the group level; as such, the effects of intelligence on potential group differences in EF could be ruled out in the current study. The control group was made up of children with NDI of at least 100 (*M* [*SD*] NDI = 109.21 [9.67]). We also excluded those who had a prior or current diagnosis of other developmental disorders (e.g., autism, Attention Deficit/Hyperactivity Disorder (ADHD)). The study was approved by institutional research ethics committee and all children and their parents gave informed consent to participate.

5.2.2 Materials.

5.2.2.1 Go/no-go task. The emotional go/no-go task was based on the work of Ladouceur et al. (2006) and was programmed in E-Prime software (Schneider, Eschman, & Zuccolotto, 2002). The task included pictures of neutral/calm, happy, and sad facial expressions of a group of men and women. All pictures were borrowed with permission from the website of the Sackler Institute for Developmental Psychology (https://www.sacklerinstitute.org), were in black and white, and had the same size and luminance. The task had four runs, divided into two blocks: in each single run neutral expressions were paired with either sad or happy faces. Each run had 28 go and 12 (or 30%) no-go trials, presented in a pseudorandom order. The go target in one run would become the no-go target in the next, with the order of blocks and runs counterbalanced over the

participants. Before the start of each run, children received clear instructions on the screen about the facial expression that would serve as the go target, and were asked to respond (by pressing the spacebar) as quickly as possible to only that particular expression, and not the other. To make sure children understood the task, they were asked to take part in 12 practice trials before the onset of each run. Each picture (10 x 13 cm) was displayed at the centre of a 13" laptop screen for 500ms, followed by a 1500ms inter-stimulus interval during which a white fixation cross was presented centrally. Therefore, for each trial, responses were permitted up to 1500ms following stimulus presentation. Omission errors and RT (to the go targets) as well as the commission errors (to the no-go stimuli) were the main dependent variables of performance in this task. Commission errors index behavioural inhibition; low errors reflect a well-developed ability to withhold responses to no-go targets. Omission errors, the measure of attention, were defined as a failure to respond to go targets, and reflect the tendency to approach each stimulus type (Tottenham et al., 2011). Similarly, RT to the go targets was also used to measure the tendency to approach go stimuli; specifically, approach tendency was assessed as a function of the emotional valence of the stimulus.

In order to confirm that the two groups were equally adept at discriminating between facial expressions, d', which is a widely used measure of perceptual sensitivity, was calculated. Low omission errors (i.e., high approach rate) do not necessarily reflect a more developed ability to recognise the stimuli. For example, if a child presses the spacebar constantly in response to both go and no-go faces, omission errors would be negligible; however, commission errors would be maximal. This pattern indicates poor perceptual sensitivity to the target. The d' index provides a highly valid measure of sensitivity by combining the likelihood of correctly detecting go stimuli with the likelihood of commission errors (aka false alarms), and is calculated as follows: $d' = z(H) - z(F)$, where $z(H)$ is the standardised score for correct hits, and $z(F)$ is the standardised score for false alarms. This

index takes into account the respondent's bias and provides an index of emotion recognition (Tottenham et al., 2011) where high values indicate a more developed ability to discriminate facial expressions as go and no-go stimuli.

5.2.2.2 McCarron Assessment of Neuromuscular Development (MAND). The MAND (McCarron, 1997) is a test of motor ability that can be administered to individuals from 3.5 to adulthood. The MAND comprises 10 items, including five fine-motor and five gross-motor. A summary of performance, or NDI, is derived from the sum of scaled scores on the 10 tests, relative to age norms, and conforms to a normal distribution with a mean of 100 and SD of 15. The psychometrics for the MAND are good. Test-retest reliability of sub-tests over a 1-month period ranges between .67 and .98 (McCarron, 1997). Good construct validity has also been reported in Australian (Hands, Larkin, & Rose, 2013; Tan, Parker, & Larkin, 2001) and US populations (McCarron, 1997).

5.2.3 Procedure. All testing sessions were conducted during children's class time, but in a separate room, free from distractions. In order to control for order effects, half of the children were first assessed on the MAND, while the others took part in the go/no-go task first. Each child took approximately 15 minutes to complete the experimental tasks.

5.2.4 Data analysis. Based on the prior observation of strong group effects on measures of cold and hot EF (Wilson et al., 2013; Rahimi-Golkhandan, Steenbergen et al., 2014), a sample size of 12 children per group was sufficient to achieve a recommended power level of .80 (Faul, Erdfelder, Buchner, & Lang, 2009). When based on previous data using the same or similar paradigm, mixed designs of this type are particularly efficient in testing hypotheses at the group level (Cohen, 1988). In lieu of my specific hypotheses, I further optimised statistical power by running a small set of parametric planned contrasts on the main dependent measures – commission errors, omission errors, and RT. Gender was included as a covariate in all group comparisons to control for differences in gender ratio. For

commission errors, planned comparisons were conducted for happy and sad faces. These analyses addressed the question of whether reduced inhibitory control affects the performance of children with DCD in response to both positive and negative no-go cues or is specific to rewarding stimuli (i.e., positive facial cues). Individual differences in commission errors were also analysed within each group. Omission errors and RT to go stimuli were also compared between groups using planned contrasts to better understand approach tendencies to 'go' stimuli, particularly negatively-valenced ones. To temper the interpretation of significance tests, effect sizes and their 95% confidence intervals (CI) were calculated for all group comparisons on the above outcome variables. We use the abbreviations, 'neutral (H)' and 'neutral (S)' to refer to the stimulus runs in which neutral faces ('go') were paired with happy and sad faces ('no-go'), respectively.

5.3 Results

5.3.1 Commission errors. Figure 5.1 shows mean commission errors for each group.

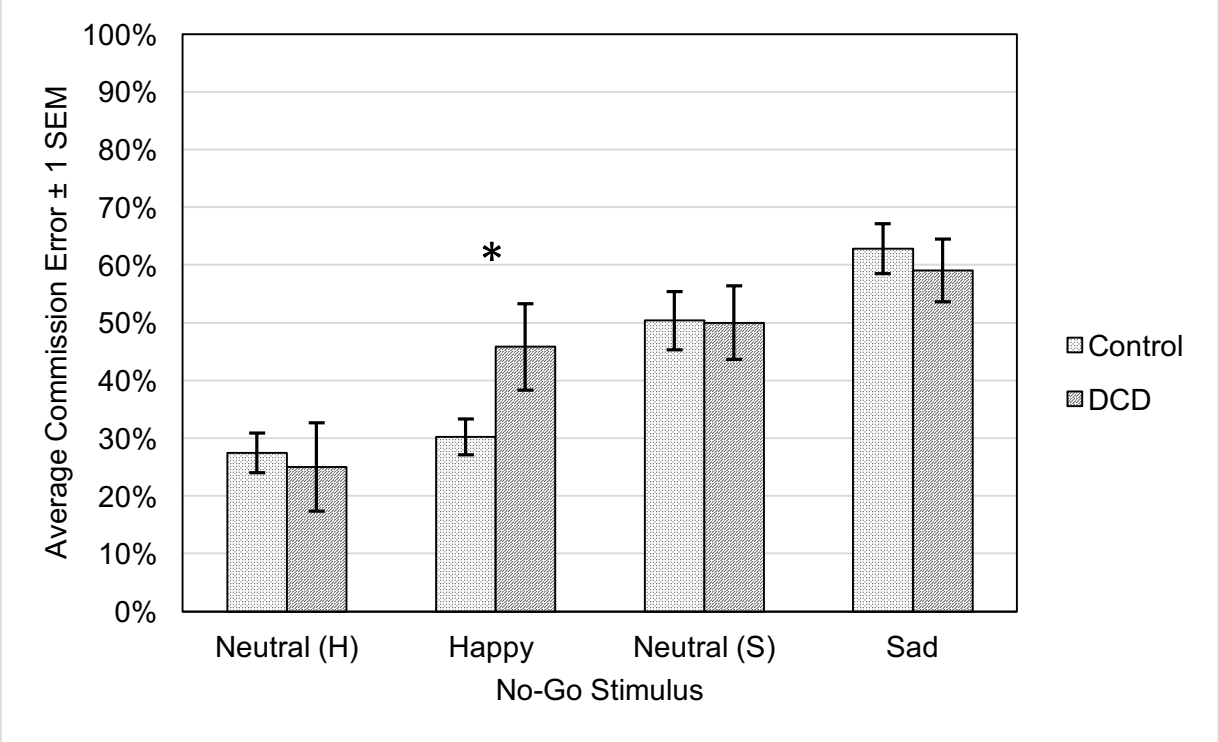

Figure 5.1. Mean (±SE) commission errors to no-go targets in the DCD and control groups (*$p < .05$)

The planned comparison between groups confirmed that the DCD group made significantly more commission errors than controls for happy faces (p = .019) while the comparison for sad faces was not significant (p = .70). Figure 5.2 (left panel) confirms that effect sizes varied as a function of stimulus type.

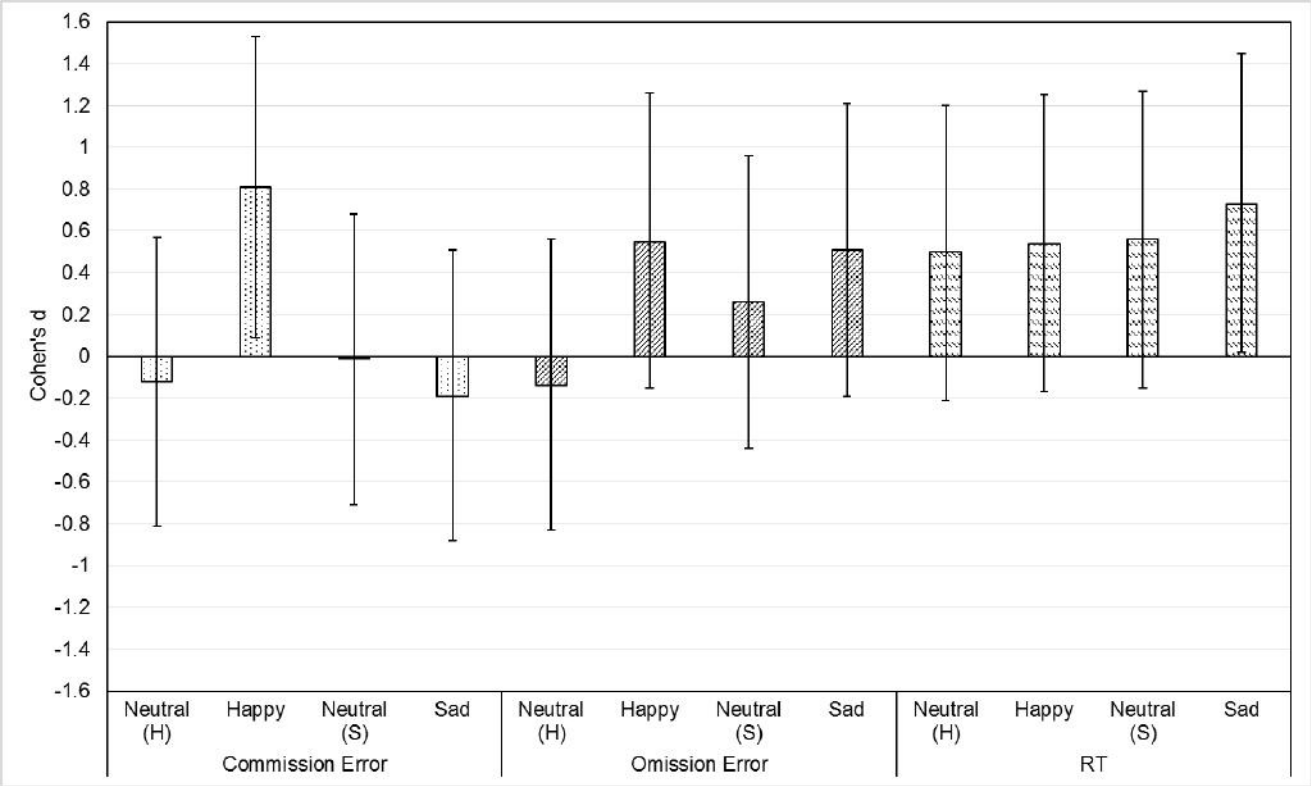

Figure 5.2. Effect sizes (Cohen's d ± 95%CI) for group differences. Positive values indicate more commission and omission errors and slower RT for children with DCD.

Within-group comparisons revealed a similar pattern for each group. For controls, the average commission error to sad faces was higher than that of any other no-go stimulus (0.46 < d < 1.88). For the DCD group, errors for sad no-go stimuli were higher than those for neutral (H) (d = 1.56) and happy (d = 0.68) faces, but not neutral (S) faces (p = .07, d = 0.58). Finally, both groups had significantly more errors to the neutral (S) stimuli than neutral (H) (DCD: p = .003, d = 1.06 (95% CI [0.33, 1.76]); control: p < .001, d = 0.82 (95% CI [0.35, 1.28])).

5.3.1.1 Individual differences. Figure 5.3 presents the number of commission errors to happy no-go targets for each child, divided by group. Almost half (n = 5) of the DCD group approached happy no-go faces on more than 50% the trials, compared with only 1 of 24 (or 4%) controls; all five children with DCD recorded errors above the upper limit of the 95% CI for controls. Alternately, four children with DCD had fewer errors than the average for controls.

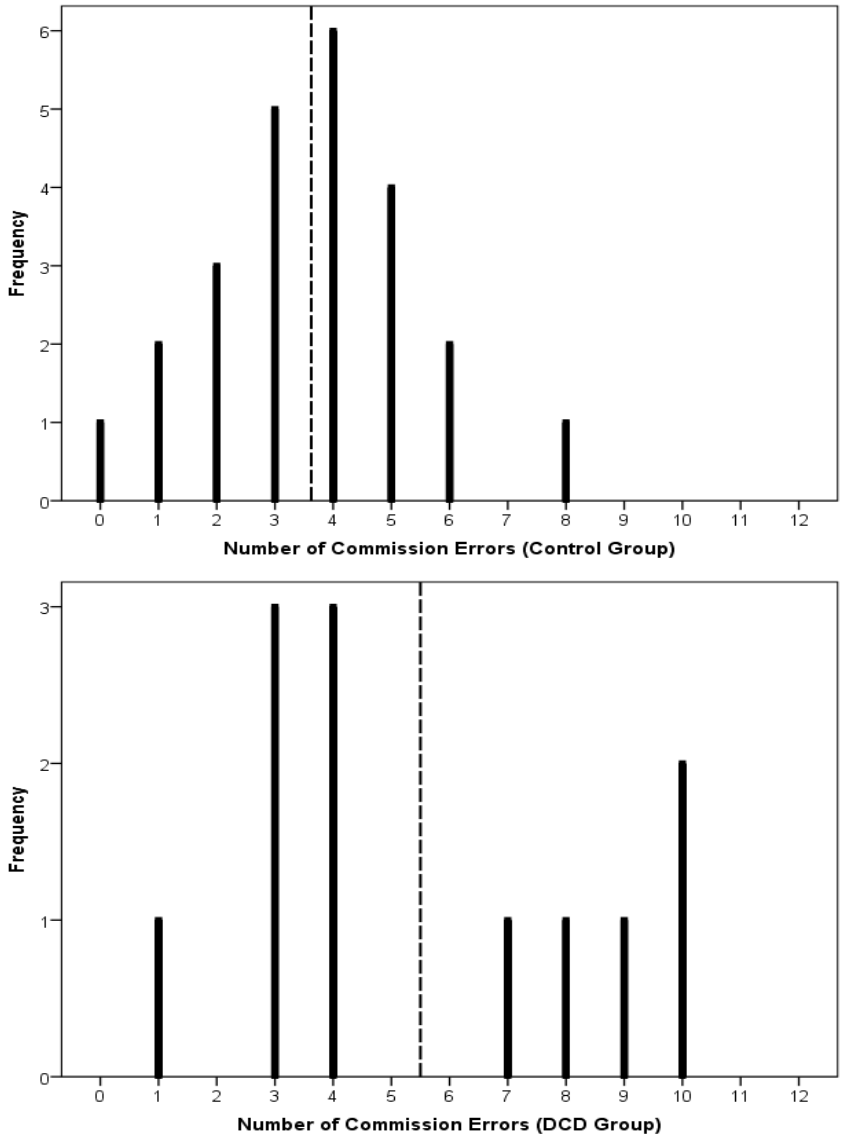

Figure 5.3. Frequency distribution of commission errors to happy no-go targets. The dotted line represents group average. Maximum possible number of commission errors was 12 (trials).

5.3.2 Omission errors. The average proportion of trials that children failed to respond to a go target is presented in Figure 5.4.

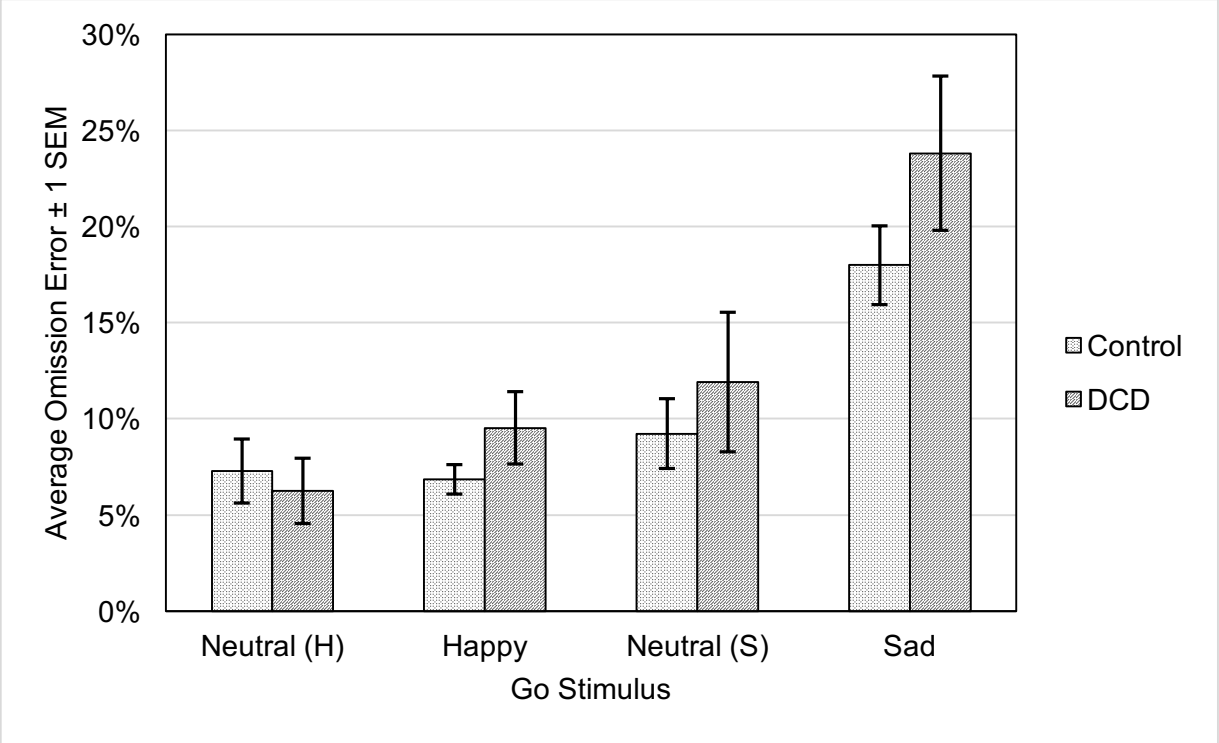

Figure 5.4. Mean (±SE) omission errors to go targets in the DCD and control groups

The DCD group had a lower approach rate to all go stimuli with the exception of neutral faces paired with happy no-go targets. However, there was no statistically significant difference between the groups in response to either happy (p = .12) or sad (p = .18) faces (see Figure 5.2 middle panel for effect size measures). Within-group comparisons showed more omission errors to sad faces than any other stimulus (DCD: p <.003, $d_{\text{neutral(H)}}$ = 1.43, d_{happy} = 0.89, $d_{\text{neutral(S)}}$ = 0.98; Control: p <.002, $d_{\text{neutral(H)}}$ = 0.79, d_{happy} = 1.05, $d_{\text{neutral(S)}}$ = 0.66). More importantly, for the DCD group, omission errors to neutral faces were higher when paired with sad no-go faces (i.e., neutral (S)) compared with neutral (H), p = .045, d = 0.60; the same was not true of controls, p = .32, d = 0.21.

5.3.3 Reaction time. Figure 5.5 shows the average RT (±SE) for go trials in each group.

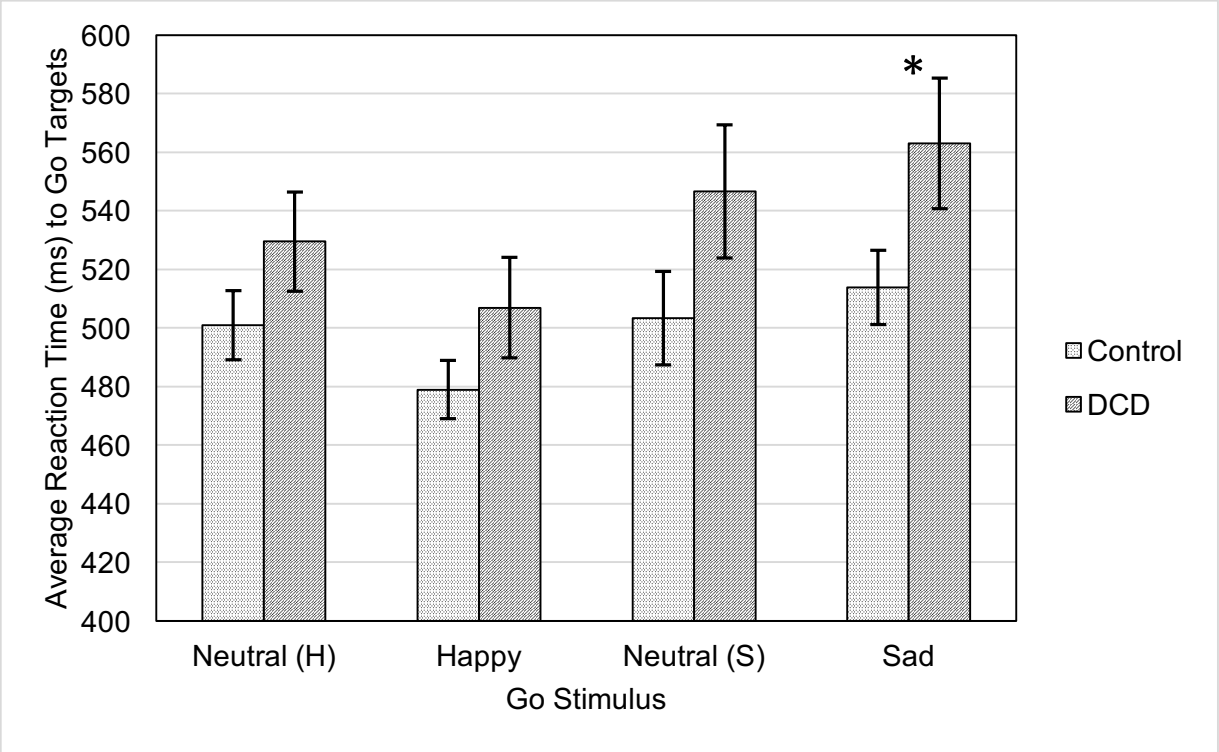

Figure 5.5. Mean (±SE) RT to go targets in the DCD and control groups (*$p < .05$)

There was a significant group difference in RT for sad faces only ($p = .048$). However, effect sizes for other stimuli were also moderate (see Figure 5.2 – right panel). Within-group analyses indicated that the RT of each group to happy faces was significantly faster than their RT to any other go stimulus (DCD: $d_{neutral(H)} = 0.48$, $d_{sad} = 1.03$, $d_{neutral(S)} = 0.86$; Control: $d_{neutral(H)} = 0.64$, $d_{sad} = 0.72$, $d_{neutral(S)} = 0.40$).

5.3.4 D-prime (d'). This measure indicates the degree of sensitivity to each type of facial expression. Figure 5.6 shows that happy faces were the most easily discriminated of the go targets, and sad faces the least. For both groups, the d' for sad faces was significantly less than that for happy faces (DCD: $p < .001$, $d = 1.13$ (95% CI [0.38, 1.84]); Control: $p < .001$, $d = 1.23$ (95% CI [0.69, 1.76])) and neutral (H) (DCD: $p < .001$, $d = 1.28$ (95% CI [0.49, 2.04]); Control: $p < .001$, $d = 1.34$ (95% CI [0.77, 1.88])). Moreover, a comparison of effect sizes showed that both children with DCD and controls had more difficulty responding to neutral faces when they were paired with sad no-go faces than happy no-go targets (DCD: $p = .004$,

d = 0.73 (95% CI [0.07, 1.36]); Control: *p* <.001, *d* = 1.75 (95% CI [1.10, 2.39])). None of the between-group comparisons of d' was significant (see Figure 5.6 for effect size measures).

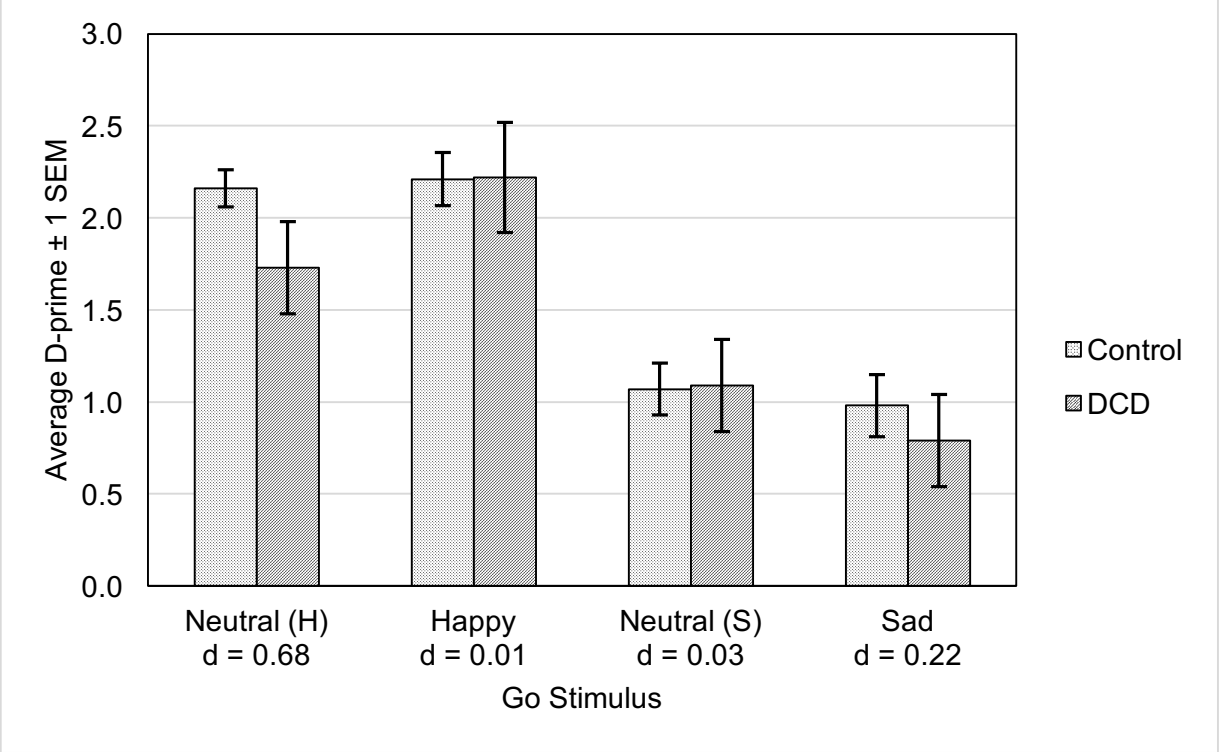

Figure 5.6. Mean (±SE) d' for DCD and control groups in response to the go stimuli

5.4 Discussion

The broad aim of the current study was to investigate hot EF in children with DCD. Specifically, I examined the ability of children with and without DCD to respond to both positively and negatively valenced stimuli in the context of an emotional go/no-go task. Broadly, the pattern of responses confirmed my working hypothesis that children with DCD have difficulty modulating their approach to rewarding stimuli when the task demands that this behaviour be inhibited. In the discussion that follows, I first interpret group differences in the tendency to approach 'negative' stimuli and, second, the finding of reduced response inhibition in DCD when responding to emotionally salient (and 'positive') stimuli. The

discussion then turns to the broader implications of these results and those presented in my earlier studies of hot EF in DCD, for theory development and clinical practice.

5.4.1 Approaching negative social cues. In the current study, slower RTs to sad faces in DCD could be interpreted as a reduced ability to control affective interference and approach 'negative' stimuli. Behavioural responses are biased by affective signals about the rewarding or aversive qualities of a stimulus; however, we are also required to override this immediate bias when required to tackle novel tasks that may be experienced as emotionally challenging or aversive. This ability to ignore the unpleasant aspects of a stimulus and act in a manner incongruent with the affective signal is something that improves gradually with age (Hare et al., 2008).

Developmental factors may explain the difficulty both groups had in distinguishing sad and neutral faces, as shown by low d' values. For each group, the lowest d' was in response to sad faces; the value for neutral (S) was also significantly lower than neutral (H). Children as young as 7 are quite adept in detecting exemplars of facial expressions, but only when these expressions are intense. For example, children are less sensitive than adults to typical expressions of sadness. The confusion associated with recognising a low intensity sad face could bias children to perceive the expression as neutral (Gao & Maurer, 2009). Sad and neutral faces are also considered less arousing than the other expressions (e.g., fearful) (Dennis et al., 2009). Similar emotion recognition difficulties have been reported in developmental disorders like autism (Yerys, Kenworthy, Jankowski, Strang, & Wallace, 2013). Immaturities in younger children's sensitivity to sad and neutral (S) faces may explain the associated d' prime values, as well as high commission and omission errors in response to sad and neutral (S) faces.

5.4.2 Withholding responses to positive social cues. Data reported here corroborated an earlier study showing a reduced ability in DCD to inhibit responses to

emotionally rewarding cues. In general, the DCD group made significantly more commission errors when the no-go stimulus was a positive social cue. Earlier I argued that the mechanism underlying this issue was likely to involve neural integration of inhibitory control and emotion processing systems (Rahimi-Golkhandan, Steenbergen et al., 2014). Disruption to the coupling between these systems would compromise affective decision making (Kohls, Peltzer, Herpertz-Dahlmann, & Konrad, 2009) and self-regulation (Hare & Casey, 2005). Intriguingly, problems with day-to day planning and organisation are persistent issues in DCD, with implications for adaptive behaviour in adolescence and early adulthood (Tal Saban, Ornoy, & Parush, 2014).

Evidence linking motor skill difficulties and EF is based not only on behavioural data but also recent work into the neurobiological mechanisms of motor and cognitive development in both typically and atypically developing children (Johnson, 2011; Koziol, Budding, Chidekel, 2011; Zwicker, Missiuna, Harris, & Boyd, 2011). It is estimated that about half of the children diagnosed with DCD also show reduced EF (Sugden, Kirby, & Dunford, 2008; Willcutt, & Pennington, 2000), particularly deficits of inhibitory control (Wilson et al., 2013). These deficits are evident across a range of tasks and paradigms: inhibition of attentional shifts to compelling but invalid cues (Mandich, Buckolz, & Polatajko, 2002; Wilson, Maruff, & McKenzie, 1997), non-verbal inhibition of motor responses on the Verbal Inhibition, Motor Inhibition (VIMI; Henry, Messer, & Nash, 2012) task (Leonard, Bernardi, Hill, & Henry, 2015), as well as anti-reach movements (Ruddock et al., 2015). Indeed, a direct relationship between the severity of DCD symptoms and deficits of inhibitory control has been suggested (Mandich et al., 2002). Critically, my recent work has shown that EF deficits in DCD are not confined to ‘cold’ aspects of cognitive control, but also affect performance in task contexts that have a strong emotional/motivational valence. In the current study, for instance, deficits of inhibitory control in the DCD group, as

operationalised by commission errors on an emotional go/no-go task, was stimulus-specific: relatively high commission errors were confined to compelling, positively-valenced cues (i.e., happy faces).

5.4.3 The possible neural underpinnings of EF deficits in DCD. Results of the current study can be reconciled within a current neurodevelopmental framework—the theory of interactive specialisation (Johnson & Munakata, 2005). This theory suggests overlap in the development of motor and cognitive functions (Diamond, 2000) and the possibility that disruptions in one domain can have far reaching consequences for another. Of the six major white matter networks (WMN) in the brain, two consistent hubs from early childhood to adulthood include regions across parietal and frontal lobes (Chen, Liu, Gross, & Beaulieu, 2013). A maturational lag or disruption of the fronto-parietal network, which underlies adaptive cognitive control (Sripada, Kessler, & Angsdadt, 2014), may compromise the development of not only EF but other processes arising from specialisation within and interaction between WMNs. More specifically, disruption of the fronto-parietal network would undermine inhibitory control, with implications for motor development and supervisory attentional control (Sripada et al., 2014).

Recent fMRI studies have linked executive control deficits in DCD to inefficient activation of the inhibition network comprising middle frontal, anterior cingulate (ACC) and inferior parietal cortices (Querne et al., 2008), and visuomotor deficits to hypoactivation of parietal regions (Kashiwagi & Tamai, 2013). Maturation of WMNs spanning frontal and parietal sites are also associated with the development of cognitive control in typical children (Casey, Galvan, & Hare, 2005), and response inhibition, more specifically. One possibility is that inhibitory control deficits in DCD may disrupt performance on both traditional tests of (cold) EF and hot EF tasks.

Moreover, WM and inhibition have an interactive relationship such that activation of mental representations relevant to a particular goal-directed task is accompanied by automatic inhibition of irrelevant stimuli and actions (Diamond, 2002; Roberts & Pennington, 1996). Both of these processes are subserved by overlapping neural networks (i.e., DL-PFC, ACC) (Braver et al., 2001; Owen, 2000). Situations that are high in response uncertainty or that present numerous distractors or extraneous stimuli, constitute a high load on EF—this is problematic for children with DCD.

Cognitive re-appraisal strategies have an important role in suppressing or regulating (automatic) responses to highly salient stimuli—those with a strong appetitive or motivational component. For example, mental readying or rehearsal can help prime preferred responses, even in the face of compelling cues (Williams, Bargh, Nocera, & Gray, 2009). These aspects of the cognitive control of action mature with development of DL-PFC (selective attention, WM), and VL-PFC (inhibition) (Ochsner et al., 2012).

5.4.4 Implications for motor learning and control in DCD. In DCD, delayed maturation of executive control networks (spanning PFC and its reciprocal connections to posterior association cortex) may explain broad-based difficulties in the planning, execution, and control of action. More precisely, hypoactivity of the attentional network in DCD including the DL-PFC (Querne et al., 2008) would compromise the initial stages of motor learning and action planning (Brown-Lum & Zwicker, 2015). Further, reduced prefrontal involvement in action planning may contribute to the high comorbidity of attentional problems with DCD (Deng et al., 2014), with consequences for both motor learning and selective inhibition of responses. My current findings suggest that a reduced ability to inhibit responses to emotional cues may bias the child's attention in ways that do not facilitate skill learning. To illustrate, certain movement contexts, especially novel or difficult ones, may be experienced as aversive by children with DCD, especially if their prior learning experiences

have been negative. Conversely, passive or less physically-intense activities may be more appealing or enable the child to avoid negative emotions (like the embarrassment that might be felt when attempting a new motor skill). It is important to think about the process of learning and motor behaviour as something that is inexorably entwined with the experience of emotion. Indeed, most real-world problems enlist a blend of hot and cold EF when action must be planned and implemented with respect to a goal that holds some motivational/emotional significance (Zelazo, 2015).

5.4.5 Implications for intervention. Interventions designed to improve motor skill in children with DCD are more likely to be effective when reasonable adjustments are made for co-development of EF (Reinert, Po'e, & Barkin, 2013). Indeed, a recent review by Diamond (2013) shows that the strongest effect of cognitive training across a range of disorders is shown for children with the most severe EF deficits. This finding and my data underscore the importance of assessing EF in DCD as a standard protocol and then designing interventions that target both motor and cognitive skills, hopefully mitigating the adverse consequences of each (Gonzalez et al., 2014). In the very least, motor interventions may need to modified in delivery to accommodate the reduced level of EF in many children with DCD.

One important aspect of training is the mode of delivery for task instructions. Action simulation (observation plus imagery) using video-based modelling has been shown to be equally effective as traditional physical therapy in promoting skill acquisition in DCD (Wilson, Thomas, & Maruff, 2002). By comparison, verbal instructions may not yield the same training effects in DCD as those seen in TD children, at least with respect to motor imagery (Williams, Thomas, Maruff, & Wilson, 2008). While the reasons for this effect remain unclear, it is possible that task instructions that place excessive demands on WM and/or attention may disadvantage the child with DCD (Wilson et al., 2013). Among other strategies, part-whole learning, providing more opportunities for rehearsal, and increasing

task complexity in a more gradual manner – compared with the rate used for TD children – may also help scaffold the learning process for children with DCD, compensating for reduced EF and a generally slower rate of learning.

Interestingly, preferred approaches to EF training in children involve aspects of physical activity and skill training (Diamond, 2013). Such blended approaches are likely to be particularly effective for children with DCD given the overlap between emotional regulation, cognitive control and movement skill; blended approaches afford ways of breaking down mediation factors that lie between DCD and its psychosocial consequences (Cairney, Rigoli, & Piek, 2013). Task-specific training (like martial arts or dance) presents a potentially powerful means of modifying not only cognitive control, but also the risks posed by inactivity and reduced movement skill. Participation in such training requires that child offset the immediate (emotional) challenge of learning novel tasks in a novel context, by the experience of a different set of valued outcomes (e.g., enhanced participation, the immediate joys of success, enhanced movement capability and improved self-efficacy). Indeed, interventions are more effective through an integrated approach that aims to improve happiness, physical ability, self-efficacy, and feelings of social belonging and support (Diamond, 2012).

5.4.6 Limitations and future research. The heterogeneity of performance within the DCD group on the go/no-go task has previously been reported on related cognitive and motor tasks (Green, Chambers, & Sugden, 2008), particularly when DCD samples include children with other comorbid conditions, either explicitly or in an uncontrolled manner. In the current study, I recruited children without any previous or existing neurological or psychiatric conditions, including other developmental disorders such as ADHD. This raises the possibility that there might be subtypes of DCD based on their motor and cognitive profile – even when no comorbid developmental disorder exists. While a sub-type analysis was

beyond the scope of this study, follow-up studies are encouraged to consider such an analysis, and to extend sampling over a broader age range. Finally, my interpretation of hot EF deficits in DCD was based on behavioural data generated from experimental tasks like the HDT and go/no-go task. Needed now are morphological studies into the development and connectivity of major WMNs in DCD, as well as controlled fMRI evaluations using parametric techniques (Wilson et al., 2013). Together, this work will clarify what appear to be some intriguing points of differentiation in the development of neurocognitive systems in DCD and TD.

5.4.7 Conclusion. The current study shows that EF deficits in DCD are not confined to 'cold' aspects of cognitive control, but also disrupt functioning in 'hot', affective contexts. Disrupted coupling between cognitive control and emotion processing networks, such as fronto-parietal and fronto-striatal networks, may contribute to reduced inhibitory control in DCD. More specifically, emotionally significant and rewarding stimuli constitute a higher load on inhibitory control than neutral or negative stimuli, making it more difficult for children with DCD to modulate their approach tendencies. Reduced inhibitory control in emotionally-laden situations, involving both motor and non-motor activities, would undermine action planning, execution and control, and potentially disrupts self-regulation in DCD; such deficits have been reported in recent studies (e.g., Tal Saban et al., 2014). I suggest interventions that address both motor skills and the co-development of EF are more likely to facilitate skill learning and cognitive control in DCD, with transfer to organisation and execution of daily activities.

Chapter 6: General Discussion

6.1 Overview

The general discussion is divided into five major sections. First, the main theoretical interpretations of the findings are presented. The focus will be on the interaction of cognitive control and emotion processing centres, and, more specifically, how emotionally significant and rewarding stimuli make the tasks more taxing for inhibitory control, particularly for children with Developmental Coordination Disorder (DCD). Second, the possible neural underpinnings of reduced hot executive function (EF) in DCD are discussed. Attention is drawn to the potential involvement of fronto-striatal and fronto-parietal networks. Third, the major implications of deficits of hot EF for children with DCD are reviewed. Reduced inhibitory control in affectively-laden situations, involving motor and non-motor activities, is linked to difficulties with motor functioning, as well as reduced self- and emotion-regulation in DCD. Fourth, the possibility of mitigating EF deficits in DCD is discussed. This section shows how well-established EF interventions could be tailored to children with motor coordination problems, and also identifies the major benefits of such interventions for children with DCD. Finally, an in-depth review of the limitations of the three studies reported in my thesis are provided. Some of these limitations include lack of a formal diagnosis of DCD, small sample size, restricted age range of participants, and so forth. I finish with suggestions for future research.

6.2 Summary of the Findings

The three studies, reported in chapters 3-5, suggest that EF deficits in DCD are not confined to ‘cool’ aspects of cognitive control, but also affect performance in hot, affective contexts. Study 1 shows that children with DCD perform worse than same-age typically developing (TD) controls on the child-friendly variant of the Iowa Gambling Task (IGT; Bechara, Damasio, Damasio, & Anderson, 1994), known as the Hungry Donkey Task (HDT; Crone & van der Molen, 2004). The DCD group opted for high immediate reward options

associated with overall loss. Study 2 links hot EF deficits in DCD to a heightened sensitivity in this cohort to emotionally-significant stimuli. Deficits of inhibitory control in DCD, as operationalised by commission errors on an emotional go/no-go task, was stimulus-specific. Rather than performing poorly on *all* no-go trials, the DCD group had significantly more commission errors only in response to compelling cues (i.e., happy faces). Study 3, which optimised the assessment of sensitivity to reward using a different emotional go/no-go task, once more showed that rewarding stimuli disrupt inhibitory control in DCD. The go/no-go task, however, failed to find any group differences in the ability to approach negatively-valenced stimuli. Next, I discuss the underlying mechanisms for deficits of hot EF in DCD – as shown by the findings of Studies 1-3 – and argue that the interaction of cognitive control and emotion-processing networks underpins reduced inhibitory control of children with DCD in affectively-laden contexts.

6.3 The Interpretation of Findings

This section first discusses two hypotheses (i.e., impaired use of somatic markers, and reduced inhibitory control) that may explain the performance of children with DCD on the HDT. It then discusses the interaction of cognitive control and emotion processing networks as a more comprehensive explanation of performance deficits in the DCD group on *both* the HDT and emotional go/no-go tasks. Lastly, it considers the role of comorbidity and the impact of confounding variables.

6.3.1 Somatic marker hypothesis. The performance of the control group on the HDT, in Study 1, was similar to what has been reported for other TD children (e.g., Huizenga, Crone, & Jansen, 2007; van Duijvenvoorde, Jansen, Visser, & Huizenga, 2010) in that they opted for the two low-frequency loss options (i.e., B and D). Overall, the DCD group had a lower total net score than the controls. Moreover, they selected option B (*high immediate reward*) at a significantly higher than the chance level, unlike controls. This

pattern is similar to adolescents with attention deficit/hyperactivity disorder (ADHD) on the IGT, who also opted for the disadvantageous option B (Toplak, Jain, & Tannock, 2005). It is unclear whether Toplak and colleagues (2005) excluded those with motor coordination problems from their sample. Given the high comorbidity of DCD and ADHD (Pitcher, Piek, & Hay, 2003), the poor performance of the ADHD group on the IGT could be associated with the underlying (and comorbid) motor coordination problems. A similar argument was presented by Piek, Dyck, Francis and Conwell (2007) in their investigation of 'cool' EF when they found that once motor deficits were controlled, there was little evidence of EF deficits in ADHD. Finally, there was no significant difference between the reaction time (RT) of the controls to disadvantageous and advantageous options; however, children with DCD had a significantly faster response to the disadvantageous (*high immediate reward*) options. The performance of the DCD group on the HDT, and their persistence on approaching the disadvantageous options throughout the 100 trials of the HDT indicate a reduced ability to use feedback from earlier trials and anticipate the negative outcome of opting for high immediate reward choices.

The Somatic Marker Hypothesis (SMH; Damasio, 1994), introduced in chapter 4, provides a useful model for interpreting the performance of children with DCD on the HDT. In short, the SMH suggests that under complex conditions where a logical cost-benefit analysis of all response options is difficult (e.g., as on the HDT where the characteristics of the options are not disclosed to children, and they need to utilise feedback over 100 trials to infer differences across options), decision-making is biased by emotion-based signals and sensations that arise from the body and/or the central representation of it (Damasio, 2004). These 'somatic markers' then signal the likely outcome of an action and the emotions associated with it before the overt action unfolds. These signals, which are integrated within the emotion circuitry of the brain, are used to create a forward model of changes expected to

happen in the body. The forward model then facilitates rapid responses to the stimuli (Dunn, Dalgleish, & Lawrence, 2006). For example, optimal performance on the IGT has been linked to an increase in anticipatory skin conductance response just before approaching the disadvantageous decks (Bechara, Tranel, Damasio, & Damasio, 1996).

The process by which somatic markers facilitate decision-making is similar to forward modelling of motor control of the periphery (Wilson et al., 2004; Wolpert & Ghahramani, 2000), and suggests the decision to either approach or avoid a stimulus is based on the perception of the somatic states associated with each action. In addition to ventromedial prefrontal cortex (VM-PFC), the neural circuitry underlying this process includes brain regions involved in the regulation and representation of the body-state, namely somatosensory cortices, basal ganglia, and insula (Damasio, 1998); dysfunction within these regions disrupts the use of somatic markers, and results in deficits of affective decision-making.

Interestingly, research has reported deficits in the predictive control of action in DCD (Wilson, Ruddock, Smits-Engelsman, Polatajko, & Blank, 2013), as well as dysfunction of the regions involved in the representation of the body-state, such as basal ganglia and insula in possible subgroups of children with DCD (Lundy-Ekman, Ivry, Keele, & Woollacott, 1991; Zwicker, Missiuna, Harris, & Boyd, 2011). For instance, one causal model of DCD is the internal modelling deficit (IMD) hypothesis (Wilson et al., 2004). According to this hypothesis, an underlying cause of movement difficulties in children with motor coordination problems may be their poor ability to generate and/or utilise internal models of motor control accurately (Williams, Thomas, Maruff, Butson, & Wilson, 2006). In neurocomputational models (e.g., Wolpert, 1997; Shadmehr & Krakauer, 2008), internal modelling takes two complementary forms: The inverse model, or controller, generates the motor commands necessary to achieve a desired end-state that is specified in spatial coordinates, while the

forward model utilise a copy of these commands to internally simulate/predict the future state of limb movements or body position. If the forward model does not match the goal state, error correction signals are sent from the cerebellum to the motor-planning system (the inverse model) to modify the components of action such as force and/or timing (Williams et al., 2006; Zwicker, Missiuna, Harris, & Boyd, 2012). However, an impaired ability to internally represent actions and mentally simulate movement – via forward models – results in problems with predicting the consequences of motor behaviour, organising motor responses, and monitoring/modifying action. Forward modelling deficits in children with DCD diminish their ability to modify existing internal models, notably increase the time they need to build an adequate model for action, and disrupt their learning of motor skills. Converging evidence from different lines of research supports the argument that predictive control of action is indeed a core deficit in DCD (Wilson et al., 2013).

Difficulties in generating internal models for action or modifying existing ones may be linked to an impaired ability to *predict* the consequences of selection decisions on the HDT. The default response to highly rewarding stimuli, which are tempting by their nature, is 'approach'. However, on a delay of gratification paradigm – as in the HDT – one needs to revise this default action, and predict the likely consequences of opting for immediate rewards (i.e., disadvantageous options). Somatic markers facilitate the ability to create this *forward* model of action. Thus, one speculation is that deficits of hot EF in children with DCD, operationalised as their performance on the HDT, could be, at least partly, explained by an impairment in the use of somatic markers, which in turn increases the chances of making disadvantageous decisions such as choosing high immediate reward at the expense of later loss. Deficits in predictive control of action may be more generalised than just real-time motor control.

On the other hand, reduced use of somatic markers has been linked to an increased rate of selecting *both* disadvantageous options on the IGT – for instance, in patients with VM-PFC lesions. Study 1, however, showed that the DCD group opted for only one of the disadvantageous options: Option B which is associated with high immediate reward, but also low frequency loss. Children with DCD did not choose option A at a rate higher than the chance level. Dunn, Dalgleish, and Lawrence (2006) suggested that this particular pattern of performance is indeed a reflection of a reduced complexity of rule use, rather than impaired use of somatic markers. To illustrate, children only consider frequency of loss, and opt for options B and D, because they have equal frequency of loss. However, adolescents and particularly adults also consider the amount of loss, and only opt for D. In the case of DCD, heightened sensitivity to rewarding stimuli is likely to increase the tendency to select option B, only. The mechanism explaining this pattern of performance is likely to involve the network of structures that bind motor, cognitive and affect control in DCD.

6.3.2 Deficits of inhibitory control. In addition to the possible impact of somatic markers on affective decision-making, reduced inhibitory control may also contribute to deficits of hot EF (Hodel, Brumbaugh, Morris, & Thomas, 2015) in DCD. While SMH may explain performance on the HDT, reduced inhibitory control could be the mechanism affecting performance on *both* the HDT and the two emotional go/no-go tasks. Inhibitory control is not only relevant to the other cool EFs (i.e., WM, and mental flexibility), but also plays a crucial role in hot EF (e.g., the ability to resist temptation and delay gratification) and advantageous decision-making (Diamond, 2013; van Duijvenvoorde, Jansen, Bredman, & Huizenga, 2012). The HDT, for instance, taps into both the reward and inhibitory circuitry of the brain controlled by the PFC and amygdala (Ernst et al., 2002; Li, Lu, D'Argembeau, Ng, & Bechara, 2010; Smith, Xiao, & Bechara, 2012). Successful performance on the HDT relies on the activation of VM-PFC, as well as different regions such as dorsolateral prefrontal

cortex (DL-PFC), anterior cingulate, the cerebellum, the insula, and the inferior parietal cortex (Ernst et al., 2002). Some of these areas (e.g., anterior cingulate) are also activated during inhibitory control tasks (Braver, Barch, Gray, Molfese, & Snyder, 2001), and there is evidence for cerebellar dysfunction and hypoactivity of the insula in DCD during motor performance (Geuze, 2005; Zwicker et al., 2011). Children with motor coordination difficulties also show inhibitory control deficits; for instance, inhibition of attention to compelling but invalid cues (Wilson, Maruff, & McKenzie, 1997), or manual response inhibition on a visual variation of the traditional Simon task (Mandich, Buckolz, & Polatajko, 2002).

Moreover, difficulty in *reversal learning* has been linked to performance deficits of some clinical groups on the IGT, and by extension its variants such as the HDT. For instance, a number of studies have found that patients with VM-PFC lesions, who often show impaired IGT performance, also perform poorly on tasks of simple reversal learning (Fellows & Farah, 2003; Rolls, Hornak, Wade, & McGrath, 1994). However, these patients performed like controls on a modified version of the IGT in which disadvantageous decks no longer had an initial advantage (Fellows & Farah, 2003). This suggests that difficulty in reversing early learning – understood as a deficit in response inhibition and mental flexibility (Rescorla, 1996) – explains impaired IGT performance. In a similar vein, children with DCD persisted in their choice of disadvantageous options and failed to reverse their initial perception of the high immediate reward options. In summary, the involvement of specific neural structures and networks that have been implicated in both the HDT performance and motor coordination, along with a generalised deficit of response inhibition which disrupts reversal learning on the HDT may underlie poor performance of the DCD group on the HDT.

6.3.3 Interaction of cognitive control and emotion processing networks. Chapters 4 and 5 show that children with DCD had significantly more commission errors than controls

to happy no-go faces on the emotional go/no-go tasks, regardless of whether these positive social cues were paired with negative or neutral facial expressions. The reduced ability of the DCD group to suppress responses in the go/no-go tasks was stimulus-specific, indicating that this group has heightened sensitivity to emotionally significant stimuli. Successful regulation of responses in an emotional go/no-go task relies on both emotion discrimination skills and cognitive control abilities (Tottenham, Hare, & Casey, 2011). Accordingly, high commission errors by the DCD group could be due to one or both of these factors.

6.3.3.1 Reduced emotion discrimination. A reduced ability to recognise and respond to static and changing facial expressions of emotion has been reported in DCD (Cummins, Piek, & Dyck, 2005), which suggests that high commission errors to happy faces on the go/no-go task may reflect visuospatial processing deficits, affecting their ability to detect, code, and process facial expressions. It follows that difficulties processing facial expressions coupled with deficits of response inhibition in DCD may lead to more false alarms. However, even though children with DCD might not be as quick as TD children in processing facial expressions, the DCD group showed roughly similar levels of omission and commission errors to both the negative and the neutral stimuli. Moreover, faster RT of both groups to happy faces is in line with the argument that happy facial expressions are perceived as rewarding stimuli and are instinctively approached faster than negative expressions (Schulz et al., 2007). This shows that the emotional valence of the positive stimuli was apparent to both groups. There were also no significant group differences in omission errors, and both groups had comparable sensitivity (d') to the happy faces. Therefore, commission errors of the DCD group to the happy no-go targets cannot be attributed to incorrect perception of some of the happy faces as negative (chapter 4) or neutral (chapter 5).

6.3.3.2 A generalised deficit of cognitive control? DCD has been also linked to a disrupted regulation of PFC (Querne et al., 2008) as well as other areas, such as parietal

regions (Kashiwagi & Tamai, 2013) that are linked to the development of cognitive control (Casey, Galvan, & Hare, 2005). However, the fact that the DCD and control groups had similar levels of commission errors to all the stimuli except happy faces indicates that a general deficit of cognitive control, particularly behavioural inhibition (i.e., the ability to inhibit a response, regardless of the type of stimulus), may not explain the stimulus-specific differences in commission errors between the two groups.

6.3.3.3 Task difficulty mediates the interaction of control and emotion processing networks. Tasks that require responses to motivationally significant and rewarding stimuli add a layer of difficulty to performance and disrupt the ability to suppress naturally occurring, prepotent responses (Kohls, Peltzer, Herpertz-Dahlmann, & Konrad, 2009). For instance, happy faces or high immediate rewards reliably activate emotion processing networks (Canli, Sivers, Whitfield, Gotlib, & Gabrieli, 2002; Killgore & Yurgelun-Todd 2001; Yang et al., 2002). This activation makes the task of withholding responses to motivationally significant stimuli more difficult because it requires greater activity in the inhibitory control network – compared to when abstract, decontextualised stimuli are presented (Hare, Tottenham, Davidson, Glover, & Casey, 2005). Adolescents, for instance, are more prone than adults to the interference of emotional information in their goal-directed actions. The interaction of relatively mature, highly active emotion centres with a developing prefrontal cortex (PFC) during adolescence results in impulsive reward-driven performance (Smith, Xiao, & Bechara, 2012) and an 'emotional overshoot' – characterised by an increase in emotional responding – in that the activity of emotion centres is not properly controlled by the inhibitory processes (van Duijvenvoorde, Jansen, Visser, & Huizinga, 2010). Adolescents' poor performance on hot EF tasks (e.g. the IGT) has been linked to a neural imbalance between the development of emotional responding and control processes (Casey, Getz, & Galvan, 2008) in that an immature PFC fails to properly modulate the output of

highly active emotion centres. Adolescents can indeed suppress response to compelling stimuli; however, to do that they need to recruit prefrontal regions more than adults do; so to compensate for their relative deficits in cognitive control (Luna & Sweeney, 2004).

Developmentally, many of the age-related differences in EF emerge when the task becomes more taxing. For instance, TD 3-year old children performed significantly worse than their 4-year old peers on a 'Less is More' task in which they had to point to a small reward (e.g., two jelly beans), instead of a larger one (e.g., five jelly beans) in order to get the large reward. The group differences disappeared when the rewards were replaced with abstract, symbolic representations of those rewards (Carlson, Davis, & Leach, 2005).

In a similar vein, the effects of deficits of cognitive control in DCD may be exacerbated when rewarding and emotionally-significant stimuli are presented – as in the HDT and emotional go/no-go task. Study 1 showed that the DCD group were more likely than the controls to approach highly rewarding stimuli. In Studies 2 and 3, children with DCD performed as well as controls in withholding responses to neutral or negative targets, and had similar levels of omission errors for all the stimuli. Deficits of response inhibition in DCD emerged only in response to compelling stimuli on the go/no-go task. A large body of literature exists on deficits of cognitive control and disrupted prefrontal regulation in DCD (e.g., Kashiwagi & Tamai, 2013; Wilson et al., 2013). Thus, in the case of DCD, emotionally significant stimuli, which increase cognitive demands, put more pressure on control networks, and result in significantly lower response inhibition in affectively-laden situations. This suggests that the reduced response inhibition of children with DCD to emotionally significant stimuli is more likely due to an interaction between the salience of the rewarding stimuli, which increases task difficulty, and the deficits of cognitive control in this cohort. Previous research indicates the reduced regulation of emotion processing by control networks

creates an imbalance between these two systems, and increases the risk for the onset of affective disorders (Ernst, Pine, & Hardin, 2006; Spear, 2000; Steinberg, 2005).

6.3.4 The impact of comorbid conditions. An alternate explanation for reduced hot EF in DCD could be the co-occurrence of conditions that are also associated with executive dysfunction – namely, ADHD. Existing literature suggests that comorbidity exacerbates DCD symptoms (e.g., motor impairment and risk for psychosocial maladjustment), and that the number of co-occurring conditions is associated with the severity of symptoms (Crawford & Dewey, 2008; Jongmans, Smits-Engelsman, & Schoemaker, 2003). To illustrate, a comorbid diagnosis of DCD and ADHD has been linked to more severe depressive symptoms (around 3-5 times) than a single diagnosis of either DCD or ADHD (Missiuna et al., 2014; Piek, Rigoli, et al., 2007). Indeed, comorbid DCD and ADHD, particularly the hyperactive subtype, may exacerbate hot EF deficits. Yerys, Kenworthy, Jankowski, Strang, and Wallace (2013) found that reduced inhibitory control to emotionally significant stimuli in children with autism – operationalised as their commission errors on an emotional go/no-go task – was positively related to ADHD symptoms of hyperactivity and impulsivity. Although these findings raise the possibility that comorbidity (with ADHD) may underlie the results reported in my thesis, no child with a previous or existing neurological or psychiatric condition, including other developmental disorders, was recruited in Studies 1-3 (based on parent reports). Thus, it is possible that reduced (hot) EF in some of these children could be due to co-existing conditions not reported or identified by their parents or guardians.

6.3.5 Controlling confounding variables. In each of the three studies that I reported in this thesis, one or more confounding variables – that could explain the pattern of results – were controlled, either through the design of the tasks or the analyses. *First*, both the HDT and the emotional go/no-go task minimise the potential confounding effect of motor control deficits on the performance of the DCD group in hot EF tasks. Both tasks make few demands

on motor performance by enabling children to use only their dominant hand to attempt the task. Even though there are four response options in the HDT, the task does not have any speed requirement; therefore, children can use one finger to respond. In a similar vein, the emotional go/no-go tasks also require participants to only interact with the spacebar on a laptop keyboard. *Second*, WM, which has been linked to the IGT/HDT performance in some studies (e.g., Hinson, Jameson, & Whitney, 2002), was measured in Study 1 using the One-Back task. However, the results found no significant relationship between WM and the HDT outcome measures in both DCD and control groups. *Third*, given the differences in the ratio of boys and girls in DCD and control groups, gender was considered as a covariate in preliminary analyses; however, the results revealed no significant covariate effect for this variable. To illustrate, the inclusion of gender did not lead to any notable changes in *p* values or effect size indices. *Fourth*, given that none of the children, who were students in three mainstream primary schools, were attending remedial classes for literacy or mathematics, I inferred that the groups were sufficiently matched on intelligence. *Finally*, one may suggest that variables such as language, perceptual organisation, social cognition and the like should be considered as covariates, too. However, treating these variables as covariates would cloud the very classification of children with DCD (or TD children), and, as such, obfuscate research hypotheses; therefore, it is neither possible nor customary to control for such variables.

6.4 Neural Underpinnings of Hot EF Deficits in DCD

6.4.1 Different patterns of brain activation. There is minimal evidence for overt structural differences in the brains of children with DCD; however, neuroimaging studies have linked functional difficulties of this cohort to different patterns of brain activation compared with their TD peers (Zwicker, Missiuna, Harris, & Boyd, 2010; Zwicker et al., 2011). Response inhibition on a standard, ‘cool’ go/no-go task requires coordinated

activation of the frontal and parietal regions as well as the anterior cingulate. Querne and colleagues (2008) reported that even though children with DCD performed as well as the controls on a 'cool' go/no-go task – which required response to all letters except X – they demonstrated significantly lower prefrontal activity, and higher anterior cingulate activation than TD children. The increase in the activity of the anterior cingulate, which is involved in error detection (Casey et al., 1997; Mathalon, Whitfield, & Ford, 2003), has been associated with higher inhibitory demands, as when the no-go target is an emotionally significant stimulus (Garavan, Ross, Murphy, Roche, & Stein, 2002). This is consistent with the hypothesis that children with DCD find the inhibitory component of go/no-go (and related) tasks more cognitively demanding than TD children, particularly when required to process highly salient or emotive stimuli (e.g., rewards, or positive social cues). Thus, differences in the cognitive control abilities of DCD and control groups are more likely to emerge when the task becomes more difficult because children with DCD may have to rely on an already overtaxed network of brain activation.

6.4.2 Reduced connectivity of white matter networks. Delayed development (i.e., more fine-tuned, focal activation) of prefrontal, posterior parietal, and striatal regions, or disrupted connectivity of the white matter networks (WMN) between them could have significant implications for deficits of cognitive control in both cool and hot contexts (Casey, Galvan et al., 2005; Chen, Liu, Gross, & Beaulieu, 2013; Kohls et al., 2009). Here, I discuss the possible relationship between deficits of hot EF in DCD and reduced functional connectivity in WMNs.

6.4.2.1 Fronto-striatal networks. The ability to resist temptation and delay gratification relies on the activation of fronto-striatal circuitry (Casey et al., 2011). More specifically, the process of withholding responses to salient cues has been linked to increased blood oxygen level dependent (BOLD) signal in fronto-striatal circuits, and the degree of

activation is correlated with the inhibitory load of the task (Hare et al., 2005). The ability to avoid happy faces, for instance, is associated with an increase in BOLD signals in the caudate nucleus – a structure within the dorsal striatum – which has been implicated in impulse control and behavioural inhibition (Casey et al., 1997; Durston et al., 2002; Vaidya et al., 1998). There is an indirect relationship between caudate activity and the number of commission errors to positive social cues. In the case of DCD, reduced functional connectivity between the primary motor cortex and the striatal regions, particularly the caudate (Figure 6.1a), raises the possibility that motor execution regions may receive a degraded input signal from structures involved in motor control (McLeod, Langevin, Goodyear, & Dewey, 2014). Given the compelling nature of rewarding stimuli, the default response is to approach them, unless the activity of motor execution regions is modulated by inhibitory processes. Thus, reduced connectivity within the fronto-striatal networks increases the likelihood that approach tendencies are not modulated properly.

In contrast, strong activation of the ventral striatum, which is involved in the encoding of the reward value of stimuli (Ochsner, Silvers, & Buhle, 2012), has been linked to adolescents' reduced EF in hot, affective contexts (Somerville, Hare, & Casey, 2011). The strong signalling of the ventral striatum in adolescents is not well modulated by the control systems which increases the likelihood of approaching salient, rewarding stimuli. However, this pattern of activation is not specific to adolescents. Generally, reduced ability to withhold responses to compelling stimuli is associated with elevated recruitment of the ventral striatum and hypoactivity of the inferior frontal gyrus (Casey et al., 2011). Additionally, small difference in the recruitment of the inferior frontal gyrus between go and no-go trials by those who make many commission errors to salient stimuli is an indication of a reduced ability to invoke cognitive control in emotionally-laden contexts (Casey et al., 2011). Ventral striatum has not been implicated in networks with disrupted connectivity in DCD; however, reduced

cognitive control in this cohort raises the possibility that control systems may not adequately modulate the activity of ventral striatum (Figure 6.1b), thereby increasing approach behaviours.

6.4.2.2 Fronto-parietal network. Recent literature suggests that appetitive cues increase the excitability of motor system and the likelihood of approaching stimuli (Chiu, Cools, & Aron, 2014). In order to inhibit prepotent responses, control systems should modulate the activation of motor system, and prevent motivational cues from quickly spilling over into the motor system. For instance, reappraisal or reframing strategies (e.g., cooling the hot, appetitive features of emotionally significant stimuli) facilitate the ability to overcome motivational spillover and suppress responses to compelling stimuli (Casey et al., 2011). For example, thinking of a white marshmallow as a cotton ball facilitates delay of gratification in children. However, the coupling of cognitive control and motor planning depends on the integrity of fronto-parietal network, which in DCD has been shown to be hypoactive under certain task conditions (Zwicker et al., 2011). McLeod and others (2014) also identified reduced functional connectivity within the fronto-parietal network; more specifically between the primary motor cortex and parietal areas such as right supramarginal gyrus, and angular gyri. The angular gyri, for instance, act as a multimodal integration hub for internal and external information required for response generation (Binkofski et al., 1999). Disrupted connectivity between this region and primary motor cortex in DCD (Figure 6.1c) raises the possibility that information provided by this region to the primary motor cortex may be degraded, which in turn could impact motor behaviour, such as the decision to approach a stimulus or not.

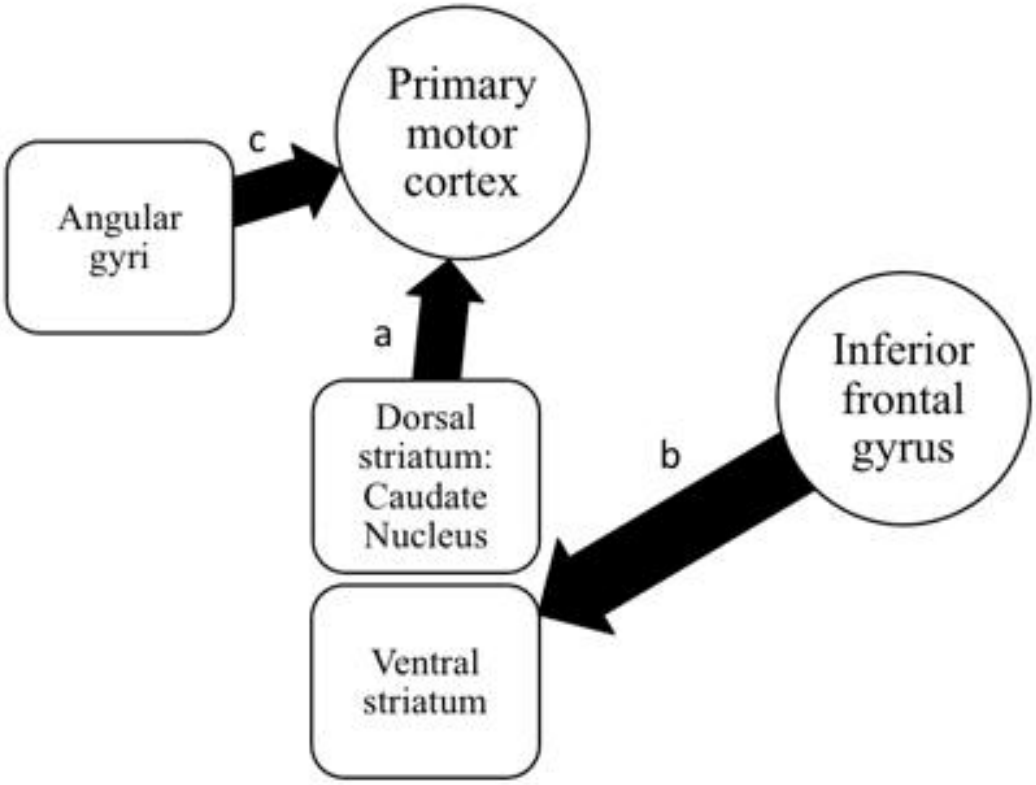

Figure 6.1. The potential involvement of fronto-striatal and fronto-parietal regions in hot EF deficits of children with DCD

6.4.3 Three postulates regarding the neural underpinnings of hot EF deficits in DCD. The information provided in this chapter regarding different patterns of brain activation, and reduced connectivity of WMNs in DCD, as well as those in previous chapters about the brain regions/structures implicated in both hot EF and DCD points to three, not mutually exclusive, postulates for the underlying neural mechanisms of deficits of hot EF in DCD.

One possibility is that reduced hot EF in DCD could be due to the hyperactivity of regions involved in representing the reward value of stimuli (e.g., ventral striatum) and/or the hypoactivity of those contributing to the cognitive control of action (e.g., caudate nucleus). Any one or the combination of these two factors increases the likelihood of approaching

compelling stimuli that should be avoided. *Secondly*, the aforementioned structures could show the same level of activity in TD children and those with DCD, but instead a maturational lag across the frontal regions involved in cognitive control may reduce the ability to adequately modulate the activation of emotion processing centres. For instance, poor performance of young children on the IGT and a focus on high immediate reward (Crone & van der Molen, 2004) has been linked to the protracted development of PFC in this cohort (Casey, Tottenham, Liston, & Durston, 2005). Delayed development of frontal control network, which is highly interconnected with the limbic structures, has also been linked to reduced self- and emotion-regulation (Dennis, Malone, & Chen, 2009). *Thirdly*, heightened sensitivity of children with DCD to rewarding stimuli may reflect a reduced level of coupling between cognitive control and emotion processing networks. Recent literature shows reduced maturation of white matter architecture (i.e., lower fractional anisotropy (FA), and higher diffusion) in DCD compared to TD children (Brown-Lum & Zwicker, 2015). More specifically, the integrity of WMNs in both frontal and parietal areas is impacted in children with DCD, with reductions in FA in parts of the corpus callosum underlying the parietal lobe, as well as in the left superior longitudinal fasciculus (Langevin, MacMaster, Crawford, Lebel, & Dewey, 2014). More importantly, PFC which is involved in working memory (WM) and planning of motor responses also shows reduced functional connectivity in children with single diagnosis of DCD (McLeod et al., 2014).

Therefore, even if one assumes that structures implicated under hypotheses one and two above are functionally intact in both DCD and TD populations, reduced integrity of WMNs in DCD means the coupling of motor and cognitive control regions is degraded (Christoff & Gabrieli, 2000). Rewarding stimuli increase the excitability of motor system, and improve the likelihood of approach behaviour (Chiu et al., 2004). The ability to inhibit the default approach tendency relies on the integrity of neural pathways connecting frontal

control regions to both emotion processing networks and motor system. Disrupted connectivity of WMNs results in degraded inputs from control centres, which translate into reduced inhibitory control in emotionally-laden situations.

It is, however, worth noting that the ability to withhold responses – in both hot and cool contexts – does not only rely on the functional integrity of neural networks or the connectivity among them, but is also influenced by the significance of a task/stimulus and prior learning experiences. In the case of DCD, for example, approach or avoidance tendencies are also influenced by the degree of motor involvement in a task. Given that the DCD literature has provided scant evidence regarding the activity and functional connectivity of the regions implicated in response inhibition – particularly in 'hot' contexts – the validity of the aforementioned hypotheses in this section need to be established in a two-step process: First, replication of hot EF deficits in DCD in different cohorts and into adolescence; and second, functional magnetic resonance imaging (fMRI) studies using parametric techniques as well as morphological studies into the development and connectivity of fronto-parietal and fronto-striatal WMNs in DCD.

6.5 Major Implications of Hot EF Deficits in DCD

6.5.1 Motor functioning. The learning and execution of different motor tasks do not occur in an environmental vacuum, and free from others. Children often have to attempt various tasks in the presence of significant others, including their family members, teachers, and/or peers; and it is essential that they can regulate their emotional reactions and learn to modulate responses to objects and events that are compelling and sometimes irrelevant. In the case of DCD, the outcomes of movement may not always meet with success or the action itself performed fluently, and as a result, movement contexts can be perceived as a source of stress. Therefore, one default response by children with DCD may be to avoid physical activity and take part in more passive tasks (Cairney, Veldhuizen, & Szatmari, 2010).

Choosing a passive activity here can be conceptualised as avoiding a negative consequence (e.g., embarrassment or ridicule), or indeed, opting for a sedentary activity that provides an immediate but *short-term* relief, perhaps at the expense of gaining larger *longer-term* rewards (e.g., self-competence, peer acceptance, etc.) through physical participation. In this way, the ability to regulate emotional responses during motor activity 'cools down' the affective components of motor task, reduces the significance of immediate 'rewards' (e.g., sedentary activities), and, consequently, increases the likelihood of participation in physical activities. Increased participation affords greater opportunities for the acquisition of motor skills.

Deficits in hot EF may disrupt the ability of children with DCD to regulate the impact of emotional experiences on motor performance. Deployment of emotion-regulation strategies (e.g., reappraisal or attention switching) is subserved by prefrontal areas that are also involved in cognitive and motor control (Coombes, Corcos, Pavuluri, & Vaillancourt, 2012; Goldin, McRae, Ramel, & Gross, 2008; Kanske, Heissler, Schönfelder, Bongers, & Wessa, 2011; McRae et al., 2009). The inhibition of automatic responses to compelling stimuli (e.g., opting for sedentary tasks in children with DCD) during motor planning – via strategies such as attentional deployment – effectively creates a 'dual-task' condition in that there is a competition for brain regions involved in *both* planning of motor responses and implementation of the emotion-regulation strategy (Beatty, Fawver, Hanock, & Janelle, 2014; Rémy, Wenderoth, Lipkens, & Swinnen, 2010). For example, diverting attention from rewarding stimuli via reappraisal strategies places demands on cognitive networks that have also been implicated in motor planning. Given that DCD is associated with reduced cognitive control in both hot and cool contexts, the ability to allocate attention and processing resources appropriately to different task demands may be limited (i.e., resulting in higher response latencies, and reduced accuracy). Supporting this argument are data showing poor dual-task performance in DCD, whether the secondary task is cognitive or motor in design (e.g.,

Cherng, Liang, Chen, & Chen, 2009; Rodger et al., 2003). Taken together, it is likely that immaturity in the development of fronto-parietal networks impacts broadly on the response capabilities of children with DCD. Reduced inhibitory control increases task difficulty in affectively-laden contexts due to competition for brain regions involved in both emotion regulation and action control. Affected are important motor control functions (like prediction) and the ability to regulate actions in response to compelling, emotional cues; the net effect is disruption to the process of learning functional motor skills and participating broadly in activities that are dependent on them. Thus, hot EF deficits appear to mediate the relationship between DCD and reduced participation in physical activities.

6.5.2 Goal-directed behaviour, and self-regulation. The interaction of higher-order cognitive control systems and basic emotional, motivational processes is an important determinant of children's goal-directed behaviour (Hare & Casey, 2005). Resistance to temptation and the ability to override internal and external impulses facilitate adjustment in different contexts by enabling individuals to consider alternative choices and implement other response options to suit the situation. Indeed, the ability to maintain sustained attention on tasks, and persevere in the face of difficulty or to control responses to compelling cues underlies the ability to delay gratification, and improves self-regulation. At a neural level, the ability to withhold responses to a compelling stimulus relies on modulating the strong signalling of the ventral striatum by the PFC (Kohls et al., 2009).

Self-regulation, however, is not a clearly articulated construct because it has been defined and measured in various ways. To illustrate, Zelazo and Müller (2011) suggest that tests of delay of gratification provide a measure of hot EF, while Diamond (2013) argues that delay of gratification is a test of self-regulation, but one resting on inhibitory control. Perhaps not surprisingly, self-regulation has been defined in a number of different ways and measured using a variety of tests (Hoyle & Davisson, 2011). Notwithstanding this, the general

consensus is that different aspects of self-regulation exist: the ability to plan, monitor, and modify actions, feelings, and behaviour are all aspects of it, and significant predictors of achievement and success in everyday life (Moffitt et al., 2011). Hot EF deficits disrupt self-regulation, which arguably relies on both the inhibitory control and the ability to hold active representations of task-relevant goals in mind (i.e., WM), and reduce the ability to suppress thoughts and actions (Riggs, Blair, & Greenberg, 2004). Reduced self-regulation is associated with a number of poor developmental outcomes, including difficulties in self-control, concentration, and academic achievement. It may also contribute to the higher incidence of externalising behaviours, such as aggression (Tseng, Howe, Chuang, & Hsieh, 2007) and conduct problems (Kanioglou, Tsorbatzoudis, & Barkoukis, 2005) in children with DCD.

Reduced self-regulation and low impulse control in emotionally-laden contexts may also be linked to the physical well-being of children with DCD, particularly greater risk for obesity in this group, especially among boys (Cairney, Hay, Faught, & Hawes, 2005). A systematic review of literature by Reinert, Po'e, and Barkin (2013) showed that obese children and adolescents often perform significantly worse than their healthy weight peers on different tasks of inhibitory control, including go/no-go task, delay of gratification, and the IGT. At a neurological level, obese children and adolescents often show increased activity in the orbitofrontal cortex (OFC) and dorsal striatum – the regions associated with inhibitory control and reward anticipation, respectively (Rothemund et al., 2007). Hyperactivity of the OFC in obese children and adolescents following food intake suggests that this region is working to counter the effect of (over-active) appetite-stimulating areas (Reinert et al., 2013). In the case of DCD, reduced ability to inhibit responses to compelling cues (e.g., sedentary activities, overeating), along with the avoidance of movement contexts that might be experienced as aversive reduce participation, and increase the risks for obesity. On the flip side, there is also growing awareness that obesity has a negative impact on cognitive

functioning – possibly through hypertension – and leads to executive dysfunction (Sabia, Kivimaki, Shipley, Marmot, & Singh-Manoux, 2009). This creates a vicious cycle whereby EF deficits (esp. reduced inhibitory control in affectively-laden contexts) disrupt physical fitness, which in turn have negative effects on cognitive functioning.

6.5.3 Emotion-regulation. Deficits of hot EF in DCD disrupt the development and functioning of processes that support emotion-regulation. For instance, delay of gratification in childhood facilitates coping skills, and predicts better self-control in adulthood (Casey et al., 2011). In a similar vein, reduced inhibitory control in hot, affective contexts – as operationalised by the performance of children with DCD on the HDT and the emotional go/no-go tasks – hinders the use and efficiency of emotion-regulation strategies. Reappraisal strategies (i.e., cooling down the hot aspects of motivationally significant stimuli), for instance, facilitate suppressing and/or regulating responses to stimuli with some emotional and motivational component. The ability to enlist reappraisal strategies in order to regulate emotions is subserved by the neural systems involved in cognitive control of action, including DL-PFC (selective attention, working memory), and VL-PFC (inhibition) (Ochsner et al., 2012). However, given that DCD has been linked to deficits in core aspects of EF (i.e., response inhibition, WM, and executive attention) (Wilson et al., 2013), deficits of cognitive control – subserved by L-PFC – manifest in a reduced ability of children with DCD to utilise emotion-regulation strategies.

Hot EF deficits in DCD may also disrupt emotion-regulation by interacting with individual and environmental constraints specific to DCD. Based on the Environmental Stress Hypothesis (Cairney et al., 2010), the motor coordination difficulty that is DCD results in a visible performance issue in children that may not clearly be identified as a disorder by their teachers or parents; this can create the impression that these children are simply lazy or defiant (Barnett, Dawes, & Wilmut, 2013; Fox & Lent, 1996), and their problems are due to

lack of effort. Their performance deficits can then leave these children open to criticism and ridicule (Miyahara & Piek, 2006), as well as isolation by their peers (Cairney, Rigoli, & Piek, 2013). The net disruption to social engagement can become a chronic source of stress that plays a significant role in the aetiology of affective disorders in DCD. Prefrontal control circuitry can indeed counter the effects of these environmental stressors that increase the signalling of emotion processing centres (Hare et al., 2008). However, in the case of DCD, reduced inhibitory control in affectively-laden contexts may compromise the regulatory function of control system, and reduce the ability to cope with stressors. In this way, hot EF deficits mediate the effects of environmental stressors on emotional symptoms of DCD.

6.6 How Do the Findings Inform Contemporary Theoretical Models of DCD

As noted in Chapter 1, the current aetiological account of DCD, which adopts the cognitive neuroscience approach, has converged into three main accounts: Impairments in the predictive control of action, problems with rhythmic coordination and timing, and reduced EF. Indeed, the pattern of EF deficits in DCD provides a clear indication that DCD is not purely a motor disorder, but rather a broader neurodevelopmental syndrome (Wilson et al., 2013); one with secondary socio-emotional consequences (Zwicker, Missiuna, Harris, & Boyd, 2012). As a result, aetiological research in DCD have examined both motor control and EF. The findings of the three studies that I presented in Chapters 2-4 complement the existing neurocognitive account of DCD by showing that the disorder may need to be characterised by EF deficits in *both* 'cold' and 'hot' contexts (Figure 6.2), and that some of the consequences of DCD may be linked to reduced hot EF. To illustrate, in addition to the processing costs associated with poor inhibitory control, hot EF deficits may contribute to disruptions in psychological and physical well-being in DCD, as well as participation. This assertion is consistent with the Environmental Stress Hypothesis (Cairney et al., 2013; Cairney et al., 2010) which maps the important interaction between individual and

environmental constraints. Deficits in hot EF may mediate the relationship between environmental stressors for children with DCD (see Environmental Stress Hypothesis) and the onset and/or intensity of emotional issues in this cohort. Environmental and other stressors increase the activity of the emotion processing centres; and for one to adjust well, this activity must be properly regulated by the prefrontal control circuitry (Hare et al., 2008). Reduced inhibitory control in DCD may compromise this regulatory function and reduce the ability to organise coping responses.

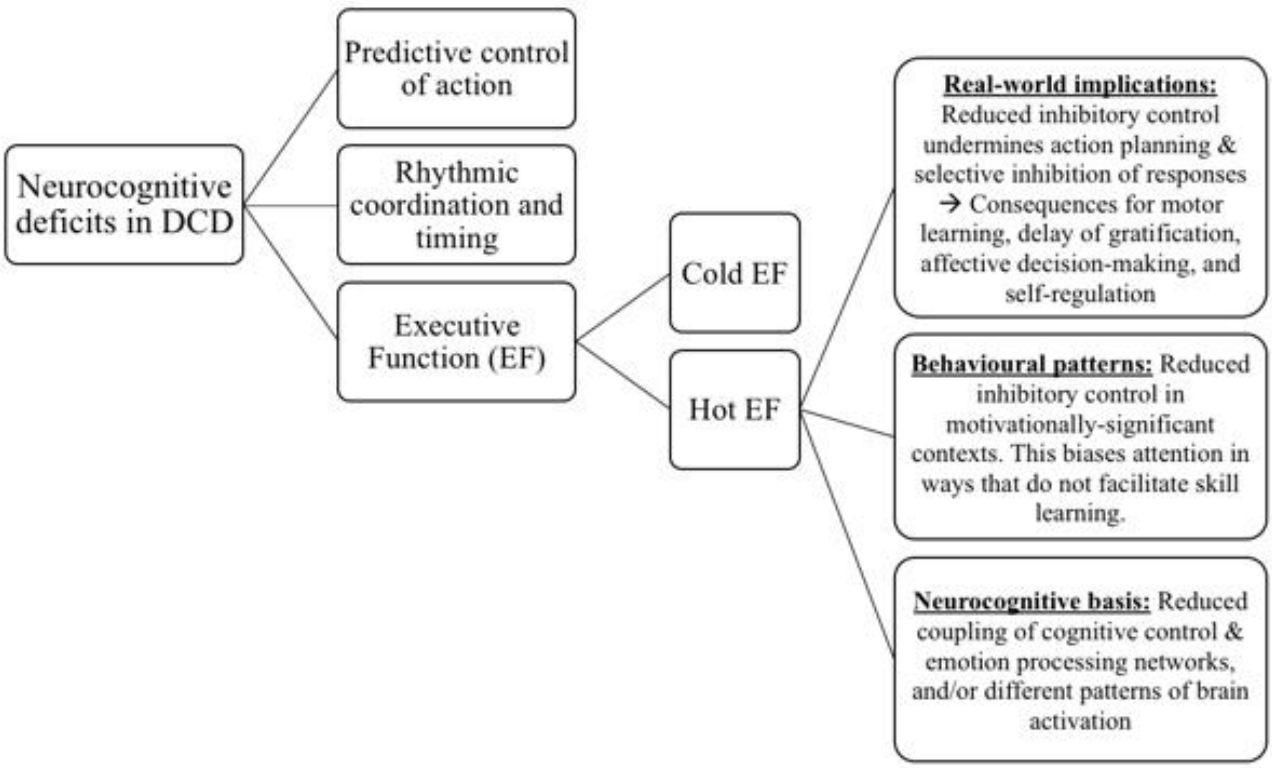

Figure 6.2. Neurocognitive basis, behavioural patterns, and real-world implications of hot EF deficits in DCD

Figure 6.3 summarises a conceptual model which maps the prime factors that mediate the relationship between DCD and its consequences. *First*, the effect of DCD on poor physical fitness and higher rates of obesity (Figure 6.2a) is thought to be mediated by inactivity and lower levels of participation (Cairney & Veldhuizen, 2013). *Second*, the

relationship between DCD and participation is itself mediated (Figure 6.2b) by emotional problems (e.g., anxiety, depression) experienced by children with DCD (Liberman, Ratzon, & Bart, 2013). *Third*, the relationship between DCD and internalising symptoms is not direct but rather mediated by self-perceived competence in different settings (Rigoli, Piek, & Kane, 2012) (Figure 6.2c).

However, as discussed in section 6.5, reduced EF (esp. poor inhibitory control of dominant responses, such as opting for immediate rewards) may also mediate the relationship between DCD and emotional problems (Figure 6.2c). In short, reduced coupling of cognitive control and emotion processing networks, which reciprocally modulate the function of one another and underlie response inhibition in affective contexts (Kohls, Peltzer, Herpertz-Dahlmann, & Konrad, 2009), undermines the efficacy of goal-directed action (Hare & Casey, 2005), and increases the risk for the onset of affective disorders (Dahl, 2004; Ernst, Pine, & Hardin, 2006; Steinberg, 2005). For instance, a fearful approach style for novel motor tasks and a tendency to partake in less physically-intense activities will curtail social interactions while creating a risk for affective disorders (Ferguson, Jelsma, Versfeld, Smits-Engelsman, 2014). Conversely, the ability to divert attention from conflicting or distressing stimuli and to exercise inhibitory control (both hot and cold) will enhance emotional regulation, a buffer against anxiety and other emotional issues (White, McDermott, Degnan, Henderson, & Fox, 2011).

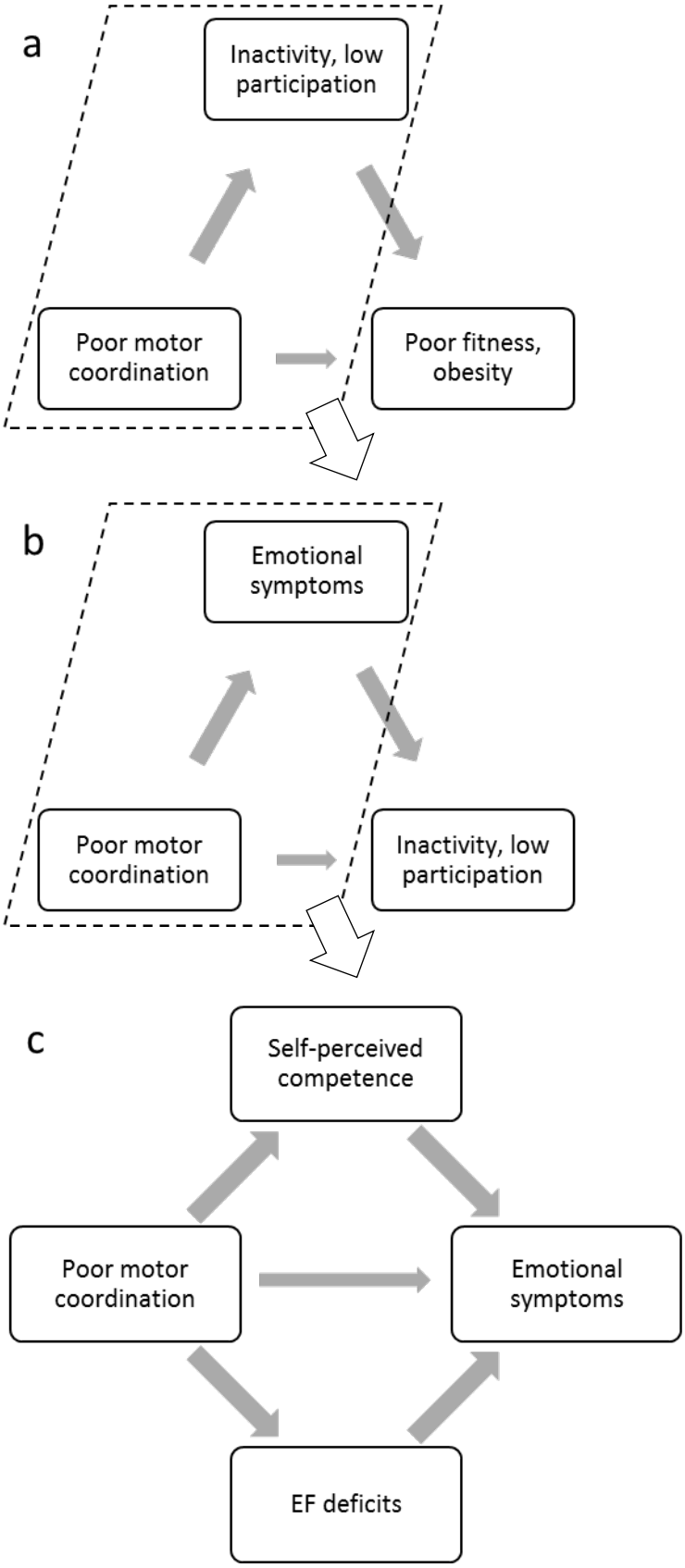

Figure 6.3. EF and self-perceived competence toward physical activity mediate the relationship between DCD and emotional problems as well as motor symptoms

6.7 How to Deal with Hot EF Deficits in DCD?

6.7.1 EF is malleable. If left untreated, individual differences in EF may persist through lifespan, affecting different aspects of development (Carlson & Zelazo, 2011). For example, individuals who performed poorly on a delay of gratification task (i.e., the Stanford marshmallow experiment; Mischel, Ebbesen, & Zeiss, 1972) at age 4 were more likely to have reduced self-control as adults (Casey et al., 2011). Therefore, it may be prudent to assess for possible EF deficits during critical periods of development, and at times when cognitive control (and its underlying neural systems) are particularly open to stimulation and learning (Zelazo & Carlson, 2012). The preschool years mark a period of most rapid development of EF (Carlson, Zelazo, & Faja, 2013). Another is the transition to adolescence (Zelazo & Carlson, 2012). This is when prefrontal systems undergo significant reorganisation, and the volume of grey matter reaches a peak in PFC (Giedd et al., 1999).

Fortunately, recent reviews of EF interventions suggest that well-designed programs and training lead to behavioural improvement and corresponding changes in neural function. For instance, computerised training, particularly CogMed, which targets WM using a series of increasingly challenging games, have been shown to enhance the performances of both TD children (Thorell, Lindqvist, Bergman, Bohlin, & Klingberg, 2009) and those with ADHD (Holmes et al., 2010; Klingberg et al., 2005) on different measures of WM. Computerised training, however, has been less effective in improving inhibitory control in pre-school children (Rueda, Rothbart, McCandliss, Saccomanno, & Posner, 2005; Thorell et al., 2009).

In contrast, sport activities that not only improve fitness – as in aerobic exercise – but also challenge EFs and bring joy and social bonding, constitute the second class of EF interventions. An example with strong evidence is traditional martial arts, which promotes self-control, discipline, and character development. Lakes and Hoyt (2004), who randomly assigned a group of 5-11 year olds to standard physical education or tae-kwon-do (with

increasing challenges), found that the martial arts program led to significant improvements in cognitive inhibitory control and emotion regulation. The effects persisted in different contexts and on different measures of inhibition.

The third major class of EF interventions are add-ons to school curricula, such as Promoting Alternative Thinking Strategies (PATHS; Riggs, Greenberg, Kusche, & Pentz, 2006) and the Chicago School Readiness Project (CSRP; Raver et al. 2008, 2011). PATHS, for instance, promotes inhibitory control in affective contexts, and enhances interpersonal problem-solving in children by emphasising the ability to verbalise feelings and practice conscious self-control strategies (e.g., waiting before acting, self-talk). When children get upset, they need to take a deep breath, identify their feelings and what caused them, and – with the help of their teachers – construct an action plan to solve the problem. Riggs and others (2006) showed that a year of PATHS results in increased inhibitory control and cognitive flexibility in 7-9 year old children. However, the positive effects of add-ons to school curricula have not been replicated in older children (Diamond, 2012).

Other interventions that require active engagement and practice in a range of motor activities – for instance, aerobic exercise (Chaddock, Pontifex, Hillman, & Kramer, 2011; Hillman, Erickson, & Kramer, 2008), bimanual basketball dribbling (Davis, Pitchford, & Limback, 2011) and handwriting (Rosenblum, 2013) – have been shown to improve different aspects of EF, possibly through stimulating structural and functional changes in the brain regions underlying cognitive control (Ferguson et al., 2014). However, the effects of these programs are not as widespread as programs that target EF more globally – that is computer-based training, physical training like traditional martial arts, and add-ons to school curricula (Diamond, 2012; Diamond & Lee, 2011). These programs that have the strongest evidence are characterised by random assignment of children to intervention and control groups, having an active control group, pre- and post-intervention assessments, and also transfer of

effects to more than one objective measure of EF on which the training was focused (Diamond, 2013).

Physical and psychological well-being of individuals, as well as the difficulty of training programs also contribute to the efficacy of EF interventions and whether the benefits last. Existing literature (e.g., Diamond, 2013) shows that PFC and, consequently, EFs are the first to suffer when a person is physically unfit, sad, stressed, or lonely. Thus, those EF programs that not only train and increasingly challenge different EFs, but also indirectly enhance EFs by addressing factors that support cognitive control (e.g., physical fitness, social support, self-confidence) are predicted to most successfully improve EF (Diamond, 2015). Training programs that address stressors in one's life, include joyful activities, and promote a sense of belonging and social acceptance have better chances of enhancing EFs. Moreover, real-world activities that involve repeated practice of diverse motor and EF skills (e.g., martial arts, and school curricula) result in more widespread benefits than targeted EF interventions such as computerised training. Finally, benefits will be minimal if children continue doing the same activity with no changes in difficulty throughout an intervention program; therefore, it is important to incrementally increase the difficulty of motor and EF activities so to prevent children from getting bored.

6.7.2 Potential benefits of EF interventions for children with DCD. The benefits of the healthy development of hot EF for various developmental outcomes and psychosocial adjustment have been discussed in earlier chapters. EF interventions often target skills that facilitate academic and vocational success and improve physical and mental health (Diamond, 2012). The benefits of EF interventions, in general, are transferrable to novel situations. For instance, the skills learned in task switching, transfer to both novel shifting tasks, as well as to measures of response inhibition, verbal and non-verbal WM, and reasoning (Karbach & Kray, 2009). This is due to task-switching relying on both WM and

inhibition: One needs to keep an active mental representation of task goals (i.e., WM) while inhibiting prepotent responses shaped by earlier task goals that are no longer relevant. EF interventions have the potential to enhance processes that support emotion-regulation like inhibitory control and attention shifting (White et al., 2011). Deficits in these processes can undermine emotion-regulation and increase the risk for the onset of affective disorders (Muris, Mayer, van Lint, & Hofman, 2008). Here, I focus on the merits of those EF interventions that involve some physical training for children with DCD. Attention is drawn to the potential role of EF interventions in improving self-efficacy, self-concept and symptoms of anxiety.

In DCD we see a cascading effect of motor difficulties on other aspects of functioning, which can often persist into adulthood (Missiuna et al., 2014). Hence, well-targeted intervention plays a significant role in addressing the long-term effects of the disorder. Notably, poor motor skill can impact self-perceived competence which has a number of adverse flow-on effects for *both* physical and psychological functioning in children with DCD. Self-competence is thought to mediate the relationship between DCD and participation in physical activities (Batey et al., 2013). In turn, reduced participation in physical activities, particularly active play (e.g., sports or free play with peers), leads to low physical fitness and increased risk of obesity in children with motor coordination problems. Poor physical health and inactivity can exacerbate the risk of internalising problems, mainly by disrupting the availability of personal (e.g., self-esteem, self-competence) and social (e.g., peer support) resources required to cope with daily stressors associated with DCD (Cairney et al., 2013). In short, low self-perceived competence in DCD (e.g., minimal participation, limited social contacts, low self-esteem) is a known risk factor for depression and anxiety (Missiuna et al., 2014). Cairney and colleagues (2013) suggested that building self-perceived

competence will act as a protective mechanism against the consequences of DCD, reducing longer-term internalising problems.

Well-designed EF interventions that involve physical activity, as well as character development, could prepare children with DCD for the psychological and emotional challenges they may face in performing novel skills, and help change their attitudes toward physical activity by improving their self-perceived competence and self-control. Children become more adept at avoiding stimuli and situations they might initially find comforting (e.g., isolated and passive activities), and instead may be more willing to take part in activities that are generally intimidating to them (e.g., active play and social interactions with peers) but lead to meaningful rewards in the long run. Thus, training programs such as martial arts that promote *both* inhibitory control and self-perceived competence are particularly beneficial in this context because they help children with DCD to shift their attention away from tempting stimuli, and improve their participation in physical activities, which in turn increases the amount of social interactions, the possibility of forming relationships with peers, and the availability of social support. Recent studies suggest that exercise-based interventions improve electrophysiological (i.e., enhanced P3 amplitude) and behavioural performance of children with DCD on measures of inhibitory control (10-week soccer training; Tsai, Wang, & Tseng, 2012) and visuospatial WM (16-week aerobic exercise; Tsai et al., 2014) tasks.

6.7.3 Implementation of EF interventions in children with DCD. Children with motor coordination problems are not usually referred for help until their motor difficulties start to interfere with their school performance (Missiuna et al., 2014). For instance, difficulty with handwriting is the most common reason for referrals to health care professionals (Cantin et al., 2014). Even though motor coordination problems are often identified at a younger age by parents, the guidelines of the European Academy for Childhood Disability (Blank, Smits-

Engelsman, Polatajko, & Wilson, 2012) suggest the minimum age at which DCD can be diagnosed is 5 years. The preschool period (i.e., around 5-6 years of age) is an appropriate time for addressing possible EF deficits; first, because EF is most malleable during this stage of development (Zelazo & Carlson, 2012) and, second, because children of this age do not yet perceive major differences in competence across domains compared with peers (Pless, Carlsson, Sundelin, Persson, 2002; Rodger et al., 2003). This gives clinicians an ideal opportunity to mitigate EF and motor difficulties before they start to undermine cognitive control and self-perceived competence.

However, this does not mean EF interventions at an earlier age could not be beneficial. Difficulties with skilled motor ability can be identified at an earlier age than EF problems (Gonzalez et al., 2014). For instance, reaching and grasping, which develops by 6 months of age, can be reliably assessed by age one (Jacquet, Esseily, Rider, & Fagard, 2012; Sacrey, Arnold, Whishaw, & Gonzalez, 2013). Developmental delays in this ecologically-valid measure of motor functioning could be an early sign for more widespread motor and cognitive problems. Gonzalez and colleagues (2014) suggest that the identification of motor learning problems in young children should be followed immediately by interventions for both the motor skill difficulty and training of EF abilities. In a similar vein, Diamond (2013) argues that EFs can be improved at any point across the lifespan, even in infancy. For instance, exposure to bilingual input has been shown to accelerate EF development in infants. (Kovács & Mehler, 2009; Wass, Porayska-Pomsta, & Johnson, 2011).

Even though the aforementioned evidence suggests it is possible to run EF interventions throughout childhood, certain aspects of existing programs need to be modified to make them more beneficial for children with DCD. One important variable is the mode of delivery for task instructions. To illustrate, while interactive DVDs were shown to be as effective as traditional physical therapy to train motor imagery in children with DCD

(Wilson, Thomas, & Maruff, 2002), verbal instructions failed to produce the same level of response in children with motor coordination problems (Williams, Thomas, Maruff, & Wilson, 2008). This shows that there may be subgroups within the DCD population who respond better to visual than verbal instructions. Thus, EF interventions may need to utilise both modes of instructions to maximise the likelihood of benefits in children with DCD. Moreover, given that DCD is linked to reduced WM, attention, and inhibition (Wilson et al., 2013), it is recommended to break down each task to its constituent parts in order to facilitate learning and retention. The use of shorter intervals between training sessions, and increasing task difficulty in a more gradual manner – compared to the rate used for TD children – may also reduce cognitive demands, and lead to longer lasting benefits in children with DCD.

6.7.4 Limitations of EF interventions. In spite of the large number of studies on EF interventions, there remain some important unanswered questions that might limit the use of training programs, particularly in developmental disorders. For instance, it is obvious that the amount of training and the maturity of EFs – prior to the start of training – contribute to the success of interventions; however, other factors that distinguish who benefit from EF training and who does not are not yet clearly identified. Moreover, no study has yet investigated whether benefits last even as long as a month; therefore, we still do not know whether and how long training-induced changes might last; and if they do last, what can increase the duration of benefits, or whether there is a need for refresher sessions to maintain change following training (Diamond, 2015). The other significant questions are whether specific programs should be designed for each age group; and whether the dose or frequency of training varies by age (Diamond, 2013). To illustrate, computerised training and martial arts are suggested to be more beneficial for children aged 8-12 years than those in the pre-school period. The use of martial arts was limited to children; therefore, it is not clear whether the benefits of this intervention over aerobic exercises are due to the nature of the program or the

age of the participants. Moreover, to the best of my knowledge, only one study to date has examined the effect of dosage (i.e., time spent on each activity per session) on the outcome of EF interventions (Davis et al., 2011). Finally, given that there is still limited data supporting specific EF interventions in children with DCD, one must exercise caution in the generalisation of the effects obtained from studies of TD children. Having said that, even small improvements in EF skills, particularly inhibitory control, could have cascading effects in that the entire distribution of outcomes are shifted in a salutary direction (Moffitt et al., 2011).

6.8 General Limitations and Future Directions

6.8.1 Diagnosis of DCD, and different cut-off points. In all studies reported in my thesis, a nominal cut-off of 80-85 on the McCarron Assessment of Neuromuscular Development (MAND; McCarron, 1997) was used to identify children meeting criterion A of DCD diagnosis in DSM-5 (APA, 2013) and the research guidelines for DCD (Blank et al., 2012; Geuze, Jongmans, Schoemaker, & Smits-Engelsman, 2001). However, the screening of normative samples of school-aged children for motor difficulties was not followed by a formal clinical diagnosis of those in the DCD group. Even though the performance of 'at-risk' populations for DCD – identified via screening – on a range of EF tasks is highly similar to that of children with a clinical diagnosis of DCD (e.g., Leonard, Bernardi, Hill, & Henry, 2015), it is still important for future studies to distinguish 'at-risk' children from those with a clinical diagnosis because the severity of impairment is likely to moderate deficits of hot EF. There is evidence that the more severe the level of motor impairment and associated EF difficulties, the worse the long-term outcome for children with DCD (Piek et al., 2007). Moreover, recruiting children with a clinical diagnosis of DCD provides a representative sample and increases the ecological validity of findings.

Moreover, I noted in chapter 2 that in order to achieve a statistical power of .8, I had to use a higher cut-off score in study 1 (i.e., NDI = 85) than the other two studies (i.e., NDI = 80). Although different cut-scores may limit the comparability of results across studies, the use of a stricter quantitative criteria for motor skills increases the chances of selecting a more homogenous group of children with motor impairment. Low cut-off score on a motor skills test reduces the chances of false positives biasing the outcomes of research; therefore, enabling the study of DCD in its pure form (Geuze et al., 2001). Having said that, even in Study 1, which used a more lenient cut-off score than the other two, DCD group performed worse than the controls on a measure of hot EF. However, one possibility is that a lower cut-off point would result in larger effects on (hot) EF tasks between DCD and control groups. Indeed, Mandich and colleagues (2002) found a positive relationship between the severity of DCD symptoms and deficits of inhibitory control. In a similar vein, Study 2, in which children with moderate motor difficulties (55 < NDI < 70) performed worse than the rest of the DCD group (70 < NDI < 80), suggests that there might be a *dose-response relationship* (Cairney & Veldhuizen, 2013) between the level of motor impairment and performance on hot EF tasks.

6.8.2 Sample size. The size of DCD samples in each of my studies was sufficient to yield adequate statistical power (.80) in anticipation of moderate-to-large effect sizes. However, the sample size did not permit investigation of the relationship between the severity of motor impairment and hot EF (i.e., *dose-response relationship*). Large samples facilitate the investigation of this relationship by providing a continuum of scores on both the hot EF and the motor abilities tasks.

Use of larger number of children with DCD, or ‘big data’ – that is combining databases – in future studies would also enable investigation of the relationship between hot EF and severity of motor impairment in children with “pure DCD” and in those with

comorbid conditions. Analysis of individual performances in Studies 2 and 3 showed some heterogeneity in the performance of the DCD group on the go/no-go task. Even though most children in this group made a large number of commission errors, there were some who performed within the average range of controls. Given that all children with comorbid diagnoses were excluded from data analyses, this finding raises the possibility of sub-groups even within 'pure' DCD cohorts. However, numbers do not permit a more formal analysis of possible differences between sub-groups. Finally, comorbidity is likely to predict more severe EF deficits in children with DCD, as well as associated psychosocial consequences (e.g., Astill, 2007; Mandich, Polatajko, & Rodger, 2003). This is an issue for further study using larger cohorts.

6.8.3 Age group. One way to extend the findings of my thesis is to examine hot EF in adolescents and adults with DCD. Adolescence is marked by an increased risk of problem behaviours associated with reduced inhibitory control in contexts that are emotionally salient. During this period, the pattern of activity in the emotion circuitry of the brain resembles that of adults while activity within cognitive control networks resembles that of children (Reinert et al., 2013). The activity of relatively mature emotion processing centres is not properly modulated by developing control networks during this period. This may increase the likelihood of impulsive and risk-taking behaviours among adolescents. Thus, the difference in developmental trajectories of emotion processing and cognitive control networks may disrupt psychological adjustment and self-regulation skills of adolescents, particularly those with deficits of cognitive control such as individuals with DCD.

As well, it is a fact that DCD often persists into adulthood (Cantell, Smyth, & Ahonen, 2003; Rasmussen & Gillberg, 2000), with the tasks of daily living, education and vocation becoming more cognitively demanding with age (Leonard & Hill, 2015). Thus, a proper understanding of the development of (hot) EF in DCD, and of the possible age-related

changes in the effect of EF deficits on performance requires the assessment of (hot) EF in both adolescents and adults. However, it is worth noting that currently there are two important challenges in testing adults with DCD. Firstly, the availability of standardised tests of motor ability for adults is limited (Kirby, Edwards, Sugden, & Rosenblum, 2010), and secondly, DCD is not often diagnosed in adults mainly because clinicians did not have sufficient knowledge of the disorder and its progression (Leonard & Hill, 2015). Longitudinal studies will enable researchers to establish causal connections between EF deficits and its downstream effects on performance patterns in DCD.

6.8.4 Stimulus type. Approaching negatively-valenced stimuli (e.g., fearful or sad faces) requires the performer to override affective interference, caused by a conflict between the natural tendency to avoid aversive stimuli and task demands to approach such targets. The results of Study 3 (chapter 5) showed that both children with DCD and their TD peers found it difficult to distinguish sad from neutral faces. This suggests a perceptual deficit in both groups, rather than a behaviour control and emotion-regulation problem. Consequently, a full examination of approach behaviour with respect to negative stimuli was not possible. A more systematic assessment of children's sensitivity to go/no-go stimuli is suggested, particularly for different facial expressions. Establishing perceptual thresholds for different age groups is particularly important for children with DCD who show difficulties recognising static and dynamic facial expressions (Cummins et al., 2005).

6.8.5 Competing hypothesis. Impaired use of somatic markers was one of the main hypotheses used to explain the performance pattern of children with DCD on the HDT (see section 6.3.1). Advantageous decision-making on tasks such as the IGT or the HDT relies on the ability to use feedback from earlier trials, and anticipate the outcome of similar actions (Crone & van der Molen, 2007). Bechara and others (1996) showed that individuals who perform well on the IGT develop anticipatory skin conductance responses immediately

before choosing the disadvantageous options; however, these responses were absent in patients with VM-PFC lesions who perform poorly on the task. Use of skin conductance would be useful in future studies of children with and without DCD, while performing the HDT or other variants of the IGT.

6.8.6 Neuroimaging data. The postulates regarding the neural underpinnings of hot EF deficits in DCD (section 6.4.3) may be tested further using neuroimaging and morphological studies. Currently, there is limited data on the neural correlates of EF deficits in DCD. For instance, existing literature on neural underpinnings of DCD has focused on response inhibition and WM on visuospatial or motor tasks (Querne et al., 2008; Tsai et al., 2014; Tsai, Chang, Hung, Tseng, & Chen, 2012; Tsai, Wang et al., 2012). At least three methods of neuroimaging could be applied usefully to map the neural networks involved in the performance of hot EF tasks, and how they differ between DCD and TD children. First, high resolution anatomical magnetic resonance images could show whether the brains of children with DCD are morphologically different to the brains of their TD peers. Second, fMRI examines whether patterns of brain activation are different in children with motor coordination problems (Zwicker, Missiuna, & Boyd, 2009). However, it is worth noting that fMRI provides an indirect measure of brain activity, and it is also difficult to use this technique in paediatric populations (Brown-Lum & Zwicker, 2015). The third method is diffusion tensor imaging (DTI) which provides detailed information about the integrity of white matter tracts, including their maturation and pruning (Zwicker et al., 2009). This method is more informative than the other two because it helps determining whether reduced functional connectivity, for example in fronto-striatal circuits, underlies deficits of hot EF in DCD.

6.9 Conclusion

The studies reported in my thesis constitute the first attempt to examine hot EF in children with DCD. In sum, children with DCD showed deficits of hot EF compared with their same-age TD peers. Deficits were operationalised by performance on the HDT as well as two different emotional go/no-go tasks. The findings suggest that children with DCD may find it harder to resist rewarding aspects of emotionally significant stimuli, as presented using the paradigms adopted in this thesis. In this chapter, I discussed different postulates regarding the underlying reasons for hot EF deficits in DCD. I raised the possibility that these children may have difficulty using somatic markers, and discussed how deficits of cool EF (esp. inhibitory control) could undermine cognitive control of the DCD group in hot, affective contexts. Positively-valenced stimuli increase the activation of emotion circuitry of the brain, and enhance approach tendencies. Inhibiting responses to rewarding stimuli is more difficult than withdrawing responses to neutral or negative stimuli because it requires greater activation of the control networks. The existence of inhibitory control deficits in DCD makes it increasingly difficult for this group to modulate their responses to compelling stimuli. Therefore, the heightened sensitivity of children with DCD to positive social cues (i.e., go/no-go tasks) and immediate reward (HDT) may reflect impaired coupling/interaction between emotion processing centres and cognitive control networks. I presented three hypotheses (not mutually exclusive) to explain the likely impairment to the coupling between emotion processing and cognitive control networks. The first hypothesis concerns different patterns of brain activation in TD and DCD population. This hypothesis involves the hyperactivity of the brain regions involved in encoding the reward value of stimuli and/or the hypoactivity of those implicated in cognitive control of action. The second hypothesis is that delayed development of the control networks makes it difficult to modulate the activity of emotion circuitry. The third hypothesis is that a disruption to the integrity of WMNs would

reduce functional connectivity between the cognitive control and emotion processing centres, eventually resulting in more approach-oriented behaviour toward rewarding stimuli. Given the weight of recent evidence regarding disruption to the integrity of WMNs in DCD, the third hypothesis seems to provide a better account of hot EF deficits in DCD. I also discussed how reduced EF in affectively-laden contexts may affect motor functioning, goal-directed behaviour, as well as self- and emotion-regulation. The two models presented in section 6.6 also established the relevance of the findings of my thesis to a broad contemporary account of DCD – more specifically, the neurocognitive approach in understanding the aetiology of the disorder. The significance of hot EF in predicting various developmental outcomes highlights the need to address EF difficulties in children with DCD. Converging evidence suggests that EF is malleable, and that the most effective EF interventions are those that involve some degree of physical training. These interventions are particularly beneficial for the DCD population because they have the potential to increase *both* motor, and EF skills, and, consequently, self-perceived competence, which mediates the relationship between DCD and its emotional symptoms. Even though the aforementioned limitations of existing EF interventions may limit their use in children with motor coordination problems, even small improvements of EF could have far-reaching benefits for this cohort. The most important recommendations for future studies of hot EF in DCD include recruitment of large samples of individuals with DCD across a wide age range, investigation of competing hypotheses for reduced hot EF in DCD, simultaneous use of neuroimaging techniques, particularly DTI, and the adoption of a longitudinal design to unravel the underlying causes of hot EF deficits in DCD.

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Research Portfolio

Published articles

Publication #1:

Rahimi-Golkhandan, S., Piek, J., Steenbergen, B., & Wilson, P. H. (2014). Hot executive function in children with developmental coordination disorder: Evidence for heightened sensitivity to immediate reward. *Cognitive Development, 32*, 23-37. doi: 10.1016/j.cogdev.2014.06.002. **5-year impact factor: 2.25, SJR: Q1**

Acceptance Letter:

Date: Jun 09, 2014
To: "Peter Wilson" peterh.wilson@acu.edu.au
From: Deanna Kuhn cogdev@exchange.tc.columbia.edu
Subject: Your Submission

Ms. Ref. No.: COGDEVD1300077R2
Title: Hot executive function in children with developmental coordination disorder: Evidence for heightened sensitivity to immediate reward
Cognitive Development

Dear Dr. Wilson,

I am pleased to confirm that your paper "Hot executive function in children with developmental coordination disorder: Evidence for heightened sensitivity to immediate reward" is now accepted for publication in Cognitive Development. I am satisfied that all outstanding concerns have been adequately addressed.

The manuscript will undergo copyediting that you will have the opportunity to address during the course of the production process.

Thank you for submitting your work to this journal. I look forward to seeing it in print.

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With best regards,
Deanna Kuhn
Editor
Cognitive Development

Publication #2:

Rahimi-Golkhandan, S., Steenbergen, B., Piek, J., & Wilson, P. H. (2014). Deficits of hot executive function in Developmental Coordination Disorder: Sensitivity to positive social cues. *Human Movement Science, 38*, 209-224. **5-year impact factor: 2.60, SJR: Q1**

Acceptance Letter:

Subject: Your Submission HMS-D-14-00120R1
Sent: 07/09/2014 9:33 pm
From: Human Movement Science <hms@fbw.vu.nl>
To: PeterH Wilson <PeterH.Wilson@acu.edu.au>

Ms. Ref. No.: HMS-D-14-00120R1
Title: Deficits of Hot Executive Function in Developmental Coordination Disorder: Sensitivity to Positive Social Cues
Human Movement Science

Dear Prof. Peter Wilson,

I am pleased to confirm that your paper "Deficits of Hot Executive Function in Developmental Coordination Disorder: Sensitivity to Positive Social Cues" has been accepted for publication in Human Movement Science.

When your paper is published on ScienceDirect, you want to make sure it gets the attention it deserves. To help you get your message across, Elsevier has developed a new, free service called AudioSlides: brief, webcast-style presentations that are shown (publicly available) next to your published article. This format gives you the opportunity to explain your research in your own words and attract interest. You will receive an invitation email to create an AudioSlides presentation shortly. For more information and examples, please visit http://www.elsevier.com/audioslides

You will receive the page proofs for the article in due course. In correcting those page proofs please be so kind to include the full name for DL-PFC (p. 5 of the article, 3rd line), as done for the previous parts of the brain. You are also kindly required to reconsider rephrasing the last paragraph of the discussion so as to improve its clarity.

Thank you for submitting your work to this journal.

With kind regards,

Peter Jan Beek
Editor of HMS

Publication #3:

Rahimi-Golkhandan, S., Steenbergen, B., Piek, J., Caeyenberghs, K., & Wilson, P. H. (*under review*). Revealing Hot Executive Function in Children with Motor Coordination Problems: What's the Go? *Brain and Cognition*. **5-year impact factor: 3.34, SJR: Q1**

Statement of Contribution of Others

Introductory notes: My PhD thesis was part of a larger project – funded by the Australian Research Council (ARC) Discovery Grant DP1094535 – with an overall aim of investigating the coupling of online motor control and inhibitory function. The specific aim of my thesis, however, was to investigate inhibitory control in emotionally-significant contexts in children with motor coordination problems. To address this previously unexplored topic in the DCD literature, I developed various research questions (in consultation with my primary supervisor, Prof. Peter Wilson) and chose particular state-of-the-art measures designed for children to collect the data that informed the findings of my thesis. This section specifies the contribution of my two supervisors and the other co-authors in the preparation of papers that emanated from this project.

Publication: **Hot Executive Function in Children with Developmental Coordination Disorder: Evidence for Heightened Sensitivity to Immediate Reward**

I acknowledge that my contribution to the above publication is 12.5 percent.

Author: Prof. Jan P. Piek

Signature: 

Date: 01/06/2015

I acknowledge that my contribution to the above publication is 12.5 percent.

Author: Prof. Bert Steenbergen

Signature: 

Date: 31/05/2015

I acknowledge that my contribution to the above publication is 25 percent.

Author: Prof. Peter H. Wilson

Signature: 

Date: 8-6-2015

Publication: **Deficits of Hot Executive Function in Developmental Coordination Disorder: Sensitivity to Positive Social Cues**

I acknowledge that my contribution to the above publication is 12.5 percent.

Author: Prof. Jan P. Piek

Signature: 

Date: 01/06/2015

I acknowledge that my contribution to the above publication is 12.5 percent.

Author: Prof. Bert Steenbergen

Signature: 

Date: 31/05/2015

I acknowledge that my contribution to the above publication is 25 percent.

Author: Prof. Peter H. Wilson

Signature: 

Date: 8-6-2015

Publication: **Revealing Hot Executive Function in Children with Motor Coordination Problems: What's the Go?**

I acknowledge that my contribution to the above publication is 10 percent.

Author: Prof. Jan P. Piek

Signature: 

Date: 01/06/2015

I acknowledge that my contribution to the above publication is 10 percent.

Author: Prof. Bert Steenbergen

Signature: 

Date: 31/05/2015

I acknowledge that my contribution to the above publication is 10 percent.

Author: Prof. Karen Caeyenberghs

Signature: 

Date: 5/06/2015

I acknowledge that my contribution to the above publication is 20 percent.

Author: Prof. Peter H. Wilson

Signature: 

Date: 8-6-2015

Peer-Reviewed Conference Presentations and Posters

Rahimi-Golkhandan, S., Steenbergen, B., Piek, J., Caeyenberghs, K., & Wilson, P. H. (2015, July). *Hot cognition in children with motor coordination problems: Insights using a go/no-go paradigm*. Paper presented at the 11th International Conference on Developmental Coordination Disorder. Toulouse, France.

Rahimi-Golkhandan, S., Steenbergen, B., Piek, J., Caeyenberghs, K., & Wilson, P. H. (2015, July). *The relationship between hot and cold executive function in developmental coordination disorder*. Poster presented at the 11th International Conference on Developmental Coordination Disorder. Toulouse, France.

Wilson, P. H., **Rahimi-Golkhandan, S.**, Ruddock, S., Piek, J. P., Sugden, D., Green, D., & Steenbergen, B. (2015, May). *Development of executive function in children with Developmental Coordination Disorder (DCD): A two-year longitudinal investigation*. Paper presented at the 27th Annual Meeting of the European Academy of Childhood Disability. Copenhagen, Denmark.

Rahimi-Golkhandan, S., Piek, J. & Wilson, P. H. (2013, November). *Hot executive function in children with developmental coordination disorder: Speeded responses to high immediate reward*. Poster presented at the 11th Motor Control and Human Skill Conference. Melbourne, Australia.

Rahimi-Golkhandan, S., Piek, J. & Wilson, P. H. (2013, October). *Deficits of hot executive function in children with developmental coordination disorder, and their implications for theory and practice*. Paper presented at the 25th Annual Meeting of the European Academy of Childhood Disability. Newcastle, United Kingdom.

Rahimi-Golkhandan, S. & Wilson, P. H. (2013, June). *Hot executive function in children with developmental coordination disorder*. Paper presented at the 10th International Conference on Developmental Coordination Disorder. Ouro Preto, Brazil.

Appendices

APPENDICES

Appendix A Ethics Approval: Australian Catholic University

From: Gabrielle Ryan [mailto:Gabrielle.Ryan@acu.edu.au]
Sent: Thursday, 29 March 2012 10:28 AM
To: PeterH Wilson;
Cc: Gabrielle Ryan
Subject: Ethics Application approved 2012 73V

Dear Peter,

Ethics Register Number: 2012 73V
Project Title: The development of rapid on-line motor control in children End Date: 30/06/2013

This email is to advise that your application has been reviewed by the University Human Research Ethics Committee and your approval has been transferred from RMIT HREC to the ACU HREC. ACU HREC is now the primary HREC on this project. Please ensure that you have completed the relevant processes to close off this project at RMIT, if there are no ongoing links with researchers still at RMIT.

Whilst the data collection of your project has received ethical clearance, the decision to commence and authority to commence may be dependent on factors beyond the remit of the ethics review process. For example, your research may need ethics clearance from other organisations or permissions from other organisations to access staff. Therefore the proposed data collection should not commence until you have satisfied these requirements.

If you require a formal approval certificate, please respond via reply email and one will be issued.

Decisions related to low risk ethical review are subject to ratification at the next available Committee meeting. You will only be contacted again in relation to this matter if the Committee raises any additional questions or concerns.

This project has been awarded ethical clearance until 30/06/2013 and a progress report must be submitted at least once every twelve months.

Researchers who fail to submit an appropriate progress report may have their ethical clearance revoked and/or the ethical clearances of other projects suspended. When your project has been completed please complete and submit a progress/final report form and advise us by email at your earliest convenience. The information researchers provide on the security of records, compliance with approval consent procedures and documentation and responses to special conditions is reported to the NHMRC on an annual basis. In accordance with NHMRC the ACU HREC may undertake annual audits of any projects considered to be of more than low risk.

For progress and/or final reports, please complete and submit a Progress / Final Report form:
http://www.acu.edu.au/about_acu/research/staff/research_ethics/

For modifications to your project, please complete and submit a Modification form:

http://www.acu.edu.au/about_acu/research/staff/research_ethics/

Researchers must immediately report to HREC any matter that might affect the ethical acceptability of the protocol e.g.: changes to protocols or unforeseen circumstances or adverse effects on participants.

Please do not hesitate to contact the office if you have any queries.

Kind regards,
Gabrielle Ryan

Ethics Officer | Research Services
Office of the Deputy Vice Chancellor (Research) Australian Catholic University Locked Bag 4115, Fitzroy, VIC, 3065
T: 03 9953 3150 F: 03 9953 3315

Appendix B Ethics Approval: Department of Education

Department of Education and Early Childhood Development

Office for Policy, Research and Innovation

2 Treasury Place
East Melbourne, Victoria 3002
Telephone: +61 3 9637 2000
DX 210083
GPO Box 4367
Melbourne, Victoria 3001

2010_000645

Associate Professor Peter Wilson
Psychology, School of Health Sciences
Royal Melbourne Institute of Technology
GPO Box 2476V
MELBOURNE 3001

Dear Associate Professor Wilson

Thank you for your application of 3 June 2010 in which you request permission to conduct a research study in government schools titled: *The development of rapid on-line motor control in children.*

I am pleased to advise that on the basis of the information you have provided your research proposal is approved in principle subject to the conditions detailed below.

1. Should your institution's ethics committee require changes or you decide to make changes, these changes must be submitted to the Department of Education and Early Childhood Development for its consideration before you proceed.

2. You obtain approval for the research to be conducted in each school directly from the principal. Details of your research, copies of this letter of approval and the letter of approval from the relevant ethics committee are to be provided to the principal. The final decision as to whether or not your research can proceed in a school rests with the principal.

3. No student is to participate in this research study unless they are willing to do so and parental permission is received. Sufficient information must be provided to enable parents to make an informed decision and their consent must be obtained in writing.

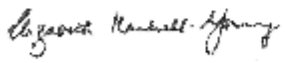
4. As a matter of courtesy, you should advise the relevant Regional Director of the schools you intend to approach. An outline of your research and a copy of this letter should be provided to the Regional Director.

5. Any extensions or variations to the research proposal, additional research involving use of the data collected, or publication of the data beyond that normally associated with academic studies will require a further research approval submission.

6. At the conclusion of your study, a copy or summary of the research findings should be forwarded to Education Policy and Research Division, Department of Education and Early Childhood Development, Level 3, 33 St Andrews Place, GPO Box 4367, Melbourne, 3001.

I wish you well with your research study. Should you have further enquiries on this matter, please contact Jonathan Howcroft, Policy and Research Officer, Education Policy and Research, by telephone on (03) 9947 1892 or by email at <howcroft.jonathan.j@edumail.vic.gov.au>.

Yours sincerely



Dr Elizabeth Hartnell-Young
Group Manager
Education Policy and Research

08/07/2010

enc

Appendix C Ethics Approval: Catholic Education Office

Catholic Education Office

In reply please quote:

GE10/0009
1610

20 May 2010

Prof P Wilson
RMIT University
GPO Box 2476V
MELBOURNE VIC 3001

Dear Prof Wilson

I am writing with regard to your research application received on 19 May 2010 concerning your forthcoming project titled *The development of rapid on line motor control in children*. You have asked approval to approach Catholic primary schools in the Archdiocese of Melbourne, as you wish to have students and parents participate in your research.

I am pleased to advise that your research proposal is approved in principle subject to the nine standard conditions outlined below. Additionally I ask that you forward to this Office a copy of the notification of approval from the University's Ethics Committee when it becomes available.

1. The decision as to whether or not research can proceed in a school rests with the school's principal, so you will need to obtain approval directly from the principal of each school that you wish to involve.

2. You should provide each principal with an outline of your research proposal and indicate what will be asked of the school. A copy of this letter of approval and a copy of notification of approval from the university's Ethics Committee, should also be provided.

3. A *Working with Children* (WWC) check – or registration with the Victorian Institute of Teaching (VIT) – is necessary for all researchers visiting schools. Appropriate documentation must be shown to the principal before starting the research in each school.

4. No student is to participate in the research study unless s/he is willing to do so and informed consent is given in writing by a parent/guardian.

5. You should provide the names of schools which agree to participate in the research project to the Knowledge Management Unit of this Office.

6. Any substantial modifications to the research proposal, or additional research involving use of the data collected, will require a further research approval submission to this Office.

1 of 2

7. Data relating to individuals or schools are to remain confidential.

8. Since participating schools have an interest in research findings, you should consider ways in which the results of the study could be made available for the benefit of the school communities.

9. At the conclusion of the study, a copy or summary of the research findings should be forwarded to this Office. It would be appreciated if you could submit your report in an ***electronic format*** using the email address provided below.

I wish you well with your research study. If you have any queries concerning this matter please contact Mr Martin Smith of this Office.

The email address is <km@ceomelb.catholic.edu.au>.

Yours sincerely

Nancy Bicchieri
DEPUTY DIRECTOR

Appendix D Plain Language Statement for School Principals – Studies 1-3

INVITATION TO PARTICIPATE IN A RESEARCH PROJECT
PROJECT INFORMATION STATEMENT

Project Title:
Development of rapid, online motor control in children

Investigators:

- Associate Prof. Peter Wilson (Principle Investigator: Psychology, RMIT University, peter.h.wilson@rmit.edu.au, (03) 9925 2906.)
- Prof. Jan Piek (Principle Investigator, School of Psychology and Speech Pathology, Curtin University, J.Piek@curtin.edu.au)
- Prof David Sugden (Principle Investigator, School of Education, Leeds University, d.a.sugden@education.leeds.ac.uk)
- Mr Scott Ruddock (BSocSc (Psych), Honours Student, RMIT)
- Miss Rhianna Mann (BSocSc (Psych), Honours Student, RMIT)
- Mr Henry Bell (BSocSc (Psych), Honours Student, RMIT)
- Mr Christian Hyde (BSc, Grad. Dip. Psych, PhD Candidate)
- Ms Daniela Rigoli (BA Psychology – Honours, Curtin University)

Dear <Insert Name of Principal of School>,

Your school 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 children from your school to be approached 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?
Our names are Scott Ruddock, Rhianna Mann, Henry Ball, Daniela Rigoli, and Christian Hyde and we are conducting a research project with Associate Professor Peter Wilson in the School of Psychology which has been funded by the Australian Research Council (ARC). This means that we will be preparing a research report from the results of this study. We would like to invite children from your school to participate in this research subject to their parent's written consent. This project has been approved by the RMIT University Human Research Ethics Committee and <insert relevant educational body>.

What is the project about? What are the questions being addressed?
Our project examines how children learn motor skills and the strategies they use to assist them. This knowledge will also help us understand why some children have more difficulty performing movements than others. To do this we will assess children at different points over time, and examine how their performance changes with age.

If I agree for my school to participate, what will those children who are involved be required to do?
Children's motor skills will first be assessed using a small set of movement tasks. These include manual skills like bead threading and larger skills like standing broad jump. Children will also complete a set of computer-based tasks assessing thinking skills and speed. Using a small tablet PC, children will be asked to press keys in response to a set of playing cards displayed on the screen. For example, they will be asked to hit a YES key whenever they see a red card, or decide if a displayed card is the same as one displayed previously. Finally, they will be asked to point and touch targets displayed on a larger touch screen as they appear. We will assess their speed and accuracy on these tasks. Finally, parents will also be asked to complete a short questionnaire about their child's participation in physical activities and factors that may impact on this.

Since we are interested in changes in the strategies that people use to perform movements over time, children will be assessed *once every 6 months for a period of 2 years* (5 times in total). Each session will take roughly 30 to 45 minutes to complete and be conducted at school.

What are the risks or disadvantages associated with participation?
Very occasionally, people find being assessed uncomfortable or upsetting. If at any stage during the study your child feels uncomfortable or upset about the tasks, they are encouraged to let the researcher know and the assessment will cease.

What are the benefits associated with participation?
Children will find the tasks both enjoyable and challenging. They will be aware that their participation will help us add to knowledge about the way children and adults learn new skills, and why some children find it difficult. There will be no financial benefit or reward for participating in this study.

What will happen to the information provided by the research?
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 children's names will not appear on any of the data. A code number will be assigned each child's data. The consent forms will not be kept in the same place as each child's results so there will be no way to identify which results have been obtained of each 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, each child's anonymous data may be used for other projects in this area. All data will be completely anonymous and each child's identity will not be disclosed.

What are the rights of my students as participants?
As this study is completely voluntary, children and their parents are under no obligation to consent to participation and children may withdraw at any stage for any reason. Further, children have the right to ask questions regarding the project at any time.

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

Yours sincerely,

A/Prof Peter Wilson
BAppSc (PE), BBSc (Hons), PhD

A/Prof Jan Piek
BSc (Hons), PhD

A/Prof David Sugden
PhD

Scott Ruddock
B/SocSc (Psychology)

Rhianna Mann
BSocSc (Psych)

Henry Bell
BSocSc (Psych)

Daniela Rigoli
BA Psychology (Honours)

Christian Hyde
Bachelor of Science, Grad. Dip. Psych.

Any complaints about children from your school'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 E Plain Language Statement, Consent Form for Parents – Studies 1-3

INVITATION TO PARTICIPATE IN A RESEARCH PROJECT
PROJECT INFORMATION STATEMENT

Project Title:
The development of rapid online motor control in children

Investigators:

- Associate Prof. Peter Wilson (Principle Investigator: Associate Professor, Psychology, RMIT University, peter.h.wilson@rmit.edu.au, (03) 9925 2906.
- Prof. Jan Piek (Principle Investigator, School of Psychology and Speech Pathology, Curtin University, J.Piek@curtin.edu.au)
- Prof David Sugden (Principle Investigator, School of Education, Leeds University, d.a.sugden@education.leeds.ac.uk)
- Mr Scott Ruddock (BSocSc (Psych), Honours Student, RMIT)
- Miss Rhianna Mann (BSocSc (Psych), Honours Student, RMIT)
- Mr Henry Bell (BSocSc (Psych), Honours Student, RMIT)
- Mr Christian Hyde (BSc, Grad. Dip. Psych, PhD Candidate)
- Ms Daniela Rigoli (BA Psychology – Honours, Curtin University)

Dear Parent,

Your child has been invited to participate in a research project being conducted by RMIT University in partnership with Curtin University (WA) and Leeds University (UK). 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?
Associate Prof. Peter Wilson from the Discipline of Psychology at RMIT University leads a team of investigators (listed above) on this project, which is funded by the Australian Research Council (ARC). The project is designed to add to our understanding of how children acquire motor skills and some of the potential barriers. We will be preparing a number of interesting research reports 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 (HREC).

Why has my child been approached?
The Principal of your child's school has agreed to allow us to approach students to invite them to participate in our project.

What is the project about? What are the questions being addressed?
Our project examines how children learn motor skills and the strategies they use to assist them. This knowledge will also help us understand why some children have more difficulty performing movements than others. To do this we will assess children at different points over time, and examine how their performance changes with age.

If I agree for my child to participate, what will they be required to do?
Your child's motor skills will first be assessed using a small set of movement tasks. These include manual skills like bead threading and larger skills like standing broad jump. Your child will also complete a set of computer-based tasks assessing thinking skills and speed. Using a small tablet PC, children will be asked to press keys in response to a set of playing cards displayed on the screen. For example, they will be asked to hit a YES key whenever the see a red card, or decide if a displayed card is the same as one displayed previously. Finally, they will be asked to point and touch targets displayed on a larger touch screen as they appear. We will assess their speed and accuracy on these tasks. Finally, you will also be asked to complete a short questionnaire about your child's participation in physical activities and factors that may impact on this.

Since we are interested in changes in the strategies that people use to perform movements over time, your children will be assessed *once every 6 months for a period of 2 years* (5 times in total). Each session will take roughly 30 to 45 minutes to complete and be conducted at school.

What are the risks or disadvantages associated with participation?
Very occasionally, people find being assessed uncomfortable or upsetting. If at any stage during the study your child feels uncomfortable or upset about the tasks, they are encouraged to let the researcher know and the assessment will cease.

What are the benefits associated with my child's participation?
Your child will find the tasks both enjoyable and challenging. Your child will be aware that their participation will help us add to knowledge about the way children and adults learn new skills, and why some children find it difficult. 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 A/Prof. 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 A/Prof. Peter Wilson on the contact details above.

Yours sincerely,

A/Prof Peter Wilson
BAppSc (PE), BBSc (Hons), PhD

A/Prof Jan Piek
BSc (Hons), PhD

A/Prof David Sugden
PhD

Scott Ruddock
B/SocSc (Psychology)

Rhianna Mann
BSocSc (Psych)

Henry Bell
BSocSc (Psych)

Daniela Rigoli
BA Psychology (Honours)

Christian Hyde
Bachelor of Science, Grad. Dip. Psych.

Portfolio **Science, Engineering and Health**
School of **Health Sciences**
Name of participant:
Project Title: The development of rapid online motor control in children

Name(s) of investigators(1)	**A/Prof Peter Wilson**	Phone:	c/o (03) 9925 2906
(2)	**Prof Jan Piek**	Phone:	c/o (03) 9925 2906
(3)	**Prof David Sugden**	Phone:	c/o (03) 9925 2906
(4)	**Scott Ruddock**	Phone:	c/o (03) 9925 2906
(5)	**Christian Hyde**	Phone:	c/o (03) 9925 2906
(6)	**Rhianna Mann**	Phone:	c/o (03) 9925 2906
(7)	**Christian Hyde**	Phone:	c/o (03) 9925 2906
(8)	**Daniela Rigoli**	Phone:	c/o (03) 9925 2906

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.

I consent to the participation of _____ in the above project

Signature: (1) (2) Date: _____

(Signatures of parents or guardians)

Witness: _____ Date: _____

(Witness to signature)

PLEASE RETURN YOUR SIGNED CONSENT FORM BACK TO YOUR CHILD'S CLASS TEACHER

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 F Consent Form for Older Children – Studies 1-3

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

Hello, our names are Dr Jan Piek, Dr David Sugden, Scott Ruddock, Rhianna Mann, Henry Ball, Daniela Rigoli and Christian Hyde and we would like to invite you to participate in a project that we are 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 most children really enjoy like threading beads, balancing on one leg, and jumping as far as you can. We will also ask you to play some games on a computer. On one game you will touch playing cards as quickly as you can as they appear on a computer screen. On another you will try to find a hidden path through a maze, and remember objects that appear on the screen. Last, you will touch targets as they jump from one place to another.

What if I do NOT 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 below.

Thank you. ☺

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

Participant's name .....

Signature.....
Date...../ ..... /.....

Appendix G Consent Form for Younger Children – Studies 1-3

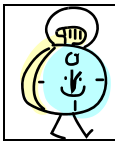
INVITATION TO PARTICIPATE IN A RESEARCH PROJECT
PROJECT INFORMATION SHEET- CHILD VERSION

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

Who is running this project?
- Dr Peter Wilson from RMIT University peter.h.wilson@rmit.edu.au, tel. 9925-2906, Dr Jan Piek, Dr David Sugden, Scott Ruddock, Rhianna Mann, Henry Bell, Daniela Rigoli and Christian Hyde.

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.

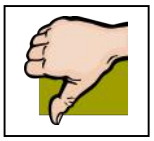
	You will be asked to do some activities that most children really enjoy like threading beads, balancing on one leg, and jumping as far as you can. We will also ask you to play some games on a computer. On one game you will touch playing cards as quickly as you can as they appear on a computer screen. On another you will try to find a hidden path through a maze, and remember objects that appear on the screen. Last, you will touch targets as they jump from one place to another.	

	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?

YES 

OR

NO! 

Yes, I would like to do the activities. – Please sign the form ✓

No – That's ok!

Name: _____ **Date:** _____

THANK YOU ☺

Appendix H Child Development Parent Questionnaire – Studies 1-3

Child Development Questionnaire for Parents/Guardians

The following questionnaire is designed for parent/s who have agreed for their child to participate in the RMIT University study, *'The development of rapid on-line control in children'*. Parents will be asked to answer some questions about their child's development, level of physical activity, and other things associated with physical activity. Your answers will be confidential so please answer them as accurately as possible. If for any reason you do not wish to complete one or more question/s, just go to the next question.

Identifying Information

Your Name: _____

Please provide the following details about your child:

Name: _____

Date of Birth: / /

Gender: Male ☐ Female ☐

Background Information and Participation

Question 1

Has your child had any difficulties learning movement skills? Yes ☐ No ☐

If YES, have these movement difficulties affected any of the following:

Their ability to complete school work	Yes ☐ ☐	No
Their ability to perform everyday activities at home (e.g., dress themselves, clean their teeth or cut their food)	Yes ☐ ☐	No
Their ability to participate in recreational activities involving movement (i.e., sport, free play, music lessons, etc.)	Yes ☐ ☐	No

Question 2

Has your child ever been diagnosed with a major medical condition (e.g., asthma, epilepsy, etc.)? Yes ☐ No ☐

If YES, please specify the condition(s):

_____

_____

_____

_____

Question 3
Has your child ever been diagnosed by a health professional with one/or more of the following:

	Tick if Yes
• Motor Coordination Problems	☐
• ADHD	☐
• Conduct Disorder	☐
• Autism Spectrum Disorder (i.e. Asperger's Syndrome or Autism)	☐
• Dyslexia	☐
• Specific Language Impairment	☐
• Other Learning Disorder	☐
• Intellectual Disability	☐

Is your child receiving support for a learning disability? Yes ☐
No ☐
If YES, please specify the disability and type of support:

_____
_____

Is your child receiving ongoing support for any other disability? Yes ☐
No ☐
If YES, please specify the disability and type of support:

_____
_____

Question 4
This question asks you to think about all your child's physical activities in the past month.
(i) List the **organised physical activities** that your child has participated in during this time (like netball, football, dancing, lessons, etc.):

_____
_____

How many hours a week? _____ hours.

(ii) List the types of **free play involving physical activity** that your child has participated in during this time (like hide and seek, chasing games, climbing, etc.):

_____
_____

How many hours a week? _____ hours.

Question 5
Does your child participate in any seasonal physical activities that they may not have done in the past month? (e.g., football, skiing, swimming) Yes ☐
No ☐
Types of seasonal activity (not listed in Q.4): _____
_____
_____

Hours per week: _____

Question 6
Please rate your child's **interest in participating** in organised physical activities (like netball, football, dancing lessons, etc.) or free play involving movement?

☐	☐	☐	☐	☐
Very Disinterested	Somewhat disinterested	Neutral	Somewhat interested	Very interested

Question 7
Please rate your child's **skill level** when performing physical activities (like those referred to in Question 4 and 5)?

☐	☐	☐	☐	☐
Well below average	Somewhat below Average	Average	Somewhat above average	Well above average

Appendix I Published Article in *Cognitive Development* – Study 1

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Cognitive Development

Hot executive function in children with Developmental Coordination Disorder: Evidence for heightened sensitivity to immediate reward

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ABSTRACT

Deficits of cool executive function (EF) have been shown in children with motor problems (or Developmental Coordination Disorder – DCD), but little is known of hot EF in this group. Given some evidence of poor self-regulation in DCD, we predicted poorer performance on a measure of hot EF, the Hungry Donkey Task (HDT), relative to typically developing (TD) children. Participants were 14 children with DCD and 22 TD children aged between 6.5 and 12 years. The DCD group performed significantly worse than the TD group on a 100-trial version of the HDT, making more selections from disadvantageous options and less from advantageous ones. Within-group analyses showed that children with DCD had faster responses to disadvantageous options than to advantageous. These results suggest high sensitivity to immediate reward in DCD. This sensitivity may reflect a more generalized deficit in the ability to resist the rewarding aspects of emotionally significant stimuli.

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0885-2014/

1. Introduction

Developmental Coordination Disorder (DCD) is a neurodevelopmental disorder characterized by problems with motor coordination and skill acquisition that significantly disrupt the daily living activities and/or academic achievement of children (APA, 2013; DSM-V). The clinical diagnosis of DCD is motor coordination significantly lower than expected from chronological age and intellectual ability, and also not due to a pervasive developmental delay or medical conditions such as cerebral palsy or muscular dystrophy. DCD is not a trivial disorder. It affects about 5–6% of school-aged children and is associated with a range of psychosocial and behavioural problems, such as low academic achievement, poor social interaction, poor self-concept, and higher incidence of psychological disorders (Missiuna, Moll, King, Stewart, & McDonald, 2008; Rigoli, Piek, & Kane, 2012). Numerous studies exist of the underlying motor control and learning issues that may explain the disorder, but very few unifying accounts have been put forward. However, a recent quantitative review of the literature by Wilson, Ruddock, Smits-Engelsman, Polatajko, and Blank (2013), compared the performances of children with DCD and typically developing (TD) children on behavioural measures, and revealed that deficits in DCD tend to coalesce around several aspects of control, including predictive control, ability to develop stable coordination patterns, and executive function (EF), the last of these the focus of the present study.

1.1. Executive function deficits in DCD

Given that the control of action is supported by a complex and interactive network of neural structures, motor coordination problems are likely to be constrained by not only motor processes but also cognitive and affective ones (Alloway, 2007; Rigoli, Piek, Kane, & Oosterlaan, 2012a; Rigoli, Piek, Kane, & Oosterlaan, 2012b), for example visual attention and EF (Dewey, Kaplan, Crawford, & Wilson, 2002; Green, Baird, & Sugden, 2006), consistent with both modern information processing theory and recent advances in the cognitive neuroscience of action. For example, in Sergeant's (2000) cognitive-energetic model, EF refers to the complex array of neurocognitive processes that support the conscious and goal-directed control of thought, emotion, and action (Zelazo & Carlson, 2012). In this model, cognitive control and motor behaviour are intertwined. From the perspective of interactive specialization (Johnson & Munakata, 2005), cognition and action become increasingly coupled over the course of development. Initially distinct systems interact according to the timescales of neural maturation and the moderating effect of experience. From this perspective, there are two prime hypotheses about EF deficits in DCD. The first is that the biological process by which specific neurocognitive and neuromotor systems mature is impaired, and the second is that the emerging neural systems are not stimulated (via appropriate learning experiences) in a way that promotes coupling between specialized sub-systems (e.g., the modulating effect of frontal planning on more primitive limbic structures that resolve stimulus reward).

Studies of clinical populations, such as children with DCD (Piek, Dyck, Francis, & Conwell, 2007) or ADHD (Barkley, 1997; Sergeant, 2000), and non-clinical TD children (Pennequin, Sorel, & Fontaine, 2010) support the interactive relation between cognition and action. We see ample indication that poor coordination, such as in children with DCD, is associated with deficits in EF. Wilson et al. (2013) identified very large effect sizes across core domains of EF, including working memory (WM) ($d = 1.07$, averaged over visuospatial and verbal), inhibitory control (1.03), and executive attention, which includes deficits in set-shifting and planning (1.46). Most striking was degree of generalized executive dysfunction (operationalized by performance deficits on different EF tasks), in excess of that reported in children with ADHD (Piek, Dyck, et al., 2007).

1.2. Are deficits of EF in DCD confined to 'cool' EF?

The executive functions studied in children with DCD are generally grouped under the label 'cool' EF. Cool EF is associated with lateral prefrontal cortex (L-PFC) and is required in situations characterized by abstract, decontextualized stimuli with no affective or motivational component (Zelazo & Carlson, 2012). Recent research has broadened the conceptualization of EF to include neurocognitive processes

APPENDICES

that operate in emotion-laden or motivation-laden situations. These contexts are likely to elicit 'hot' affective aspects of EF, mainly associated with ventromedial prefrontal cortex (VM-PFC) (Zelazo & Muller, 2011). Hot EF facilitates flexible reappraisal of emotionally significant stimuli, and assists decision-making when a task involves some affective or motivational component (Prencipe et al., 2011).

Deficits of hot EF are linked to poor ability in anticipating future consequences of actions, poor impulse- and self-control, and, consequently, poor affective decision-making, and even low academic achievement (Casey et al., 2011). Patterns of deficit in different aspects of EF differ across disorders. Autism may be considered primarily as a disorder of hot EF with secondary deficits of cool EF (Zelazo & Muller, 2002; cf. Dawson, Meltzoff, Osterling, & Rinaldi, 1998), while ADHD may be associated with cool EF deficits (Zelazo & Carlson, 2012). However, this variability will also depend on the comorbidity of the disorder see Dinn, Robbins, & Harris, 2001). There is a lack of evidence as to whether EF problems in DCD are confined to cool EF or extend to both cool and hot EF. Accordingly, current interventions for DCD do not address potential deficits in affective decision-making. Given that different behavioural and psychological problems (e.g., internalizing and externalizing disorders) that are associated with DCD) are also linked to the deficits of hot EF (Casey et al., 2011; Dolan & Lennox, 2013; Must et al., 2006), and considering the important contribution of hot EF to functioning in daily life activities, there is a need to better understand hot EF in DCD in order to inform interventions and consequently improve functioning and quality of life for those diagnosed with DCD.

1.3. Assessment of hot EF

Measures of hot EF (e.g., delay discounting or gambling/card tasks) generally involve some obvious rewards and losses (Zelazo & Carlson, 2012), unlike the cool EF tasks where there is little to be gained or lost (e.g., Wisconsin Card Sorting Test; Grant & Berg, 1948). Evidence from neuroscientific research supports the construct of hot EF and shows that hot and cool EF are dissociable. For example, patients with lesions to orbitofrontal cortex perform poorly on the Iowa Gambling Task (IGT; Bechara, Damasio, Damasio, & Anderson, 1994), but not the Wisconsin Card Sorting Test (Bechara, 2004; Eslinger, Flaherty-Craig, & Benton, 2004).

Indeed, the IGT is one of the most widely used hot EF tasks that simulates uncertainties of decision-making in real life by necessitating the weighing of potential rewards and losses. Participants are required to repeatedly select cards from four different card decks. Two decks (A & B) are characterized by high constant gain; however, deck A has a high loss in 50%, and B a very high loss in 10% of the trials (disadvantageous options). The other two (C & D) are characterized by low immediate gain. Deck C has a low loss in 50%, and D a high loss in 10% of the trials (advantageous options). Therefore, amount of constant gain, frequency of loss, and amount of unpredictable loss are the three properties of each deck. Although many healthy adults gradually opt for the advantageous options, patients with lesions of VM-PFC opt for disadvantageous options (Bechara et al., 1994). Given the protracted development of PFC, the failure to anticipate future consequences of actions is also observed in children and adolescents (Smith, Xiao, & Bechara, 2012).

An age-appropriate analogue of the IGT for children and adolescents is the Hungry Donkey Task (HDT; Crone & van der Molen, 2004), in which participants help a donkey win as many apples as possible. Older children and adolescents up to age 15 often opt for options with low frequency loss (Huizinga, Crone, & Jansen, 2007; van Duijvenvoorde, Jansen, Visser, & Huizinga, 2010). Children younger than 12, however, either choose randomly or prefer options with high immediate reward (A & B). These patterns of performance differentiate hot EF abilities not only in TD children, but also in children with developmental disorders (Geurts, van der Oord, & Crone, 2006). Moreover, WM has also been linked to IGT/HDT performance in some studies (Hinson, Jameson, & Whitney, 2002); therefore, investigation of hot EF in children with and without DCD needs to control for WM.

1.4. Deficits of hot EF in children with DCD

Inhibitory control, which is disrupted in children with DCD, is a significant predictor of affective decision-making (van Duijvenvoorde, Jansen, Bredman, & Huizinga, 2012). A deficit of response

APPENDICES

inhibition can exacerbate the consequences of hot EF deficits, reflected in poor self-control (Casey et al., 2011) and self-regulation (Zelazo & Carlson, 2012). Indeed, children with DCD are at risk for a range of emotional and behavioural problems associated with poor impulse control (Dewey et al., 2002) such as aggression (Tseng, Howe, Chuang, & Hsieh, 2007), conduct problems generally (Kanioglou, Tsorbatzoudis, & Barkoukis, 2005), and poor (motor) self-regulation (Sangster Jokic & Whitebread, 2011).

Moreover, the involvement of particular brain structures in both motor coordination and hot EF may underlie possible deficits of hot EF in DCD. The high incidence of internalizing disorders and emotional problems in DCD may be attributed to common neurodevelopmental causes, such as cerebellar dysfunction (Cairney, Veldhuizen, & Szatmari, 2010). Cerebellar activity does not only facilitate motor coordination, but also appears to be involved in the regulation of emotion and mood (Schmahmann & Caplan, 2006; Schutter & van Honk, 2009) and is linked to better performance on the IGT (Ernst et al., 2002). Reciprocal connections between the cerebellum and both the limbic system structures, such as the amygdala, and the prefrontal areas of the cerebral cortex provide some neuroanatomical evidence for the involvement of the cerebellum in the control of mood and emotion (Schutter & van Honk, 2009). Therefore, the high incidence of social and emotional problems, along with the involvement of the cerebellum in emotion regulation, suggest that children with DCD might show deficits of hot EF compared to their same-age TD peers.

1.5. Aim and hypothesis

The aim of this study was to investigate hot EF in children with and without DCD, using an age-appropriate test of hot EF. We predicted that children with DCD would show an impaired pattern of affective decision-making and perform less well than their same-age TD peers on the HDT.

2. Method

2.1. Participants

The sample consisted of 14 children (6 girls) in the DCD group ($M = 9.03$, $SD = 1.59$, range 6.7–11.9) and 22 TD children (15 girls) in the control group ($M = 9.02$, $SD = 1.73$, range 6.6–11.7). The DCD group included children who scored at or below 85 on the Neurodevelopmental Index (NDI) of the McCarron Assessment of Neuromuscular Development (MAND; McCarron, 1997). These children are identified as being at risk for DCD. The average NDI in the DCD group was 75.18 ($SD = 8.64$, range: 51–85). The control group consisted of those who had an NDI of 100 or above ($M = 108.63$, $SD = 7.04$, range: 100–130). Given that sometimes up to 50% of children with DCD also meet the diagnostic criteria for ADHD, and links exist between DCD and learning disabilities (Zwicker, Missiuna, Harris, & Boyd, 2012), children with a current or past diagnosis of ADHD, learning, neurological, or any other physical disorder were excluded. All children were recruited from two mainstream primary schools in Australia. Their IQ levels were assumed to be within the normal range, eliminating any need to conduct neurological or medical examination to ensure the criterion C of the DCD diagnosis in DSM-IV-TR (APA, 2000) was met (Geuze, Jongmans, Schoemaker, & Smits-Engelsman, 2001).

2.2. Materials

2.2.1. Hungry Donkey Task

The HDT (Crone & van der Molen, 2004) is a computerized, developmentally appropriate analogue of the IGT in which children are asked to help a hungry donkey win as many apples as possible. Each trial consists of a stimulus and an outcome display. The stimulus display shows a donkey sitting in front of four doors. Each door corresponds to a key on the keyboard (door A: A, door B: S, door C: K, and door D: L). Upon pressing one of the keys, participants see the outcome display showing the number of intact, green apples won and – in some trials – crossed, red apples lost. A vertical bar on the side of the screen presents a graphical index of performance while the amount of overall gain is displayed under the doors. The task includes 100 trials and retains the basic format of the IGT in that two options (A &

B) are characterized by high immediate gain (4 apples) but also high loss (*disadvantageous*), while the other two (C & D) are characterized by low immediate gain (2 apples), but low loss (*advantageous*). In every 10 trials, door A presents five unpredictable losses of 8, 10, 10, 10, and 12 apples, while door B present one unpredictable loss of 50 apples, leading to an overall loss of 10 apples for each of these doors. Door C, however, leads to five unpredictable losses of 1, 2, 2, 2, and 3 apples in every 10 trials, while door D has one unpredictable loss of 10 apples. Therefore, the net gain on every 10 trials of doors C and D is also equal – 10 apples. Similar to the IGT, net score is the main outcome measure. Participants are not told the properties of each door or the number of trials. They are, however, informed that they have to play many times and that they can switch doors as often as they like.

2.2.2. McCarron assessment of neuromuscular development

The MAND (McCarron, 1997) is a standardized test of motor skills comprising five tests of fine motor skills and five of gross motor skills. The sum of the scaled scores on each of these 10 tests is compared to age-appropriate norms (available for individuals aged between 3.5 and 18 years) to determine the NDI score. The test-retest reliability of the MAND (over a 1-month period) ranges between .67 and .98 (McCarron, 1997). The MAND has acceptable criterion validity and concurrent validity (McCarron, 1997), as well as good specificity and sensitivity (Tan, Parker, & Larkin, 2001).

2.2.3. One-back task

The One-Back Task is part of the CogState brief battery (Collie, Maruff, Falleti, Silbert, & Darby, 2002) and provides a measure of WM. The participant is instructed to press K (*Yes*) if the card presented in the centre of the laptop screen is exactly the same as the previous card, and to press D (*No*) if the card is different. A total of 42 cards are presented, with the task terminating after all the trials or after 3 min has elapsed. The yes or no response is correct in half of the 42 trials. Accuracy of responses is the main outcome measure, with a higher score indicating better WM. The One-Back Task demonstrates good construct validity and criterion validity (Maruff et al., 2009).

2.3. Procedure

The individual session was conducted in a quiet environment. It took approximately 10–15 min to administer the MAND, five min to complete the HDT, and five min to finish the One-Back Task. The tasks were administered in varied order across children. Some completed the HDT first and others the One-Back Task or the MAND.

2.4. Data analysis

Separate mixed-factorial analysis of variance (ANOVA) was used to compare groups on net score (per block & total) and reaction time (RT) to each response option. Although the control group had significantly better WM than the DCD ($M(SD)_{DCD} = 1.04\,(0.20)$, $M(SD)_{Control} = 1.24\,(0.15)$, $p = .002$, $d = 1.12$; r (NDI & WM) = .56, $p < 001$), WM was not considered a covariate because there was no significant correlation between net score and WM ($r = .13$, $p = .46$). Although males often outperform females on tests of hot EF like the IGT (van den Bos, Homberg, & de Visser, 2013) and HDT (Crone, Bunge, Latenstein, & van der Molen, 2005), a comparison using gender as a covariate revealed no covariate effects and as gender was not considered further. Moreover, neither gender nor age was significantly correlated with total net score, WM, or MAND NDI. For a recommended power of 0.80, and based on the expectation of at least a moderate effect size, we calculated a minimum needed sample size of 14–16 per group.

3. Results

3.1. Net score

Mean (±SE) net scores for each of the five blocks of 20 trials of the HDT are presented in Fig. 1, which shows that the control group had higher overall gain on all of the blocks. A 2 × 5 ANOVA – group (DCD vs. control) × block (blocks 1–5) – was conducted to assess whether the pattern of performance across the HDT was different between the two groups. We found no significant interaction between group and block, Wilks' $\Lambda = .96$, $F(4, 31) = 0.35$, $p = .84$, $\eta^2 = .04$. The simple main effect of

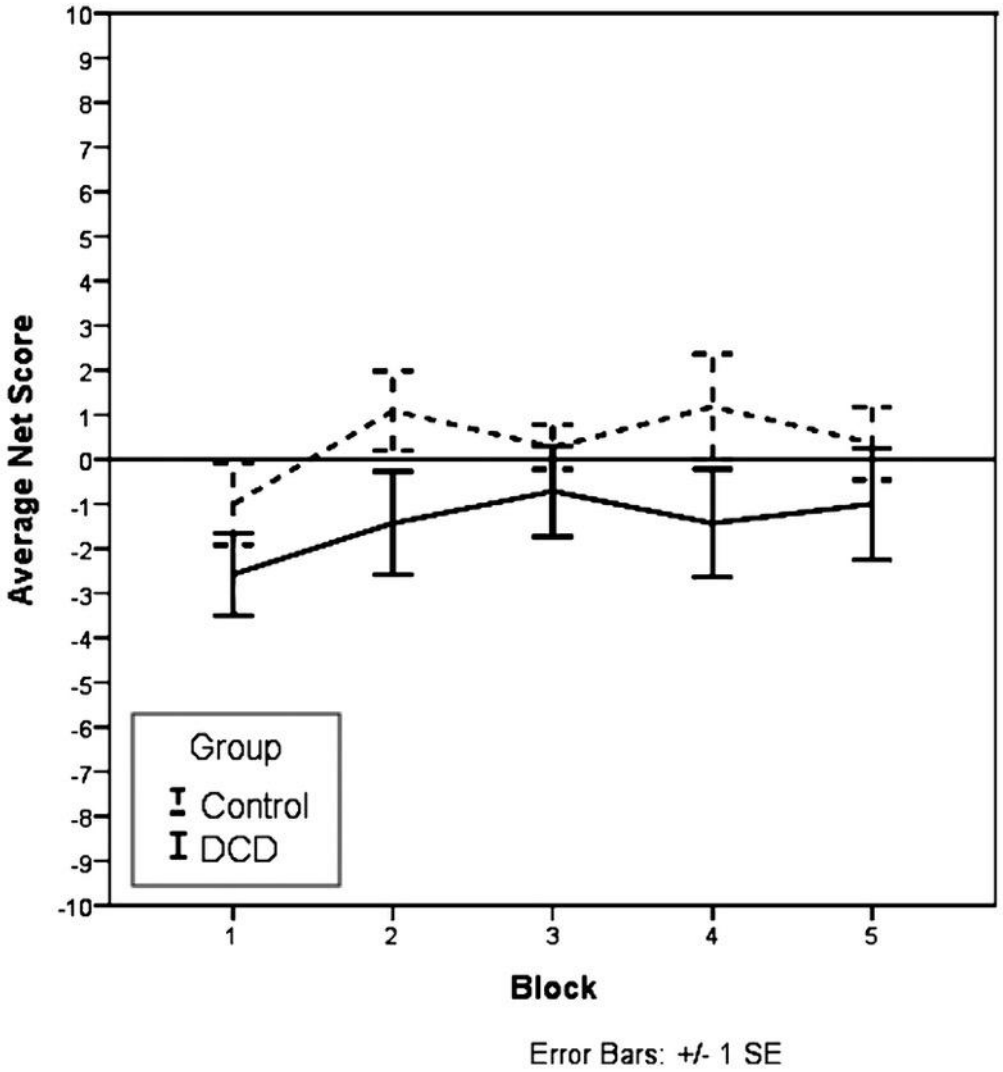

Fig. 1. Mean net scores of DCD and control groups in each block of the HDT.

block was also not significant ($p > .10$, $.03 < \eta^2 < .08$). However, the total net score was significantly lower for the DCD group, (M $(SD)_{DCD} = -7.14$ (11.55), M $(SD)_{Control} = 1.91$ (10.41), $p = .020$, $d = 0.82$ [95% CI = 0.12, 1.51]), and there was a significant positive relationship between NDI and total net score ($r = .42$, $p = .01$).

3.2. Choice strategy

To investigate whether preference for particular doors/options was responsible for the difference in net scores of DCD and control groups, we first calculated group averages (and SD) for the frequency of selecting each type of option: advantageous (low immediate reward): M $(SD)_{DCD} = 46.43$ (5.77), M $(SD)_{Control} = 50.95$ (5.20); disadvantageous (high immediate reward): M $(SD)_{DCD} = 53.57$ (5.77), M $(SD)_{Control} = 49.05$ (5.50). The interaction between group and option type (advantageous vs. disadvantageous) was significant, Wilks' $\Lambda = .85$, $F(1, 34) = 5.95$, $p = .020$, $\eta^2 = .15$. The DCD group had a significantly higher number of selections from the disadvantageous options than the advantageous ones ($p = .019$).

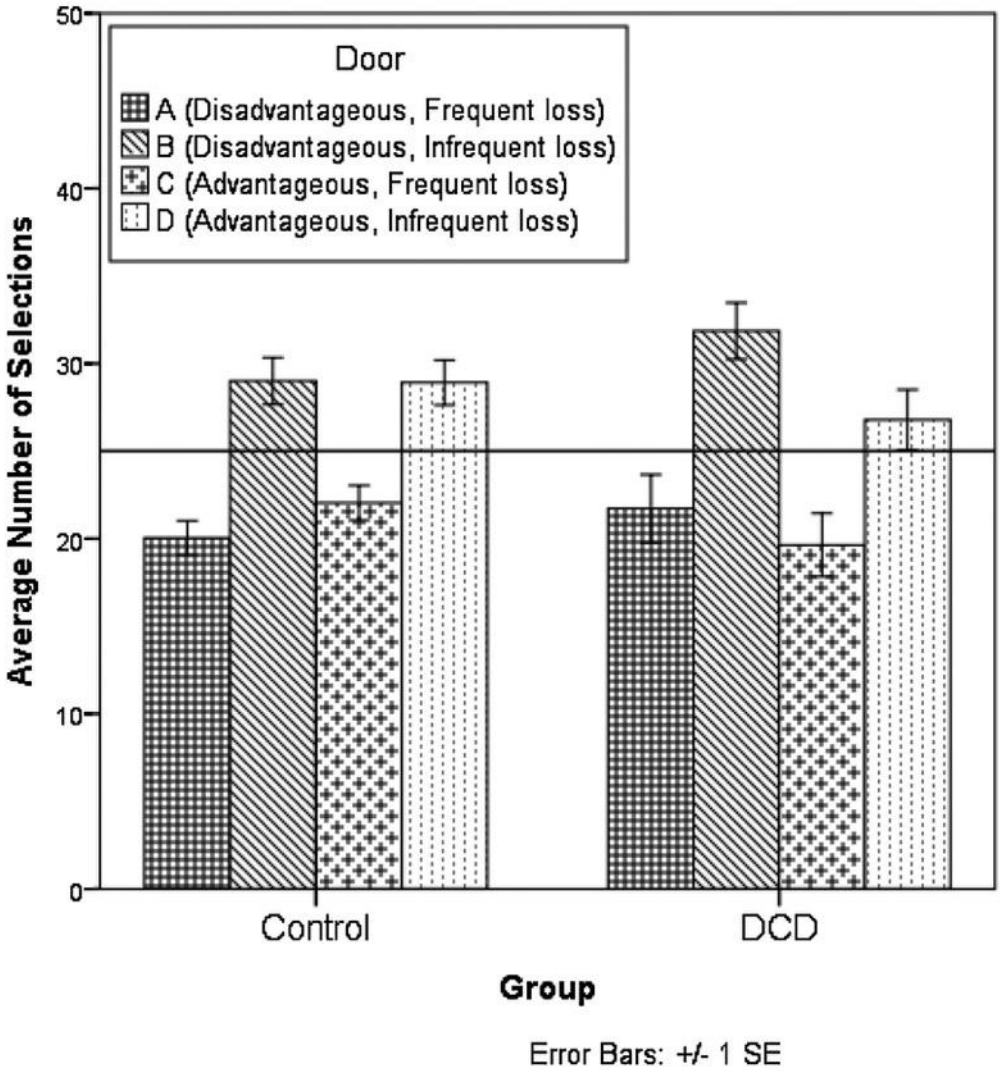

Fig. 2. Mean number of choices for each option as a function of group. The horizontal line represents chance performance, when each option would be chosen equally ($M = 25$).

Fig. 2 shows the average (±SE) number of times each option was approached. Both groups tended to select options with infrequent loss (i.e., B & D) more often; however, compared to the controls, the DCD group showed a higher tendency to approach the disadvantageous option B, and a lower preference for the advantageous option D. In the control group, the number of choices for all doors deviated significantly from the number that would be expected if choice were random, that is 100/4 = 25. The control group preferred options B ($p = .007$) and D ($p = .006$), and avoided options A ($p < .001$) and C ($p = .007$). In contrast, the DCD group only opted for B ($p = .001$). The advantageous option C was approached below the chance level ($p = .011$), while selections from A ($p = .12$) and D ($p = .32$) did not deviate from the chance level.

Fig. 3 depicts the average number of times each option was approached in each block of 20 trials. While the DCD group showed more tendency to approach disadvantageous options (A & B) throughout, the controls almost always approached the advantageous options (C & D) at a higher frequency than did the children with DCD. Moreover, feedback from earlier trials did not affect choice strategy. The DCD group, for example, had their highest number of selections from option B in block 4, which deviated significantly from the chance level (i.e., 20/4 = 5); $p = .009$). Likewise, while the controls approached option D at

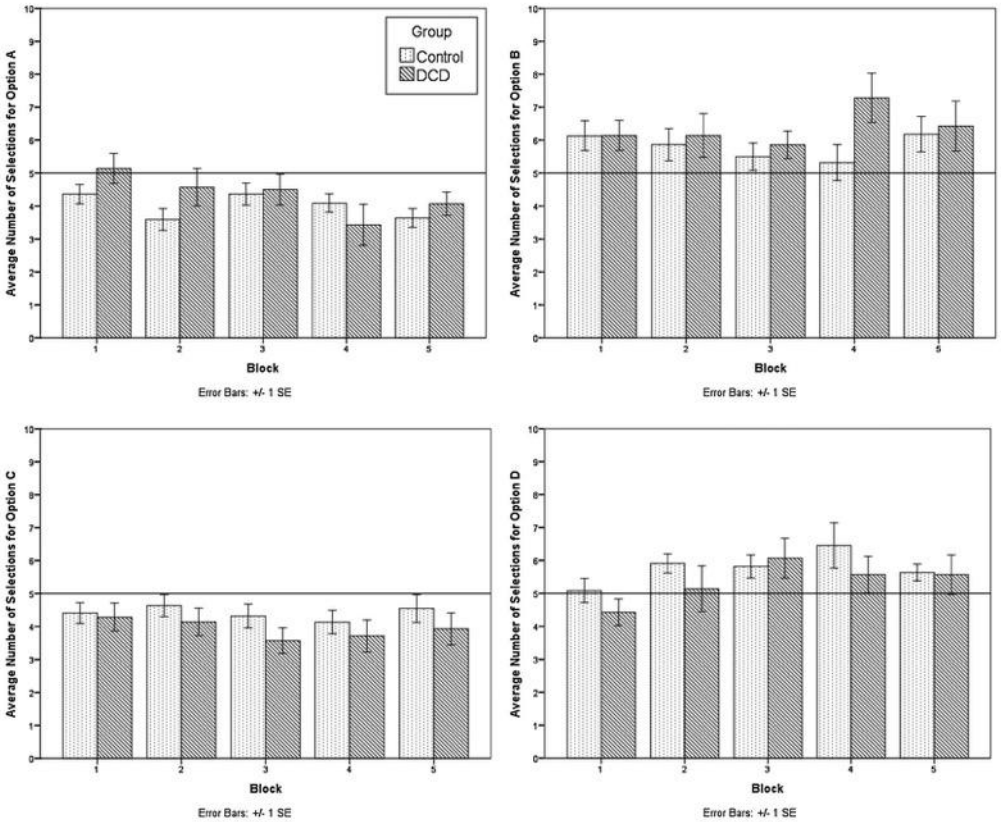

Fig. 3. Mean number of selections from each option in each block of 20 trials. The horizontal line represents chance performance ($M = 20/4 = 5$).

a significantly higher than chance level from block 2 onwards ($.005 < p < .047$), the frequency of selecting this option was never significantly different from chance level in the DCD group ($p > .10$).

3.3. *Reaction time (RT)*

We calculated the average RT of DCD and control groups to each door. For each child, the RTs that did not fall within 3 *SD* of the child's mean RT to the particular door were not included in analyses. The average RT of each group (in milliseconds) for each type of door/option was as follows: advantageous (low immediate reward): $M\ (SD)_{DCD} = 1179.69\ (368.77)$, $M\ (SD)_{Control} = 859.59\ (281.46)$; disadvantageous (high immediate reward): $M\ (SD)_{DCD} = 1019.04\ (335.49)$, $M\ (SD)_{Control} = 873.52\ (275.13)$.

There was a significant interaction between group and option type (advantageous vs. disadvantageous), Wilks' $\Lambda = .62$, $F\ (1, 29) = 18.17$, $p < .001$, $\eta^2 = .39$. Although the RT of control group did not depend on type of option ($p = .57$, $\eta^2 = .01$), the DCD group had significantly faster reactions to disadvantageous, high immediate reward options ($p < .001$, $\eta^2 = .45$). In a similar vein, the DCD group had a significantly longer RT than controls only for approaching advantageous, low immediate reward options ($p = .01$; door C: $p = .022$, $\eta^2 = .17$; door D: $p = .011$, $\eta^2 = .20$). We observed no changes in this pattern of group differences in RT across the 5 blocks.

The interaction between group and door (A, B, C, & D) was also significant, Wilks' $\Lambda = .59$, $F\ (3, 27) = 6.32$, $p = .002$, $\eta^2 = .41$. There was a significant difference ($p < .001$) between the fastest (disadvantageous option B: $M = 980$ ms, $SD = 406$ ms) and the slowest RT (advantageous option C: $M = 1233$ ms, $SD = 396$ ms) in the DCD group. In the control group, however, the fastest RT was to the advantageous option D ($M = 792$ ms, $SD = 289$ ms), significantly different ($p = .012$) from the slowest RT (option C: $M = 927$ ms, $SD = 302$ ms). Fig. 4, which presents means (±SE) of each group RT to the four options, also shows that the RT of DCD group to both disadvantageous, high immediate reward options was faster than their RT to the advantageous ones.

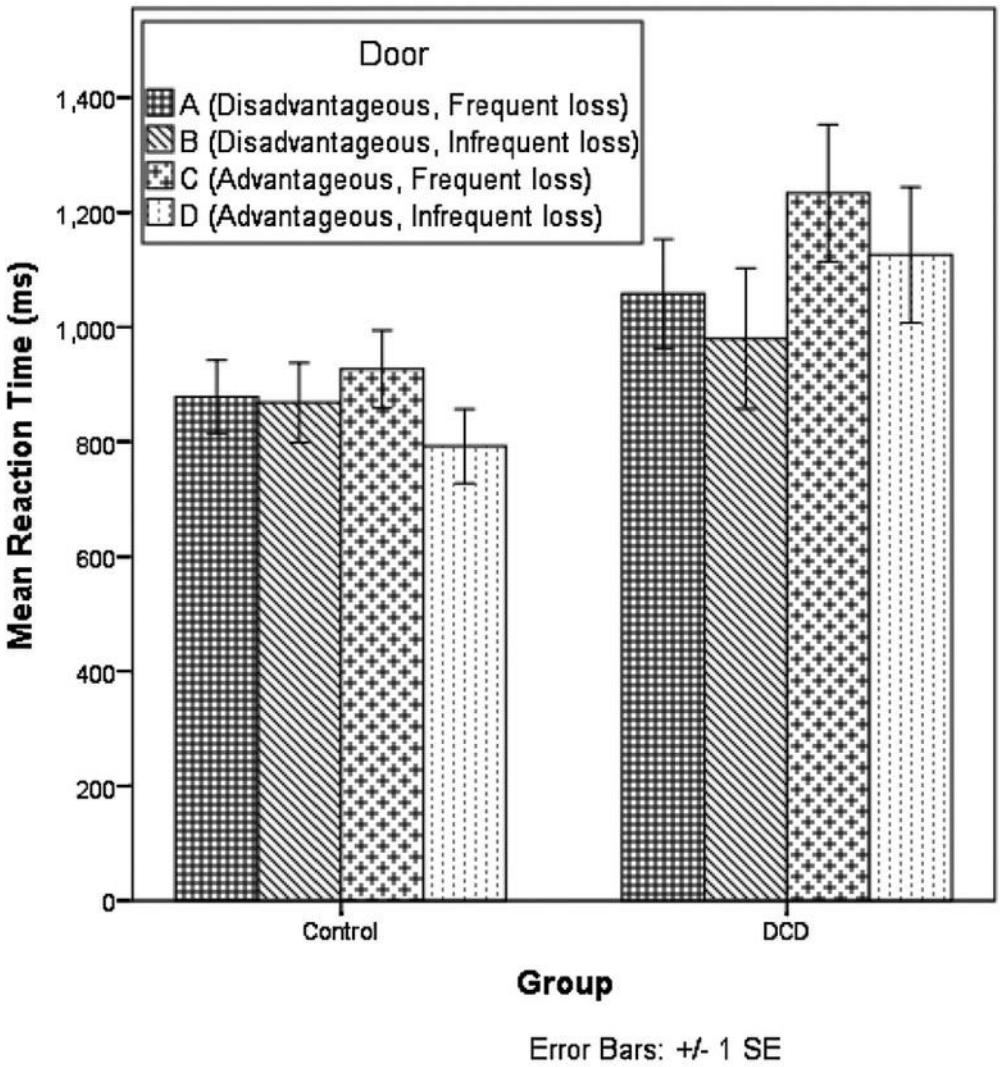

Fig. 4. Mean reaction time to each option in the DCD and control groups.

4. Discussion

The results supported the hypothesis that children with DCD would perform more poorly than controls on the HDT. Overall, the DCD group had significantly lower total net scores than controls, across blocks. In addition, children with DCD had a higher tendency to approach the disadvantageous options (high immediate reward) and a lower tendency to approach the advantageous ones (low immediate reward). In particular, the DCD group made less optimal choices by selecting the disadvantageous option B more often, but refraining from choosing the advantageous option D at a rate similar to controls. In contrast, the control group opted for both B and D – the options associated with low frequency loss. We discuss these group differences by conceptualizing the HDT within a proportional reasoning paradigm.

4.1. Choice strategy on the HDT

Bechara et al. (1994) argued that given the complexity of weighing all three dimensions of options on the IGT/HDT while making decisions, individuals rely on somatic markers (viz. somatic marker hypothesis; Damasio, 1994), and attempt the task using more intuitive decision processes. Dunn, Dalgleish, and Lawrence (2006), however, suggested that optimal outcomes on the IGT – or in this case the HDT – can be achieved if one simply considers the frequency and amount of loss, and ignores the constant gain. This means that the HDT can be framed as a proportional reasoning task, in which each option is characterized by two dimensions – a dominant (loss frequency) and a subordinate (amount of loss) – which must then be integrated to guide an appropriate response (Huizinga et al., 2007). Individuals progress through a series of developmental levels prior to using the appropriate strategy on a proportional reasoning paradigm (Siegler, 1981). This is shown developmentally by increasingly sophisticated rule use on the IGT/HDT. Young children often switch randomly between the four options, and attempt the task by guessing ('rule 0'). Hence, the probability of selecting any of the four options is equal. Later, children may consider the dominant dimension (i.e., frequency of loss). This improves the chances of selecting options B and D ('rule 1': B & D > 100/4). According to Huizinga et al. (2007), older children (10–12) and adolescents up to age 15 often use 'rule 1' k. However, the frequency of loss is equal between options B and D. A small group of adolescents, and about a third of adults, will then focus on the subordinate dimension (i.e., amount of loss) and opt for D, which leads to a positive overall outcome ('rule 2': D > 100/4). These observations suggests developmental changes in the complexity of rule use not only predict performance in contexts that are relatively devoid of emotional content or valence ("cool contexts"), but also contribute to advantageous decision-making about motivationally salient stimuli (such as the HDT), and afford better control over thoughts, impulses, and actions.

In our study, the frequency of loss was the dominant dimension for both DCD and control groups. More specifically, the opting of the control group for options B and D is similar to that shown in earlier studies of the IGT/HDT among same-age or older typical children (Huizinga et al., 2007; Prencipe et al., 2011). The DCD group, however, selected only option B above chance level (and not D as well); the disadvantageous option B yields infrequent loss, but also high immediate reward.

Although the loss frequency was the dominant dimension of choice for both groups, the amount of constant gain, instead of the unpredictable loss, became the subordinate dimension of choice for children with DCD. In other words, the choices made by these children were driven not only by a need to avoid frequent loss but also the desire to reap immediate rewards. This shows how the reward component of stimuli in motivationally salient contexts may influence cognitive processes such as proportional reasoning and response inhibition, both of which feed into cool and hot EF. That children with DCD were faster to respond to the two high immediate reward options (A & B) compared with low highlights the impact of emotionally salient stimuli in their affective decision-making, and suggests a heightened sensitivity to immediate reward. The important question here is whether the performance of the DCD group is due to some of their already acknowledged deficits of EF, such as inhibitory control (Wilson et al., 2013), or whether their performance represents an additional/distinct impairment.

4.2. High sensitivity to reward

The DCD group opted only for the disadvantageous, high immediate reward option B, and responded significantly faster to the disadvantageous options. For most chronometric tasks, children with DCD are slower than typical children (Piek & Skinner, 1999). We observed the same pattern of difference when the DCD group approached advantageous, low immediate reward options (C & D); however, it seems the propensity of the DCD group to approach high immediate reward (relative to controls) reduced the gap between the groups' RT to disadvantageous options (A & B). If the DCD group had not shown a preference for high immediate reward, we would have observed similar levels of group difference in RT in response to both types of choices. This differential pattern of performance suggests that children with DCD may have a heightened sensitivity to immediate reward and greater impulsivity. Indeed, disadvantageous performance on the IGT has been linked to faster RT to options A and B (Smith et al., 2012).

Both motor coordination problems and their psychosocial consequences reduce social interaction in children with DCD (Poulsen, Ziviani, Cuskelly, & Smith, 2007) and minimize their involvement in activities such as organized and unorganized play (Bouffard, Watkinson, Thompson, Causgrove Dunn, & Romanow, 1996), which are associated with meaningful reward. One hypothesis is that fewer chances to receive reward in real-life settings may then increase the sensitivity of children with DCD to rewarding stimuli. Reduced feelings of self-worth, social isolation, and poor social support may also contribute to a heightened sensitivity to positive cues. Depression, for instance, has been linked to higher sensitivity to reward in the IGT (Must et al., 2006), and children and adolescents with DCD are at greater risk for depression (Piek, Rigoli, et al., 2007; Rigoli, Piek, & Kane, 2012).

Deficits of hot EF are attributed to either an overactive emotional system or poor inhibitory skills (Bechara & van der Linden, 2005). Indeed, the processes of WM, response inhibition, and even set-shifting are all involved in affective decision-making, which could explain conflicting findings about the factor structure of EF (see Welsh & Peterson, 2014 for a review). These processes would still underpin the management and allocation of attention and memory when the performer must respond to emotionally salient stimuli; weighing the reward component of these stimuli may be the critical function of hot EF.

This conceptualization of EF in general may offer a better understanding of behaviour in context, compared with the traditional EF framework. To illustrate, immaturities of hot EF (e.g., poor self-control) during childhood or adolescence are explained by the interaction of cognitive control and emotion processing networks whereby the activity of emotion circuitry is not properly controlled by a developing PFC and its associated 'cool' processes, such as inhibitory control (Somerville, Hare, & Casey, 2011). Moreover, the degree to which one must draw on either aspect of EF, and the relative 'heat' of a task are largely determined by the type of stimulus and the behavioural context, moderated by individual differences in the perception of stimuli as motivationally salient (Welsh & Peterson, 2014). For instance, one aspect of hot EF that has been linked to delay of gratification has been the ability to use various cognitive reappraisal strategies to alter how the affective component of a stimulus is perceived or interpreted (e.g., envisioning an attractive marshmallow as a cloud or a cotton ball; Mischel, Shoda, & Rodriguez, 1989).

High sensitivity to rewarding stimuli in DCD and a focus on immediate reward may therefore reflect deficits of inhibitory/impulse control and a reduced ability to delay gratification; associated with this has been low self-control and impulsive behaviours (Riggs, Blair, & Greenberg, 2004). A constellation of cognitive and behavioural problems consistent with this profile appears in DCD: poor inhibitory control, impulsiveness, and a higher incidence of externalizing problems. This cluster could be explained, in part, by high sensitivity to immediate reward and poor affective decision-making associated with DCD.

One way to determine the contribution of inhibitory control to poor affective decision-making in DCD is to compare the ability of children with poor coordination to inhibit prepotent responses to neutral and emotionally significant stimuli. This will indicate whether high sensitivity to reward, and consequently deficits of hot EF, is a distinct impairment in DCD or whether it is due to poor inhibition and high impulsivity within this cohort.

4.3. Are deficits in hot EF in DCD due to impairments of learning and planning?

An alternative hypothesis is that performance of the DCD group on the HDT is due to deficits in cognitive planning (Asonitou, Koutsouki, & Charitou, 2010). Planning involves using feedback from earlier responses (learning) and anticipating the consequences of the same responses (forward thinking and modelling). Although both our groups showed impaired performance on the HDT, feedback utilization and response anticipation seemed to be disrupted more severely in children with DCD.

Throughout the task, the DCD group approached both of the disadvantageous, high immediate reward options (A & B) more often than the controls and showed a reduced ability to integrate feedback – i.e., high loss of apples – in their decisions. Moreover, they had slower responses than controls to only advantageous, low immediate reward options across all 5 blocks of the HDT. These performance patterns suggest impairment of the ability to learn from previous disadvantageous decisions and a deficit of response reversal – an ability closely associated with set-shifting. Indeed, another 'executive'

component of hot EF could be the ability to integrate the emotional representation of stimuli based on previous experience, which informs adaptive decisions about emotionally significant stimuli (Welsh & Peterson, 2014). Although DCD has been linked to some learning problems (see Zwicker et al., 2012, for a review), similar patterns of performance (i.e., inability to use and integrate feedback in decisions) has been observed among typical school-aged children (Cassotti, Houde, & Moutier, 2011; Cauffman et al., 2010; Prencipe et al., 2011) who often persist in using a particular choice strategy (e.g., 'rule 1') on either the IGT or the HDT, and fail to integrate feedback into their choices.

Developmental studies (Prencipe et al., 2011; Smith et al., 2012) have shown that with increasing age, but particularly from young adulthood, otherwise normal individuals are more likely to perform response reversal, in that they gradually learn to avoid disadvantageous decks on the IGT, and select more cards from the advantageous options. However, DCD and its associated learning problems persist into adulthood for a significant proportion of individuals diagnosed with this disorder at childhood (Zwicker et al., 2012). Therefore, if learning problems contribute to the poor performance of the DCD group on tests of hot EF, differences between DCD and control groups may become clearer with age. The assessment of hot EF in young adults with and without motor coordination problems may help us determine the exact role of learning difficulties in affective decision-making.

In addition to using feedback, anticipating the consequences of action (in real time) is another aspect of planning hypothesized to be disrupted in DCD (Williams, Thomas, Maruff, Butson, & Wilson, 2006). The Internal Modelling Deficit (IMD) hypothesis (Wilson et al., 2004) suggests that an impaired ability to internally represent actions and mentally simulate movement leads to problems with predicting the consequences of motor behaviour, organizing motor responses, and monitoring/modifying action. This leads to problems with learning and predicting motor behaviour. Indeed, Wilson et al. (2013) argued that predictive control of action is a core deficit in DCD. Although speculative, the impaired ability to generate and/or utilize a forward model of (motor) action could affect performance and decision-making in other domains as well. Thus, poor HDT performance of the DCD group might be due to poor ability in anticipating the outcome of selecting disadvantageous, high immediate reward options. In other words, the prediction deficit may be more generalized than just real-time motor control.

Forward modelling deficits in DCD diminish the ability to modify existing internal models and notably increase the time required to build an adequate model for action (Wilson et al., 2013). If a similar or related mechanism affects performance of children with DCD on a non-motor EF task such as the HDT, they may take significantly longer to reach advantageous levels of decision-making, relative to their peers. However, if high sensitivity to reward underlies their performance, they might not be able to easily change their decision-making strategy. Our study was limited to 100 trials in the HDT. The inclusion of more trials could determine the impact of forward thinking on hot EF deficits of the DCD group.

4.4. *Implications of hot EF deficits in DCD*

In most instances, decision-making depends on a combination of both cool and hot EF. DCD has been linked to cool EF deficits; however, deficits in hot EF and an impaired ability to exercise inhibitory control in motivationally relevant settings, may compromise the control of emotional responses and further disrupt decision-making and behaviour regulation. Generally, hot EF deficits have been linked to poor ability to anticipate future consequences of actions, deficits in impulse- and self-control, and consequently, poor decisions in situations with high emotional or motivational significance (Crone & van der Molen, 2004; Toplak, Jain, & Tannock, 2005; van Duijvenvoorde et al., 2010).

Hot EF in childhood is an important predictor of self-control, academic achievement, interpersonal skills, and frustration tolerance (Casey et al., 2011), highlighting the need for early interventions targeting problems in affective decision-making. Indeed, there is evidence that both cool and hot EF are malleable, particularly during 'sensitive periods' of preschool years and transition to adolescence when there is either a rapid growth or reorganization of prefrontal systems (Zelazo & Carlson, 2012). Thus, intervention programmes may ameliorate deficits of hot EF in children with DCD.

Our study is the first to show deficits of hot EF in children with DCD. Given the suggested plasticity of hot EF, if future studies corroborate that DCD is indeed linked to heightened sensitivity and lowered

resistance to emotionally significant stimuli, interventions should be designed to address deficits of hot EF in children with DCD. Such interventions could be highly effective in improving their functioning, facilitating their learning and social interactions, as well as reducing their problem behaviours, and consequently improving their quality of life.

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APPENDICES

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Appendix J Published Article in *Human Movement Science* – Study 2

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Deficits of hot executive function in developmental coordination disorder: Sensitivity to positive social cues

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Go/no-go

ABSTRACT

Recent research shows that children with motor coordination problems (or developmental coordination disorder – DCD) show deficits in not only cool executive function (EF), but also hot EF. We aimed to determine whether this deficit of hot EF is due to heightened sensitivity to rewarding stimuli, specifically, or to a general deficit of cognitive control, like inhibition. Using two versions of a go/no-go task, one with neutral facial expressions and the other with happy and fearful faces, we compared 12 children with DCD with 28 typically-developing children, aged 7–12 years. Like earlier studies, children responded faster to happy faces. Both groups showed comparable accuracy in response to go targets, and also had similar commission errors, except when the no-go stimulus was a happy face. Importantly, the DCD group made significantly more commission errors to happy faces failing to suppress their response on more than half of the no-go trials. These results suggest a heightened sensitivity to emotionally significant distractors in DCD; this type of impulsivity may undermine self-regulation in DCD, with possible implications for adaptive function and emotional well-being. We argue that the interaction of cognitive control and emotion processing networks may be disrupted in DCD or delayed in development.

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1. Introduction

Neurodevelopmental disruptions in one aspect of functioning (e.g., motor) can have far-reaching consequences beyond the primary domain (Leonard & Hill, 2014). In the particular case of poor motor coordination in children (or developmental coordination disorder—DCD), aspects of psychosocial functioning, cognition, and academic performance can also be disrupted (Zwicker, Harris, & Klassen, 2012). In this paper we explore issues of cognition in DCD using an experimental approach, specifically the nature of *executive function* (EF) in these children.

Under DSM-V, DCD is conceptualized as a neurodevelopmental disorder that is marked by motor coordination problems that negatively affect one's daily living activities and/or academic achievement (Zwicker, Missiuna, Harris, & Boyd, 2012), and is generally diagnosed in 5–6% of school-aged children (APA, 2013). The disorder is a distinct diagnostic entity, but often co-occurs with other conditions like Attention Deficit/Hyperactivity Disorder (ADHD) and Specific Language Impairment (SLI) (APA, 2013; DSM-V). Importantly, DCD has been linked to underlying difficulties in not only motor control (Wilson, Ruddock, Smits-Engelsman, Polatajko, & Blank, 2013), but also psycho-social adjustment (e.g., poor self-worth, self-esteem, feelings of loneliness, depression and anxiety, as well as externalizing problems) and cognitive control (Cairney, Rigoli, & Piek, 2013; Cummins, Piek, & Dyck, 2005; Schmahmann & Caplan, 2006; Skinner & Piek, 2001; Zwicker, Missiuna, Harris, & Boyd, 2012). More specifically, the recent review of Wilson et al. (2013) shows a quite pervasive pattern of dysfunction across (predictive) motor control, all major aspects of EF (i.e., inhibition, working memory (WM) and executive attention—Diamond, 2013), and the self-regulation of movement (e.g., Sangster Jokic & Whitebread, 2011). What remains unclear is the role of affect in the expression of these deficits, or indeed, whether certain types of problems exist only when the child's emotional investment in the task is heightened.

1.1. Cool and hot EF

EF is an umbrella term that refers to a set of neurocognitive processes involved in conscious and effortful control of thought, emotion, and behavior. Broadly, it can be divided into cool and hot EF. Cool EF is mainly subserved by lateral prefrontal cortex (L-PFC), enlisted when one deals with abstract and decontextualized stimuli. In contrast, hot EF is linked to ventromedial prefrontal cortex (VM-PFC), active in many real-life situations that are characterized by high affective involvement; here, one needs to consider or reappraise the emotional/motivational significance of stimuli and refrain from impulsive actions (Zelazo & Muller, 2011).

EF has been traditionally assessed using 'cool' tasks (e.g., WM, inhibition, and set-shifting), which include decontextualized stimuli (Miyake et al., 2000; Zelazo & Carlson, 2012). There is strong evidence of cool EF deficits in DCD. The recent meta-analysis by Wilson et al. (2013) showed very large effect sizes ($d > 1$) on tasks that assess WM, inhibitory control, and executive attention. The stimuli in cool EF tasks, however, often bear little resemblance to everyday situations where one interacts with emotionally and motivationally meaningful stimuli. By comparison, measures of hot EF aim to mimic aspects of real-life decision-making through use of reward and losses, as in delay of gratification and gambling tasks (e.g., Iowa Gambling Task (IGT); Bechara, Damasio, Damasio, & Anderson, 1994).

The studies that compared the performance of typically-developing (TD) children and adolescents on hot and cool EF tasks report that cool EF may mature earlier since adult-like levels of performance are reached later for hot EF. This fits with the view that VM-PFC or its connections might follow a protracted trajectory of development relative to more dorsal aspects of PFC (Hooper, Luciana, Conklin, & Yarger, 2004; Prencipe et al., 2011). However, it has also been suggested that regions associated with hot EF (i.e., orbitofrontal cortex) may develop earlier than those recruited in 'cool' tasks of EF (e.g., DL-PFC) (Orzhekhovskaya, 1981). The fact is that the neurocognitive networks involved in hot and cool EF overlap and form part of a larger interactive functional system. As such, it remains challenging to design 'pure' measures of each of the major two domain of EF (Hongwanishkul, Happaney, Lee, & Zelazo, 2005). Deficits of hot EF, for instance, have been linked to inadequate response inhibition which results in reduced modulation of what is otherwise a relatively mature affective system

(Bechara & van der Linden, 2005). Therefore, disruptions in the development of 'cool' regions may also impact deficits of hot EF. Data on children with ADHD – a disorder with high comorbidity for DCD – support this argument: while considered mainly as a disorder of cool EF (Zelazo & Muller, 2011), a number of studies also report deficits of hot EF, particularly the hyperactive subtype (Castellanos, Sonuga-Barke, Milham, & Tannock, 2006; Dinn, Robbins, & Harris, 2001; Toplak, Jain, & Tannock, 2005).

Both aspects of EF are vital determinants of behavior and adjustment over the lifespan. They predict important developmental outcomes and underlie age-appropriate cognitive and social functioning (Prencipe et al., 2011). EF deficits not only contribute to poor mental and physical health (e.g., higher rates of obesity, overeating, substance abuse) but are linked to reduced academic success and problems in other aspects of adaptive function (e.g., finding and keeping a job, marital satisfaction, public safety, and general quality of life) (Diamond, 2013). While deficits of hot EF have been linked to internalising and externalizing problems and poor academic achievement (Casey et al., 2011), little evidence exists about hot EF in DCD.

1.2. Preliminary evidence for hot EF deficits in DCD

In an earlier study (Rahimi-Golkhandan, Piek, Steenbergen, & Wilson, 2014), we investigated hot EF in children with DCD using the Hungry Donkey Task (HDT; Crone & van der Molen, 2004). The HDT is an age appropriate variant of the IGT in which children are required to win as many apples as possible for a donkey. Children need to maximize their win by choosing among four options presented to them over 100 trials; they are expected to use feedback from earlier trials to identify the most profitable option(s). In each trial, children can select only one of the options. Each of these options is characterized by three dimensions: amount of win, amount of loss, and frequency of loss. Two options that are disadvantageous in the long run (A and B) are characterized by higher immediate reward (4 apples per trial) but also high loss overall. The other two (C and D) are associated with lower immediate rewards (2 apples) on each trial but are advantageous in the long run because of their low overall loss. In every 10 trials, frequency of loss is high for A (five unpredictable losses of 8, 10, 10, 10, and 12 apples) and C (five unpredictable losses of 1, 2, 2, 2, and 3 apples), and low for B (one unpredictable loss of 50 apples) and D (one unpredictable loss of 10 apples); leading to equal net gain after every 10 trials: Loss of 10 apples for the disadvantageous options, and win of 10 for the advantageous ones.

TD children up to age 12 often fail to integrate feedback in their decision-making and, rather than opting for the advantageous options (C and D), simply prefer to avoid frequent loss, selecting options B and D at a significantly higher level than chance (Huizinga, Crone, & Jansen, 2007; van Duijvenvoorde, Jansen, Visser, & Huizinga, 2010). The TD children (control group) in our study showed the same pattern of performance; however, children with DCD showed a preference for option B, characterized by infrequent loss but also high immediate reward. As well, the DCD group selected option D at chance level (i.e., 100/4) and had a significantly lower total net score than the controls. In terms of response speed, these children had significantly faster responses to the disadvantageous (high reward) options, while responses of the control group were similar for both types of options. Finally, the DCD group was slower than the controls when they approached advantageous (low reward) options, only. Taken together, this pattern of performance by the DCD group, which persisted over 100 trials, suggests heightened sensitivity to rewarding, emotionally significant stimuli (Toplak et al., 2005), which underlies poor affective decision-making. However, other hypotheses could explain the performance of children with DCD on the HDT, notably impaired use of somatic markers and poor inhibitory control.

1.3. Predictors of poor performance on the HDT

1.3.1. Somatic markers

The IGT and the HDT attempt to mimic real-life decision-making in which the logical cost–benefit analysis of available response options is difficult (Bechara et al., 1994). The somatic marker hypothesis (SMH; Damasio, 1994) suggests that under complex task conditions, individuals rely on more intuitive decision processes; in order to solve the problem, use is made of emotion-based biasing signals and sensations (i.e., *somatic markers*) that arise from the body (e.g., viscera, skeletal and smooth muscles)

and/or the central representation of the body (Damasio, 2004). These markers are integrated in the emotion circuitry of the brain and signal the likely outcome of each single action (i.e., reward or punishment) and its associated emotional outcomes. The brain then uses these signals to create a *forward model* of the changes expected to happen in the body, which enables one to respond rapidly to the stimuli (Dunn, Dalgleish, & Lawrence, 2006). For instance, somatic markers indicate whether an action is going to be rewarding or punishing, and assist decision-making before the activity emerges in the periphery. Indeed, dysfunction of the brain regions involved in the representation and regulation of the body-state (e.g., basal ganglia, insula, somatosensory cortices) impairs the ability to use somatic markers and leads to poor affective decision-making (Damasio, 1998). Intriguingly, DCD has been linked to deficits in the predictive control of action (Wilson et al., 2013) as well as dysfunction of the basal ganglia and insula in possible sub-groups of DCD (Lundy-Ekman, Ivry, Keele, & Woollacott, 1991; Zwicker, Missiuna, Harris, & Boyd, 2011). In short, it is possible that hot EF deficits in DCD are at least partly due to an impaired ability to effectively use somatic markers.

Another important issue is that performance on many EF tasks is likely to be supported by a combination of cool and hot EF (Hongwanishkul et al., 2005). For instance, individuals need to keep track of wins and losses associated with each option on the IGT or the HDT, with some studies showing significant covariation between performance on these tasks and WM (e.g., Hinson, Jameson, & Whitney, 2002), with others not (e.g., Crone & van der Molen, 2007; Rahimi-Golkhandan et al., 2014).

1.3.2. Inhibition

Inhibitory control is thought to not only affect other cool aspects of EF but also play a crucial role in hot EF (e.g., the ability to resist temptation and delay gratification) (Diamond, 2013; van Duijvenvoorde, Jansen, Bredman, & Huizinga, 2012). For instance, successful performance on the IGT relies not only on the activation of emotion circuitry, but also other regions known to be active during inhibitory control tasks like anterior cingulate (Braver, Barch, Gray, Molfese, & Snyder, 2001), cerebellum, insula, and inferior parietal cortex (Ernst et al., 2002). Additionally, children need to perform a response reversal on the HDT: optimally, they should avoid options that are immediately appealing, use this feedback, and instead approach options that benefit them in the long run (Dunn et al., 2006). Difficulties in reversal learning and inhibiting such a prepotent response inevitably lead to low net scores on the HDT (e.g., Crone, Vendel, & van der Molen, 2003). It is possible, therefore, that poor affective decision-making could be due to a combination of an overactive emotional system and underdeveloped inhibitory control processes (Bechara & van der Linden, 2005), which results in an 'emotional overshoot' (van Duijvenvoorde et al., 2010) whereby impulsive reward-driven performance is not paralleled by effective control systems (Smith, Xiao, & Bechara, 2012).

DCD has been linked repeatedly to poor response inhibition in different settings using a variety of tasks (see Piek et al., 2004; Wilson et al., 2013). Poor inhibitory control and high incidence of externalizing problems in children with DCD suggest an overall degree of impulsivity within this cohort that could be linked to heightened sensitivity to immediate rewards and poor affective decision-making. To further dissect this hypothesis, it is important to test whether the performance on the HDT is a reflection of a distinct deficit in regulating responses to emotionally salient stimuli or is more attributable to a problem of inhibitory control. To this end, we compared the performance of DCD and non-DCD groups when required to inhibit prepotent responses to either neutral or emotionally significant stimuli on two versions of a go/no-go task.

1.4. Cool and hot go/no-go tasks

To tease out the contribution of inhibitory control per se, we compared two versions of the go/no-go task: the 'cool' task used neutral facial expressions of men and women, while the 'hot' version presented happy and fearful expressions of the same individuals (Casey et al., 2011). Given the attraction of a happy facial expression, people tend to associate it with positive affect and reward. Indeed, happy facial expressions activate the same brain areas (e.g., ventral striatum) involved in the processing of rewards, and like other rewarding stimuli are approached instinctively, and can provoke impulsive behavior (Hare, Tottenham, Davidson, Glover, & Casey, 2005). For this reason, it is more difficult to avoid happy faces because one must resist the natural tendency to approach a rewarding stimulus

(Hare et al., 2005). The ability to withhold responses to a happy face reflects one's level of self-control and sensitivity to alluring cues, and has been linked to the ability to delay gratification (Casey et al., 2011). No such compulsion is involved when responding to neutral or negative facial expressions.

1.5. Aim and hypothesis

The broad aim of this study was to examine whether heightened sensitivity to rewarding stimuli is a distinct deficit in DCD, associated with hot EF deficits in this cohort. More specifically, we sought to determine whether apparent deficit of hot EF is explained selectively by a heightened sensitivity to rewarding stimuli, or is due to a general deficit of inhibitory control. Results have implications for understanding other aspects of cognitive, motor and emotional functioning in DCD. We predicted that children with DCD would show significantly higher sensitivity to positive stimuli on the go/no-go task, operationalized by commission errors to happy faces, than their TD peers.

2. Method

2.1. Participants

The sample included 12 (6 boys, 6 girls) children with DCD ($M(SD)_{age}$ = 9.31 (1.47)), and 28 (10 boys, 18 girls) TD children ($M(SD)_{age}$ = 9.72 (1.61)). The age range for both groups was between 7 and 12 years, and there was no significant difference between the average ages of the groups (p = .45). Children who had a Neurodevelopmental Index (NDI) score of 80 or less on the McCarron Assessment of Neuromuscular Development (MAND; McCarron, 1997) are considered *at risk* for DCD (Piek et al., 2004), and were allocated to the DCD group ($M(SD)_{NDI}$ = 77.82 (5.73)). Four children had an NDI of between 55 and 70 (indicating moderate motor difficulties (Piek et al., 2004)) while the rest had NDIs between 70 and 80. The control group included children with a NDI of 100 or above ($M(SD)_{NDI}$ = 109.29 (6.24)). The exclusion criteria was the diagnosis of other developmental disorders, such as ADHD, or any other neurological, learning, or physical disorder. We did not conduct any neurological or medical examination, which is used to ensure the criterion C of the DCD diagnosis in DSM-IV-TR is met (Geuze, Jongmans, Schoemaker, & Smits-Engelsman, 2001), mainly because children were all enrolled in three mainstream primary schools in Melbourne, and their intelligence levels were inferred to be within the normal range; no children were attending remedial classes for literacy or mathematics.

2.2. Materials

2.2.1. Go/no-go task

The two versions ('cool' and 'hot') of the go/no-go task used grayscale pictures of 12 individuals (6 males, 6 females) from the NimStim collection, downloaded from www.macbrain.org. The size and luminance were the same for all the images. The faces used were numbers 6, 8, 11, 14, 15, 16, 27, 28, 36, 39, 44, and 45. Neutral facial expressions were used for the cool task, while the stimuli for the hot task were happy and fearful faces of the same individuals. Each task consisted of two runs in which the go target for the first run was the no-go target in the second run. Therefore, both cool and hot tasks used a 2 (either male, female or happy, fearful) × 2 (go, no-go) factorial design. Before the start of each run, which presented 40 pictures in a pseudorandom order (28 go, 12 no-go), children received a short notification on the monitor informing them of which sex (for the cool task) or facial expression (for the hot task) served as the go target. Each picture was presented for 500 ms followed by a 1500 ms inter-stimulus interval, during which children saw a fixation cross, but were still able to respond. Children were instructed to respond as quickly and as accurately as possible, pressing the spacebar for the go trials, and refraining from responding to no-go targets. Accuracy (of responding to go stimuli, and withholding a response to no-go targets) and reaction time (RT) to go stimuli were the main indices of performance in each task.

APPENDICES

2.2.2. McCarron assessment of neuromuscular development (MAND)

The MAND (McCarron, 1997), which includes 10 short tests of fine and gross motor skills (5 each), provides a standardized index of motor skills for 3.5–18 year old individuals. The NDI score is calculated by comparing the sum of scaled scores (on the 10 tests) to age-appropriate norms for each child. The MAND is recognized as a valid and reliable assessment tool for motor impairment (Piek et al., 2004). It has a good test–retest reliability (.67–.98) over a 1-month period, acceptable criterion and concurrent validity (McCarron, 1997), and good specificity and sensitivity (Tan, Parker, & Larkin, 2001).

2.3. Procedure

After the approval of this experiment by the Human Research Ethics Committee of the Australian Catholic University, different primary schools across Melbourne, Australia, were contacted, and the parents and guardians of children in the three schools that expressed their interest in our project received plain language statements about the study. Informed consents were obtained from the parents and guardians before the start of the experiments. The testing was conducted in a quiet environment, and during children's class time. Half of the children first completed the go/no-go task, while the other half were first tested on the MAND. We also counterbalanced the two versions of the go/no-go task as well as the two different runs within each task. The go/no-go task was administered on a laptop computer running the E-Prime version 1.1 (Schneider, Eschman, & Zuccolotto, 2002), and the testing session of each child took approximately 20 min.

2.4. Data analysis

The most important index of performance, which provided a direct test of our hypothesis, was the commission error. This outcome variable measured the ability to withhold a response to no-go targets: low commission error represent better performance. Commission errors were submitted to a 2-way mixed-factorial ANOVA with group (DCD vs. control) the between-subjects factor, and the type of no-go stimulus (male, female, happy, and fearful) the repeated factor. The number of consecutive go trials prior to a no-go target has been linked to a higher rate of commission errors (Durston, Thomas, Worden, Yang, & Casey, 2002). The highest number of continuous go trials, in each run of the hot task in our study, was four. To assess the impact of task difficulty on performance, we compared the groups on the proportion of commission errors after four consecutive go trials. Omission errors were operationalized as the percentage of trials in which children did not approach go targets. Omission errors were also submitted to a 2-way mixed-factorial ANOVA: group (DCD, control) and type of go stimulus (male, female, happy, and fearful). RT to go targets was compared between groups, reflecting whether the emotional significance of stimuli had any impact on the latency of responses. Finally, to investigate the potential impact of emotion discrimination on the task performance, and to determine how successfully children were able to discriminate between various facial expressions, we used *d*-prime (*d'*) which provides an index of sensitivity to each stimulus in a go/no-go task. *d'*, which takes into account the respondent's bias, can be used as an index of emotion recognition (Tottenham, Hare, & Casey, 2011). The following formula was used to calculate this index: $[d' = z(\mathrm{H}) - z(\mathrm{F})]$. For each child, $z(\mathrm{H})$ is the standardized rate of approaching the relevant go target ('hits'), while $z(\mathrm{F})$ is the standardized score for commission errors or false alarms. The higher the *d'* value, the more capable is the child to discriminate go and no-go targets.

3. Results

3.1. Commission errors

Fig. 1 shows the proportion of commission errors on no-go trials for each group. As shown in this Figure, the groups had similar accuracies during all the no-go trials, except when the no-go target was a happy face. The average commission error was notably higher for the DCD group who failed to

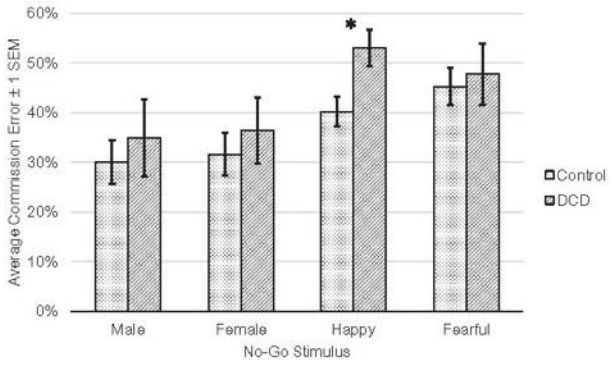

Fig. 1. Mean (±SE) commission errors to no-go targets in the DCD and control groups (*$p < .05$).

suppress their responses to happy faces on more than half of the trials. Interestingly, the 2 × 4 mixed-factorial analysis of variance (ANOVA) did not show a significant interaction between group and stimulus, Wilks' $\Lambda = .92$, $F(3,35) = 1.05$, $p = .38$, $\eta^2 = .10$. The low observed power for this particular analysis (.26) possibly accounts for the lack of a significant interaction. The analysis of simple main effects, however, provided a better reflection of group differences. There was no significant difference in commission errors on both versions of the cool task: female no-go: $p = .55$, $d = 0.21$ (95% CI [−0.49,0.91]); male no-go: $p = .57$, $d = 0.20$ (95% CI [−0.50,0.90]). In contrast, we found significant differences in commission errors on the hot task. While the groups showed no difference in commission errors in response to fearful no-go targets: $p = .73$, $d = 0.12$ (95% CI [−0.58,0.82]), the DCD group produced more commission errors when the no-go stimulus was a happy face, $p = .02$, $d = 0.86$ (95% CI [0.14,1.58]). The bootstrapped analysis (using 5000 samples, and 95% CI) produced a p value of .009 for the group differences in commission errors to happy faces. None of the other bootstrapped analyses for commission errors reached significant levels – smallest p was for female commissions (.53). Moreover, within-group analyses (of the hot task) showed that while controls had less error in response to the happy faces ($d = 0.27$), the DCD group were better able to withhold their responses to the fearful faces ($d = 0.29$).

There were three instances in each run of the 'hot' task where a no-go target was preceded by four go trials. Therefore, the maximum number of commission errors after these 'difficult' trials was 3. However, the error rate of each group to either happy or fearful non-targets was less than half of this [no-go stimulus: (a) fearful: $M\ (SE)_{DCD} = 1.27\ (0.36)$, $M\ (SE)_{Control} = 1.04\ (0.17)$; (b) happy: $M\ (SE)_{DCD} = 1.18\ (0.32)$, $M\ (SE)_{Control} = 1.11\ (0.19)$], with no significant differences between DCD and control groups ($p > .5$). We did not run the same analysis on the 'cool' task because its two runs did not have equal number of 'difficult' trials.

With respect to individual differences, Fig. 2 presents the number of commission errors to happy no-go targets by each child in the DCD and control groups. There were six children in the DCD group who made commission errors on more than half of the trials (7 or more errors out of 12 trials), 3 of which had NDIs less than 70; as well, the remaining child with moderate motor difficulties made five errors. The difference between the commission error rates of children with NDI <70 (58.3%) and the rest of the DCD group (50.0%) was about 0.7 of the *SD* of the total group average (53%).

3.2. Omission errors

Fig. 3 presents the average (±SE) percentage of omission errors in each group. Both DCD and control groups produced few errors, showing that each were able to correctly identify and approach the relevant go stimulus in each task. The 2 × 4 mixed-factorial ANOVA did not reveal any significant interaction between group (DCD, control) and the go stimulus (male, female, happy, and fearful), Wilks' $\Lambda = .90$, $F(3,35) = 1.25$, $p = .31$, $\eta^2 = .10$. Although the DCD group made more errors than controls in response to male faces ($p = .87$, $d = 0.06$ (95% CI [−0.64,0.75]), female ($p = .12$, $d = 0.57$ (95%

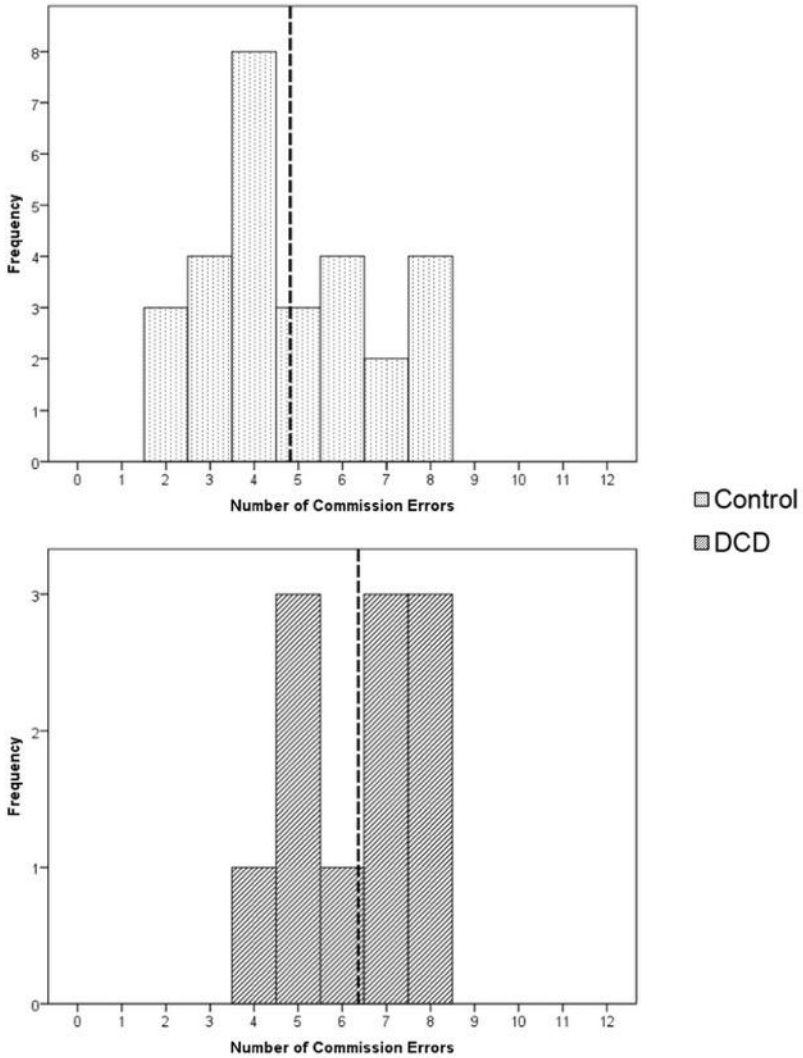

Fig. 2. Frequency distribution of commission errors to happy no-go targets. The dotted line represents group average. Maximum possible number of commission errors was 12 (trials).

CI [−0.14,1.28]), and happy ($p = .51$, $d = 0.24$ (95% CI [−0.46,0.94]), they performed better than controls in response to fearful targets ($p = .49$, $d = -0.25$ (95% CI [−0.95,0.45]). Within-group analyses showed no significant differences in the omission errors of the DCD group to different stimuli. However, the controls had significantly fewer omission errors in response to fearful faces (p for all pairwise comparison was less than .003).

3.3. Reaction time

Table 1 present the average (and SD) RT of each group to the four possible go stimuli. According to this table, children with DCD always had a slower response to different go targets. More importantly, the slowest RT for both groups was to the fearful faces. A 2 × 4 mixed-factorial ANOVA found no

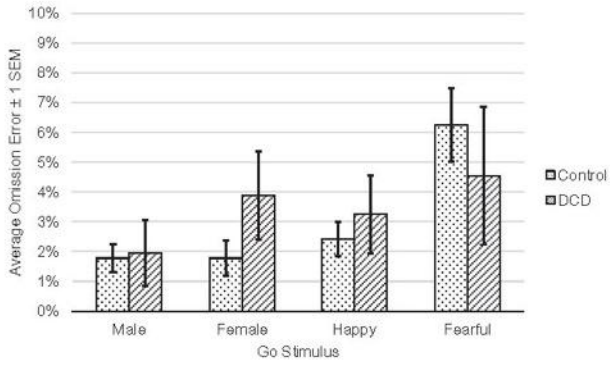

Fig. 3. Mean (±SE) omission errors to go targets in the DCD and control groups.

Table 1
Mean (SD) RT of DCD and control groups to different Go targets.

	Go stimulus			
	Male	Female	Happy	Fearful
DCD	528.53 (47.37)	543.14 (57.50)	545.16 (88.22)	567.85 (59.34)
Control	501.77 (64.15)	496.47 (75.01)	516.75 (80.43)	545.75 (95.37)

Note: RT is in milliseconds.

significant interaction between group and go stimulus, Wilks' $\Lambda = .94$, $F(3,35) = 0.69$, $p = .56$, $\eta^2 = .06$, suggesting that the pattern of RT was similar for both groups. Tests of simple main effects showed no significant difference between RTs to the go targets of the cool task (i.e., male and female faces) in each of the two groups ($p > .35$). On the hot task, however, both groups responded faster to the happy faces ($d_{control} = 0.32$, $p = .001$, $d_{DCD} = 0.28$, $p = .08$). Moreover, group differences in RT did not reach significance level for any of the go targets ($p > .07$).

We know that children with DCD are often significantly slower than their TD peers on most chronometric tasks (e.g., Piek & Skinner, 1999). However, their RT to different go stimuli was not significantly different to that of controls. This prompted us to correlate RT (to go targets) to commission errors in each group to investigate the possibility of a speed-accuracy trade-off in the DCD group (Table 2). Interestingly, however, it was the control group whose performance suggested a possibility of speed-accuracy trade-off in both runs of the 'hot' task. The correlations for the DCD group, on the other hand, were small and non-significant for both happy and fearful go targets. Even though this pattern of results is reversed on the 'cool' task, the DCD group did not have significantly more commission errors than the controls on this version of the go/no-go task.

3.4. d-Prime

Fig. 4 depicts d' for each of the go targets. The DCD group had a lower sensitivity to each of the face stimuli. However, the group (DCD, control) by stimulus (male, female, happy, fearful) interaction was not significant, Wilks' $\Lambda = .98$, $F(3,35) = 0.19$, $p = .90$, $\eta^2 = .02$. Tests of simple main effects also revealed no significant differences in d' between the two groups ($.32 < p < .58$).

Finally, it should be noted that different ratio of boys and girls in the two groups could have confounded our current findings. However, the inclusion of gender as a covariate in the analyses of outcome measures did not lead to any notable change in p values or effect sizes. For instance, the p value for the significantly higher commission errors of the DCD group in response to happy non-targets changed from .020 to .026, and the effect size remained the same.

Table 2
Pearson's *r* correlations between RT to Go targets and commission errors to no-go stimuli.

	No-go stimulus			
	Male[1]	Female[2]	Happy[3]	Fearful[4]
DCD	−.58^	−.71*	−.03	−.22
Control	−.26	−.16	−.33^	−.49*

Note: Go stimulus: 1 = female, 2 = male, 3 = fearful, 4 = happy.
* $p < .05$.
^ $p < .10$.

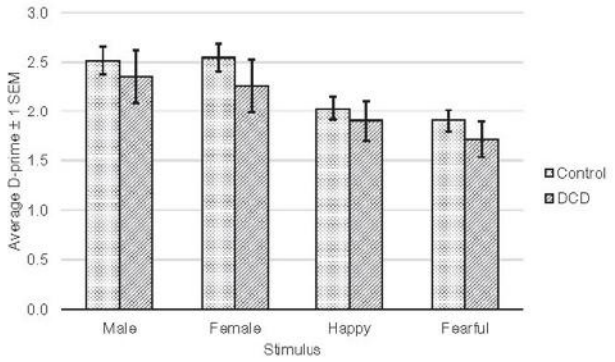

Fig. 4. Mean (±SE) *d'* for DCD and control groups in response to the go stimuli.

4. Discussion

The aim of this study was to investigate whether children with DCD show heightened sensitivity to emotionally significant stimuli. Our prediction was that the DCD group would have more difficulty than their TD peers in suppressing responses to positive social cues, namely happy non-targets in a go/no-go task. The findings supported this prediction, and suggest that poor affective decision-making in DCD on hot EF tasks can be explained in part by a high sensitivity to rewarding stimuli, and that this effect is not attributable to a generalized deficit of inhibitory control. In the discussion that follows, we explore factors underlying this pattern of performance in DCD and suggest interactions between emotion processing and action control centers. We also review the implications of these findings for understanding self- and emotion-regulation in children with motor coordination problems.

4.1. Why do children with DCD show greater sensitivity to rewarding stimuli?

Successful performance on go/no-go tasks that use emotionally salient stimuli requires a combination of emotion discrimination, cognitive control, and affect regulation (Tottenham et al., 2011).

4.1.1. Emotion discrimination

Deficits in recognizing and responding to static and changing facial expressions of emotion have been reported in DCD (Cairney et al., 2013; Cummins et al., 2005). However, poor emotion discrimination does not explain the pattern of performance we observed in DCD. Both DCD and control groups in our study responded faster to happy than fearful faces suggesting adequate facial discrimination and in-built approach behavior in both groups. Put simply, the emotional valence of the stimuli was apparent to and discriminative in both groups. This is similar to the previous reports of TD children, healthy adolescents and adults who generally show a bias for positive stimuli on emotional go/no-go tasks (Hare et al., 2005; Schulz et al., 2007; Tottenham et al., 2011; Urben, Van der Linden, &

Barisnikov, 2012). In contrast, affective disorders (e.g., major depressive disorder) have been linked to faster responses to negative stimuli (Murphy et al., 1999), while autism characterized by the absence of a preference to either stimuli (Duerden et al., 2013). There is still insufficient evidence about the performance of children with ADHD on the emotional go/no-go task, particularly in response to the positive stimuli.

Although all children responded with a high level of accuracy to go targets, and no group difference was evident, few omission errors do not necessarily mean good discrimination ability. To illustrate, selecting both go and no-go targets would result in high approach levels, but also high commission errors. The analyses of d', however, showed that DCD and control groups were equally sensitive to the go targets; indicating that differences in commission errors could not be due to poor emotion discrimination of children with DCD. Finally, our findings suggest that speed-accuracy trade-off, which was previously observed in the other developmental disorders such as autism (Yerys, Kenworthy, Jankowski, Strang, & Wallace, 2013), can also be ruled out as the contributing factor to the performance of the DCD group on the 'hot' go/no-go task.

4.1.2. Cognitive control

Apart from the need to recognize the emotional significance of stimuli, behavioral and neuroimaging data suggest emotion regulation relies on the functioning of control networks in the context of emotional information. To illustrate, an increased false alarm rate to emotional stimuli in TD children compared with otherwise normal adolescents and adults has been linked to the protracted development of cognitive control (Tottenham et al., 2011). In a similar vein, Yerys et al. (2013) reported that commission error rate was positively correlated with ADHD symptoms of hyperactivity and impulsivity among a group of children with autism, who made more impulsive responses to emotional stimuli than their TD peers. Neurophysiological evidence also highlights the impact of control processing centers in modulating sensitivity to appetitive cues. For example, adults who at age 4 performed poorly on the seminal Stanford marshmallow experiment (Mischel, Ebbesen, & Zeiss, 1972) had more difficulty avoiding happy non-target faces in a go/no-go task, and showed exaggerated recruitment of ventral striatum, and hypoactivity in the inferior frontal gyrus – indicating that resistance to temptation relies on frontostriatal regions involved in emotion regulation (Casey et al., 2011). Likewise, immature prefrontal activity has been linked to poor response inhibition in emotional contexts (Hare et al., 2008). Therefore, an alternative hypothesis is that a generalized deficit of inhibitory control may underlie heightened sensitivity of the DCD group in our study to positive stimuli.

However, this suggestion can also be ruled out as an alternative hypothesis. The higher incidence of commission errors in the DCD group was stimulus specific—it occurred when the no-go stimulus was a happy face but not for fearful or neutral faces. The absence of a significant group difference on commission errors has been previously reported in studies of DCD that used a similar go/no-go paradigm as ours (Piek et al., 2004; Querne et al., 2008). A pure deficit of cognitive control would manifest itself in a general increase in commission errors regardless of the type of stimulus. For example, children show more difficulty – than adolescents or adults – inhibiting responses to no-go targets, regardless of the emotional valence of the stimulus (Tottenham et al., 2011). Indeed, the performance of the DCD group is in line with the evidence that behavioral correlates of the ability to withhold prepotent responses are a function of not only inhibitory control, but also the salience of the stimulus to each person (Casey et al., 2011). One way to enhance the salience of stimuli, and increase false alarm rate to the targets one must resist is to manipulate task difficulty by increasing the number of consecutive go trials preceding a no-go trial. This method, which does not depend on the emotional valence of the stimulus, increases cognitive demands, and improves the chances of detecting differences in cognitive control (Durston et al., 2002; Eigsti et al., 2006). The finding that children with DCD performed comparably to controls on difficult trials of the 'hot' go/no-go task further suggests that stimulus specific deficit of response inhibition in the DCD group is probably not solely due to poor impulse and attention control.

4.1.3. The interaction of cognitive control and emotion processing networks

Alternatively, the inherent salience of a stimulus, which is independent of task design, can disrupt response inhibition by itself. To illustrate, appetitive cues are more difficult to avoid, and require

APPENDICES

greater impulse control because of the natural tendency to approach them. The ability to invoke greater cognitive control in emotional contexts (e.g., using reappraisal strategies to cool the appealing aspects of tempting stimuli) enhances delay of gratification (Mischel et al., 1972), and improves the chances of withholding a response to positive social stimuli (Casey et al., 2011). Therefore, a more likely interpretation is that emotional stimuli constitute a higher load on inhibitory control than neutral stimuli, and that children with DCD have more difficulty controlling their responses to the former. To illustrate this point, Lagattuta and colleagues examined inhibitory control using two versions of a Stroop-like card task: the neutral version used pictures of "day" and "night" as the competing stimuli, while the emotionally-laden version used happy and sad faces. It was shown that inhibition was more difficult for faces, with no ceiling effects, even for adults (Lagattuta, Sayfan, & Monsour, 2011). In DCD, heightened sensitivity to positive social cues may reflect a reduced level of coupling between emotion processing and cognitive control centers.

Differential rates of development in cognitive and affective systems can determine the drive or tendency to approach emotionally significant stimuli (Hare & Casey, 2005). For example, increased risk-taking and heightened sensitivity to appetitive cues in adolescence is linked to early maturation of brain regions (like the ventral striatum) that are involved in the representation of potential rewards compared with later developing systems that support controlled/planned behavior like frontostriatal networks (Somerville, Hare, & Casey, 2011). Immaturities in the development of cognitive control centers in children with DCD may reduce the ability to modulate the activation of emotion processing centers, resulting in more approach-oriented behavior toward positive stimuli. Children with DCD did not differ to TD children when inhibiting responses to less emotionally-rewarding stimuli (like neutral or fearful faces); higher commission errors were only evident when required to enlist inhibitory control in response to positive social cues which are known to increase the activation of emotion processing networks (Hare et al., 2005). Maturation of EF networks and their reciprocal connections with limbic and frontal motivation systems unfolds gradually over childhood and adolescence (Dennis, Malone, & Chen, 2009). Some delay in maturation could disrupt affect-regulation in children with DCD, with implications for self-regulation.

There is only scant evidence in DCD regarding the function of brain regions that support the ability to suppress responses to compelling (positive) stimuli or inhibition of impulsive behavior more generally. Activation of frontostriatal regions (Somerville et al., 2011), caudate nucleus (Hare et al., 2005), and parts of basal ganglia such as subthalamic nucleus (Frank, 2006) are known to subserve inhibitory function, particularly in the face of emotionally salient stimuli. There had been a handful of studies on the neural correlates of DCD (see Kashiwagi & Tamai, 2013 for a review). There is some debate about the involvement of basal ganglia in DCD (Groenewegen, 2003; Wilson, Maruff, & Lum, 2003), and it is possible that dysfunction within this structure and/or frontostriatal regions contribute to a heightened sensitivity of children with DCD to rewarding stimuli.

It is also noteworthy that the performance of the children with moderate motor difficulties in the DCD group raises the possibility of a *dose–response relationship* (see Cairney & Veldhuizen, 2013) between the level of motor functioning and performance on the go/no-go task; however, numbers were small for the DCD group in our study. Moreover, the analysis of commission errors at the individual level shows that some children in the DCD group perform equally to or even better than their TD peers. This finding supports the view that DCD is indeed a heterogeneous developmental disorder (Green, Chambers, & Sugden, 2008).

4.2. Self-regulation

Exerting cognitive control in the face of salient, appetitive cues can be challenging for children and, in the case of DCD, may pose particular difficulty; for instance, affecting the ability to suppress competing thoughts and actions, and undermining self-regulation. Poor self-regulation has been likened to control systems being 'hijacked' by the primitive limbic system, disrupting the neural modulation of behavior (Somerville et al., 2011). At a functional level, poor self-regulation can lead to the disturbances of goal-directed action, concentration, and academic achievement.

Self-regulation, however, is not a well-articulated construct. Various definitions of self-regulation have been proposed and with them a variety of tests to assess the broad construct (see Hoyle &

Davisson, 2011 for a review). One important point of consensus is that different aspects of self-regulation (i.e., the ability to plan, monitor, and modify attention, feelings, and behavior) are significant predictors of success in everyday life. For example, high impulsivity, and an impaired ability to control thoughts and actions during childhood have been linked to poor academic achievement (e.g., leaving school), health problems (e.g., smoking, taking drugs), and risky decision making during adolescence, as well as poor physical and mental health and lower quality of life in adulthood, even after controlling for IQ, gender, and socioeconomic status (Moffitt et al., 2011).

Diagnostic criteria for DCD suggest that the motor disturbance impacts academic achievement, participation, and everyday skills. Our data accord with the view that selective deficits of inhibitory control might be a common underlying issue explaining both poor self-regulation and motor control in DCD, with the functional outcomes being reduced success at school and psychosocial issues.

The ability to successfully regulate one's thoughts and feelings in the service of goal-directed action relies on a combination of both cool and hot EF. For example, cool EF is a significant predictor of school readiness and classroom functioning, while hot EF has been implicated in the development of early literacy and math skills (Wyatt, 2013). Our data and earlier studies of cool EF in DCD suggest that it is prudent to consider how interventions might be modified to accommodate the reduced capacity of children with DCD on EF, which we know has implications for self-regulation. Indeed, interventions that target EF have been shown to enhance psychosocial and physical functioning in children (see Diamond & Lee, 2011). At present, our direct knowledge of self-regulation in children with DCD is limited to the motor domain (Sangster Jokic & Whitebread, 2011). There is a dire need for research in the broader domain of self-regulation.

4.3. Emotion-regulation

Emotion regulation, which can be viewed as a subset of self-regulation, involves monitoring, evaluating, and modifying the intensity and temporal dynamics of emotional responses (Thompson, 1994). Underlying it is the ability to suppress responses to distracting stimuli and control attention in emotionally demanding contexts (Dennis et al., 2009)—modifying responses to both the pleasurable and the aversive (Tottenham et al., 2011). For instance, in a hot go/no-go task, the average false alarm rate to both emotional no-go stimuli is considered as the index of emotion regulation. It has been shown that children's ability to shift attention from an appetitive cue in a delay of gratification task predicts the ability to resist temptation on the task (Cole, 1986) and later ability to manage negative emotions during adolescence (Shoda, Mischel, & Peake, 1990). Our study showed that children with DCD might have higher sensitivity to positive social cues, and lower resistance to temptation than their TD peers. Whether this has direct implications for coping and psychological adjustment in DCD remains to be investigated.

In contrast, the DCD group approached negative stimuli (i.e., fearful faces) on go trials at a comparable rate and speed to controls. The absence of significant group differences here could reflect the balance between positive and negatively-valenced stimuli task protocol. The presence of happy no-go faces (positive, rewarding stimuli) on 30% of the trials could have created a prepotent tendency for the children to respond. This suggests that on go trials, if the negative stimulus was paired with a neutral stimulus, we could have seen lower response rates. Moreover, commission errors in response to fearful no-go targets could be partly due to a build-up of the tendency to approach happy faces, and not the tendency to approach negative stimuli per se. This limitation is particularly relevant to the performance of the DCD group who showed a heightened sensitivity to positive stimuli. Therefore, it is recommended that follow-up studies pair emotional targets (e.g., happy, fearful, or sad faces) with neutral facial expressions in order to obtain a more accurate measure of emotion regulation.

Results from the go/no-go task that bear on approach behavior to negatively valenced stimuli stand in contrast to the effects we would expect to see in the real world. Generally, a reduced ability to approach negatively valenced stimuli may have important repercussions for the efficacy of action, and psychological well-being of individuals. To illustrate, children with DCD often refrain from partaking in physical/sport activities mainly due to their fear of being criticized, ridiculed, and bullied by their peers. This fear of repeated failure not only reduces the desire to practice skills (Cairney et al., 2013), but also makes participation in skilled motor activities an unpleasant experience (Cairney &

Veldhuizen, 2013). The prior experience of physical activity as aversive can engender negative attitudes and feelings about participation per se; that is, the negative emotion/attitude can further reduce the tendency to approach physical play, and create the self-belief that physical competence and self-efficacy is limited. An unfortunate outcome, therefore, is anxiety and social isolation. In sum, this avoidance of 'negative' stimuli creates a vicious cycle (Skinner & Piek, 2001) which can lead to more physical and psychological problems.

4.4. Conclusion

In sum, we established, using a go/no-go task, that a small group of children with DCD showed higher sensitivity and lower resistance to positive social cues than their TD peers; no abnormality in response was seen for both neutral and negative cues. The main implication of this pattern of results is that children with DCD might experience particular difficulty enlisting inhibitory control when presented with emotionally and motivationally salient stimuli, particularly for strong appetitive cues. The ability to couple emotion processing and cognitive control networks in the service of action may be compromised. We recommend use of neuroimaging techniques, using larger samples of children with DCD, to examine more closely the action of neural systems that support emotion regulation and the coordination of goal-directed action. High sensitivity to appetitive, rewarding cues in DCD and reduced ability to enlist inhibitory control in response to them, may impair both the coordination of motor skill and self-regulation. The deficits of hot EF that we have identified may involve a constellation of processes that impact motor control, coordination, and functional behavior.

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APPENDICES

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Appendix K Article under review in *Brain and Cognition* – Study 3

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Title: Revealing Hot Executive Function in Children with Motor Coordination Problems: What's the Go?

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Keywords: Developmental Coordination Disorder; Motor development; Executive function; Cognitive control; Go/no-go; Inhibitory control

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Abstract: Recent research suggests that children with Developmental Coordination Disorder (DCD) often show deficits in executive functioning (EF) and, more specifically, the ability to use inhibitory control in 'hot', emotionally rewarding contexts. This study optimized the assessment of sensitivity of children with DCD to emotionally significant stimuli by using easily discriminable emotional expressions in a go/no-go task. Thirty-six children (12 with DCD), aged 7-12 years, completed two go/no-go tasks in which neutral facial expressions were paired with either happy or sad ones. Each expression was used as both, a go and no-go target in different runs of the task. There were no group differences in omission errors; however, the DCD group made significantly more commission errors to happy no-go faces. The particular pattern of performance in DCD confirms earlier reports of (hot) EF deficits. Specifically, a problem of inhibitory control appears to underlie the atypical pattern of performance seen in DCD on both cold and hot EF tasks. Disrupted coupling between cognitive control and emotion processing networks, such as fronto-parietal and fronto-striatal networks, may contribute to reduced inhibitory control in DCD. The implications for a broader theoretical account of DCD are discussed, as are implications for intervention.

Revealing hot EF in DCD - Cover Letter

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Editor-in-Chief
Brain and Cognition
Editorial Office

December 3, 2015

Dear Prof. Cohen,

Re manuscript submitted for publication: *Revealing Hot Executive Function in Children with Motor Coordination Problems: What's the Go?*

Please find here our manuscript, submitted for publication in Brain and Cognition.

This paper reports on deficits of executive function (EF) in children with Developmental Coordination Disorder (DCD) that extend to both cold and hot EF. In the current study, we optimised the assessment of sensitivity of children with DCD to emotionally significant stimuli by using easily discriminable emotional expressions in a go/no-go task. The findings show that reduced inhibitory control of children with DCD on the emotional go/no-go task is stimulus-specific. More specifically, the DCD group made significantly more commission errors than their typically-developing peers to happy no-go faces, only. A problem of inhibitory control appears to underlie the atypical pattern of performance seen in DCD on both cold and hot EF tasks. We highlight the potential neural underpinnings of reduced EF in children with DCD, and discuss the implications of hot EF deficits for motor learning and control in this cohort. We conclude that interventions that address both motor skills and the co-development of EF are more likely to facilitate skill learning and cognitive control in DCD.

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The submitted manuscript, or parts of it, have not been and will not be submitted elsewhere for publication. We would like to thank you for considering our paper, and look forward to your reply in due course.

Kind Regards,
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Revealing hot EF in DCD - Highlights

REVEALING HOT EF IN DCD

Revealing Hot Executive Function in Children with Motor Coordination Problems: What's the Go?

Highlights:

- Optimized the assessment of sensitivity to emotionally significant stimuli in DCD
- Comparable ability to approach negative stimuli by both DCD and control groups
- Heightened sensitivity to rewarding stimuli in children with DCD
- Reduced inhibitory control appears to underlie deficits of cold and hot EF in DCD
- Neural underpinnings of reduced EF in children with DCD are discussed

Revealing hot EF in DCD - Manuscript
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Running head: REVEALING HOT EXECUTIVE FUNCTION IN DCD

Revealing Hot Executive Function in Children with Motor Coordination Problems: What's the Go?

Abstract

Recent research suggests that children with Developmental Coordination Disorder (DCD) often show deficits in executive functioning (EF) and, more specifically, the ability to use inhibitory control in 'hot', emotionally rewarding contexts. This study optimized the assessment of sensitivity of children with DCD to emotionally significant stimuli by using easily discriminable emotional expressions in a go/no-go task. Thirty-six children (12 with DCD), aged 7-12 years, completed two go/no-go tasks in which neutral facial expressions were paired with either happy or sad ones. Each expression was used as both, a go and no-go target in different runs of the task. There were no group differences in omission errors; however, the DCD group made significantly more commission errors to happy no-go faces. The particular pattern of performance in DCD confirms earlier reports of (hot) EF deficits. Specifically, a problem of inhibitory control appears to underlie the atypical pattern of performance seen in DCD on both cold and hot EF tasks. Disrupted coupling between cognitive control and emotion processing networks, such as fronto-parietal and fronto-striatal networks, may contribute to reduced inhibitory control in DCD. The implications for a broader theoretical account of DCD are discussed, as are implications for intervention.

Keywords: Developmental Coordination Disorder; Motor development; Executive function; Cognitive control; Go/no-go; Inhibitory control

Revealing Hot Executive Function in Children with Motor Coordination Problems: What's the Go?

1. Introduction

Recent experimental work has raised several viable hypotheses about the neurocognitive underpinnings of atypical motor development (or Developmental Coordination Disorder—DCD). In a recent meta-analysis of the literature (Wilson, Ruddock, Smits-Engelsman, Polatajko, & Blank, 2013), the pattern of motor and cognitive performance in children with and without DCD was compared across 129 studies between 1998 and 2011. In addition to a broad cluster of motor control and learning issues, what was striking about this work was the consistent pattern of executive dysfunction (Cohen's d >1) across inhibitory control, working memory (WM) and executive attention tasks. Executive function (EF) refers to a group of neurocognitive processes involved in conscious and effortful control of thought, emotion, and behavior. More specifically, EF encompasses WM, executive attention, mental flexibility, and inhibition (Diamond, 2013). In recent studies, we have shown executive dysfunction in DCD extends to tasks that require so called 'hot EF'. These tasks have superimposed the requirement that emotional cues (positive and negative) be processed in order to achieve a task goal—hence the term *hot EF*. The issue of hot EF in children with DCD is particularly salient given other work showing that these children have difficulty with self-regulation (Sangster Jokic & Whitebread, 2011) and a higher incidence of anxiety associated with their motor problems (Zwicker, Harris, & Klassen, 2012). In the study reported here, we investigated hot EF using a go/no-go paradigm that used facial stimuli that were readily discriminable by children. Critically, we tested the specificity of the putative deficit in DCD that relates to the ability to inhibit responses to salient no-go stimuli.

DCD occurs in about 5-6% of children, and is characterized by problems in learning fine and/or gross motor skills, with resultant disruptions in daily living activities and/or

academic achievement (DSM-5; APA, 2013). Also associated with the disorder are a range of psychosocial issues including poor self-esteem, low self-efficacy (particularly for physical tasks), and impaired social relations. A bidirectional relationship between motor and cognitive development in DCD is supported by data showing concurrent deficits in each domain. EF deficits in DCD have led some to postulate that DCD is more than a purely motor disorder, but rather a broader neurodevelopmental syndrome (Wilson et al., 2013), one also associated with poor social-emotional adjustment (Zwicker et al., 2012). As a result, efforts to understand the underlying basis of DCD have centred on examination of motor control and EF.

While the classification of EF varies from one theorist to another (see Welsh & Peterson, 2014), two points have been brought into clear focus by recent work: first, the emotional valence of stimuli is critical in determining what nodes in a neural network are enlisted when performing an EF task, and second, component processes like *inhibition* are shared between 'cold' and 'hot' EF. Cold EF, subserved by dorsolateral prefrontal cortex (DL-PFC), is enlisted when one interacts with abstract, decontexualized stimuli, such as in the traditional lab-based tests of EF used to assess working memory, mental flexibility, and inhibition (Zelazo & Carlson, 2012). Hot EF, associated with ventromedial prefrontal cortex (VM-PFC), is more relevant to everyday decision-making, and incorporates the ability to reappraise the emotional-motivational significance of stimuli in order to voluntarilty inhibit or activate a particular behavior. The hot EF tasks, such as the delay of gratification and gambling tasks (e.g., Iowa Gambling Task (IGT); Bechara, Damasio, Damasio, & Anderson, 1994), mimic aspects of real-life decision-making through the use of rewards and losses. We recently showed that in the case of DCD, atypical patterns of performance were evident not only for traditional (cold) EF tasks but also so-called hot EF. Intriguingly, there may be a reduced ability in DCD to resist stimuli that offer high immediate reward, but longer term

loss (Rahimi-Golkhandan, Piek, Steenbergen, & Wilson, 2014). Impaired inhibitory control may contribute to this.

Response inhibition is believed to be an important determinant of not only cold EFs (e.g., WM and set-shifting), but also the ability to resist temptation (Aron, Robbins, & Poldrack, 2004; Diamond, 2013; van Duijvenvoorde, Jansen, Bredman, & Huizenga, 2012). For instance, some regions (e.g., anterior cingulate) that are active during inhibitory control tasks (e.g., go/no-go; Braver, Barch, Gray, Molfese, & Snyder, 2001) also predict optimal performance (i.e., higher net scores) on the IGT and its variants which tap into both the reward and inhibitory circuitry of the brain (Ernst et al., 2002; Smith, Xiao, & Bechara, 2012). Poor response inhibition prevents the contemplation and implementation of other response options, and eventually leads to low self-control and impulsive behaviors in children (Riggs, Blair, & Greenberg, 2004).

Recent fMRI data suggest that hypo-activity of DL-PFC in children with DCD may explain their reduced ability to switch (i.e., mentally shift) between go and no-go motor responses (Querne et al., 2008). Moreover, given the extensive connections between VM-PFC and the emotion circuitry of the brain, disruption of prefrontal regulation could underlie reduced emotional regulation that has been observed in DCD (Deng et al., 2014). Although cognitive inhibition – integral to interference control and selective attention – has been tested extensively in DCD, there is a dire need to investigate behavioral inhibition (i.e., self-control, resistance to temptation) (Diamond, 2013) in this cohort. Understanding the mutually interactive relationship between motor, cognitive and affective processes is a critical issue in both typical and atypical child development (Zelazo & Müller, 2011) and, in the case of DCD, holds significant implications for the design of interventions that target motor and/or behavioral issues, e.g. how training tasks can accommodate EF deficits in children.

1.1 Deficits of Hot EF in DCD

In an earlier study (Rahimi-Golkhandan, Piek et al., 2014), we investigated EF in DCD using a child-friendly variant of the IGT, called the Hungry Donkey Task (HDT; Crone & van der Molen, 2004). The optimal performance on the HDT relies on ignoring options that are initially rewarding but lead to an overall loss, and instead choose those associated with lower immediate reward (see Crone & van der Molen, 2004 for a description of the HDT). Children with DCD had a significantly lower total net score than typically developing (TD) children, and opted for the disadvantageous (high immediate reward) options. Moreover, even though the reaction time (RT) of the TD group did not depend on the type of option, the DCD group responded significantly faster to the disadvantageous options. One of the possible reasons for this pattern of performance in the DCD group is a deficit of inhibitory control (Rahimi-Golkhandan, Steenbergen, Piek, & Wilson, 2014).

The follow-up study (Rahimi-Golkhandan, Steenbergen et al., 2014), with the same groups of children, used an emotional go/no-go task to investigate specifically the role of inhibition in the heightened sensitivity of the DCD cohort to rewarding stimuli. Children completed both 'cold' (neutral facial expressions) and 'hot' (happy and fearful faces) versions of the task. There were no significant group differences in omission errors, a measure reflecting attention (Schulz et al., 2007). As well, analysis of d' – a measure of perceptual sensitivity – did not reveal any significant group differences, indicating that the emotional valence of the stimuli was apparent to both groups, and that both children with DCD and the controls were equally adept at recognizing facial expressions. However, commission error rate, a measure of behavioral inhibition, was similar between the two groups for all the no-go stimuli except when the target was a happy face. The DCD group made significantly more errors, and failed to withhold responses to happy no-go faces on more than half of the trials. This result suggests that poor affective decision-making of children with DCD on a hot EF task (i.e., the IGT) could be attributed to their heightened

sensitivity to emotionally and motivationally significant stimuli, and their reduced inhibitory control in emotionally rewarding contexts. We suggest the interaction between emotion processing and cognitive control networks underlies this deficit (Rahimi-Golkhandan, Steenbergen et al., 2014). More generally, these results suggest that what characterizes the performance pattern of children with DCD may be a deficit of emotion regulation.

The effects we observed, however, may have been moderated by the choice of no-go stimuli—happy faces were used as no-go stimuli in one condition, with fearful faces as the go stimuli. Being intrinsically rewarding, the presentation of happy no-go faces on 30% of the trials may have created an approach bias (or tendency to respond) that also influenced responses to go stimuli (Hare, Tottenham, Davidson, Glover, & Casey, 2005). For instance, RT to a fearful go face might be quicker immediately after exposure to a happy no-go face. Because sensitivity to rewarding stimuli was shown to be heightened in children with DCD, this "priming effect" could be enhanced, masking real differences between groups in their response to 'negative' stimuli. Moreover, commission errors of the DCD group to fearful faces could be partly due to a built-up tendency to respond to happy go targets, and not necessarily be an indication of incorrectly approaching 'negative' stimuli. Therefore, a more systematic method to investigate approach (or appetitive) tendencies is to pair emotional facial expressions with neutral stimuli.

As well, children younger than 10 years of age often fail to identify a fearful face, even at peak intensity (Gao & Maurer, 2009), while adult-like levels are reached by 5 years of age for *happy* and *sad* faces. Intensity here is defined as the degree of displacement of facial muscles from a neutral state (Hess, Blairy, & Kleck, 1997). While children may identify 'fearful' as an expression different to 'neutral', they may not perceive it as a *negative* expression. Moreover, a fearful expression is often perceived as more positive than a sad face (Dennis, Malone, & Chen, 2009). Tottenham, Hare, and Casey (2011) reported similar

findings in that the RT to fearful faces was only higher than RT to happy faces. Fearful faces were approached faster than angry faces, and significantly faster than sad faces. Generally, the more positive a facial expression, the faster is the RT to it. Taken together, sad faces are a better representation of a negative stimulus in a go/no-go task for children, particularly for those with DCD who have associated emotion recognition deficits (Cummins, Piek, & Dyck, 2005).

In the study presented here, we optimized our assessment of sensitivity to reward by pairing emotional expressions with neutral ones, and enlisting negative stimuli (i.e., sad faces) that were clearly discriminable by children. Therefore, the first of our two aims was to investigate potential differences between TD and DCD groups in the tendency to respond to negatively valenced stimuli. We predicted no group difference in line with earlier behavioral data which showed that the deficit in inhibition related specifically to rewarding (no-go) stimuli. Our second aim was to measure sensitivity to reward in DCD using a more sensitive metric. In line with earlier findings, we predicted that children with DCD would show higher commission errors than the controls in response to happy no-go faces, specifically. This would provide evidence that these children show a heightened sensitivity to reward, which suggests reduced inhibitory control in response to emotionally-laden stimulus events.

2. Method

2.1 Participants

Our total sample comprised 36 children, 12 with DCD (4 boys, 8 girls; $M\ [SD]_{age}$ = 9.82 [1.44]) and 24 TD (10 boys, 14 girls; $M\ [SD]_{age}$ = 10.25 [1.62]). The age range of children was between 7 and 12 years, and there was no significant difference between the groups' average age (p = .44). All participants were selected from a group of primary school students who took part in our earlier studies of hot EF in DCD. Data for the current study were collected 6 months after the first go/no-go study (Rahimi-Golkhandan, Piek et al.,

2014). Children with DCD were identified by parents and teachers as having reduced levels of movement skill that interfered with day-to-day functional activities (Criterion B). Parents confirmed that these motor problems were evident before school age (Criterion C). Motor difficulties, reported earlier by parents and/or teachers, were confirmed by a score of 85 or below on the Neurodevelopmental Index (NDI) of the McCarron Assessment of Neuromuscular Development (MAND; McCarron, 1997) (Criterion A); M [SD] $_{NDI}$ = 77.58 [7.64]. No child with a diagnosed intellectual disability, visual impairment, or neurological condition affecting movement was included in this study (Criterion D). Given that all children were attending mainstream primary schools and none were attending remedial classes for literacy or mathematics, we considered that children were sufficiently matched on intelligence at the group level; as such, the effects of intelligence on potential group differences in EF could be ruled out in the current study. The control group was made up of children with NDI of at least 100 (M [SD] $_{NDI}$ = 109.21 [9.67]). We also excluded those who had a prior or current diagnosis of other developmental disorders (e.g., autism, Attention Deficit/Hyperactivity Disorder (ADHD)). The study was approved by institutional research ethics committee and all children and their parents gave informed consent to participate.

2.2 Materials

2.2.1 Go/No-Go task. The *emotional* go/no-go task was based on the work of Ladouceur et al. (2006) and was programmed in E-Prime software (Schneider, Eschman, & Zuccolotto, 2002).

The task included pictures of neutral/calm, happy, and sad facial expressions of a group of men and women. All pictures were borrowed with permission from the website of the Sackler Institute for Developmental Psychology (*https://www.sacklerinstitute.org*), were in black and white, and had the same size and luminance. The task had four runs, divided into two blocks: in each single run neutral expressions were paired with either sad or happy faces.

Each run had 28 go and 12 (or 30%) no-go trials, presented in a pseudorandom order. The go target in one run would become the no-go target in the next, with the order of blocks and runs counterbalanced over the participants. Before the start of each run, children received clear instructions on the screen about the facial expression that would serve as the go target, and were asked to respond (by pressing the *spacebar*) as quickly as possible to *only* that particular expression, and not the other. To make sure children understood the task, they were asked to take part in 12 practice trials before the onset of each run. Each picture (10 x 13 cm) was displayed at the centre of a 13" laptop screen for 500ms, followed by a 1500ms inter-stimulus interval during which a white fixation cross was presented centrally. Therefore, for each trial, responses were permitted up to 1500ms following stimulus presentation. Omission errors and RT (to the go targets) as well as the commission errors (to the no-go stimuli) were the main dependent variables of performance in this task. Commission errors index behavioral inhibition; low errors reflect a well-developed ability to withhold responses to no-go targets. Omission errors, the measure of attention, were defined as a failure to respond to go targets, and reflect the tendency to approach each stimulus type (Tottenham et al., 2011). Similarly, RT to the go targets was also used to measure the tendency to approach go stimuli; specifically, approach tendency was assessed as a function of the emotional valence of the stimulus.

In order to confirm that the two groups were equally adept at discriminating between facial expressions, *d'*, which is a widely used measure of perceptual sensitivity, was calculated. Low omission errors (i.e., high approach rate) do not necessarily reflect a more developed ability to recognize the stimuli. For example, if a child presses the spacebar constantly in response to both go and no-go faces, omission errors would be negligible; however, commission errors would be maximal. This pattern indicates poor perceptual sensitivity to the target. The *d'* index provides a highly valid measure of sensitivity by

combining the likelihood of correctly detecting go stimuli with the likelihood of commission errors (aka false alarms), and is calculated as follows: $d' = z(H) - z(F)$, where $z(H)$ is the standardized score for correct hits, and $z(F)$ is the standardized score for false alarms. This index takes into account the respondent's bias and provides an index of emotion recognition (Tottenham et al., 2011) where high values indicate a more developed ability to discriminate facial expressions as go and no-go stimuli.

2.2.2 McCarron Assessment of Neuromuscular Development (MAND). The MAND (McCarron, 1997) is a test of motor ability that can be administered to individuals from 3.5 to adulthood. The MAND comprises 10 items, including five fine-motor and five gross-motor. A summary of performance, or NDI, is derived from the sum of scaled scores on the 10 tests, relative to age norms, and conforms to a normal distribution with a mean of 100 and *SD* of 15. The psychometrics for the MAND are good. Test-retest reliability of sub-tests over a 1-month period ranges between .67 and .98 (McCarron, 1997). Good construct validity has also been reported in Australian (Hands, Larkin, & Rose, 2013; Tan, Parker, & Larkin, 2001) and US populations (McCarron, 1997).

2.3 Procedure

All testing sessions were conducted during children's class time, but in a separate room, free from distractions. In order to control for order effects, half of the children were first assessed on the MAND, while the others took part in the go/no-go task first. Each child took approximately 15 minutes to complete the experimental tasks.

2.4 Data Analysis

Based on the prior observation of strong group effects on measures of cold and hot EF (Wilson et al., 2013; Rahimi-Golkhandan, Steenbergen et al., 2014), a sample size of 12 children per group was sufficient to achieve a recommended power level of .80 (Faul, Erdfelder, Buchner, & Lang, 2009). When based on previous data using the same or similar

paradigm, mixed designs of this type are particularly efficient in testing hypotheses at the group level (Cohen, 1988). In lieu of our specific hypotheses, we further optimized statistical power by running a small set of parametric planned contrasts on our main dependent measures – commission errors, omission errors, and RT. Gender was included as a covariate in all group comparisons to control for differences in gender ratio. For commission errors, planned comparisons were conducted for happy and sad faces. These analyses addressed the question of whether reduced inhibitory control affects the performance of children with DCD in response to *both* positive and negative no-go cues *or* is specific to rewarding stimuli (i.e., positive facial cues). Individual differences in commission errors were also analysed within each group. Omission errors and RT to go stimuli were also compared between groups using planned contrasts to better understand approach tendencies to 'go' stimuli, particularly negatively-valenced ones. To temper the interpretation of significance tests, effect sizes and their 95% confidence intervals (CI) were calculated for *all* group comparisons on the above outcome variables. We use the abbreviations, '*neutral (H)*' and '*neutral (S)*' to refer to the stimulus runs in which neutral faces ('go') were paired with happy and sad faces ('no-go'), respectively.

3. Results

3.1 Commission Errors

Figure 1 shows mean commission errors for each group (±*SE*). The planned comparison between groups confirmed that the DCD group made significantly more commission errors than controls for happy faces ($p = .019$) while the comparison for sad faces was not significant ($p = .70$). Figure 2 (left panel) confirms that effect sizes varied as a function of stimulus type. Within-group comparisons revealed a similar pattern for each group. For controls, the average commission error to sad faces was higher than that of any other no-go stimulus ($0.46 < d < 1.88$). For the DCD group, errors for sad no-go stimuli were

higher than those for neutral (H) (d = 1.56) and happy (d = 0.68) faces, but not neutral (S) faces (p = .07, d = 0.58). Finally, both groups had significantly more errors to the neutral (S) stimuli than neutral (H) (DCD: p = .003, d = 1.06 (95% CI [0.33, 1.76]); control: p < .001, d = 0.82 (95% CI [0.35, 1.28])).

3.1.1 Individual differences. Figure 3 presents the number of commission errors to happy no-go targets for each child, divided by group. Almost half (n = 5) of the DCD group approached happy no-go faces on more than 50% the trials, compared with only 1 of 24 (or 4%) controls; all five children with DCD recorded errors above the upper limit of the 95% CI for controls. Alternately, four children with DCD had fewer errors than the average for controls.

3.2 Omission Errors

The average proportion of trials that children failed to respond to a go target is presented in Figure 4. The DCD group had a lower approach rate to all go stimuli with the exception of neutral faces paired with happy no-go targets. However, there was no statistically significant difference between the groups in response to either happy (p = .12) or sad (p = .18) faces (see Figure 2 middle panel for effect size measures). Within-group comparisons showed more omission errors to sad faces than any other stimulus (DCD: p <.003, $d_{\text{neutral(H)}}$ = 1.43, d_{happy} = 0.89, $d_{\text{neutral(S)}}$ = 0.98; Control: p <.002, $d_{\text{neutral(H)}}$ = 0.79, d_{happy} = 1.05, $d_{\text{neutral(S)}}$ = 0.66). More importantly, for the DCD group, omission errors to neutral faces were higher when paired with sad no-go faces (i.e., neutral (S)) compared with neutral (H), p = .045, d = 0.60; the same was not true of controls, p = .32, d = 0.21.

3.3 Reaction Time

Figure 5 shows the average RT (±SE) for go trials in each group. There was a significant group difference in RT for sad faces only (p = .048). However, effect sizes for other stimuli were also moderate (see Figure 2 – right panel). Within-group analyses

indicated that the RT of each group to happy faces was significantly faster than their RT to any other go stimulus (DCD: $d_{\text{neutral(H)}} = 0.48$, $d_{\text{sad}} = 1.03$, $d_{\text{neutral(S)}} = 0.86$; Control: $d_{\text{neutral(H)}} = 0.64$, $d_{\text{sad}} = 0.72$, $d_{\text{neutral(S)}} = 0.40$).

3.5 D-Prime (d')

This measure (d') indicates the degree of sensitivity to each type of facial expression. Figure 6 shows that happy faces were the most easily discriminated of the go targets, and sad faces the least. For both groups, the d' for sad faces was significantly less than that for happy faces (DCD: $p < .001$, $d = 1.13$ (95% CI [0.38, 1.84]); Control: $p < .001$, $d = 1.23$ (95% CI [0.69, 1.76])) and neutral (H) (DCD: $p < .001$, $d = 1.28$ (95% CI [0.49, 2.04]); Control: $p < .001$, $d = 1.34$ (95% CI [0.77, 1.88])). Moreover, a comparison of effect sizes showed that both children with DCD and controls had more difficulty responding to neutral faces when they were paired with sad no-go faces than happy no-go targets (DCD: $p = .004$, $d = 0.73$ (95% CI [0.07, 1.36]); Control: $p < .001$, $d = 1.75$ (95% CI [1.10, 2.39])). None of the between-group comparisons of d' was significant (see Figure 6 for effect size measures).

4. Discussion

The broad aim of our study was to investigate hot EF in children with DCD. Specifically, we examined the ability of children with and without DCD to respond to both positively and negatively valenced stimuli in the context of an emotional go/no-go task. Broadly, the pattern of responses confirmed our working hypothesis that children with DCD have difficulty modulating their approach to rewarding stimuli when the task demands that this behavior be inhibited. In the discussion that follows, we first interpret group differences in the tendency to approach 'negative' stimuli and, second, the finding of reduced response inhibition in DCD when responding to emotionally salient (and 'positive') stimuli. The discussion then turns to the broader implications of these results and those presented in our earlier studies of hot EF in DCD, for theory development and clinical practice.

4.1 Approaching Negative Social Cues

In our study, slower RTs to sad faces in DCD could be interpreted as a reduced ability to control *affective interference* and approach 'negative' stimuli. Behavioral responses are biased by affective signals about the rewarding or aversive qualities of a stimulus; however, we are also required to override this immediate bias when required to tackle novel tasks that may be experienced as emotionally challenging or aversive. This ability to ignore the unpleasant aspects of a stimulus and act in a manner incongruent with the affective signal is something that improves gradually with age (Hare et al., 2008).

Developmental factors may explain the difficulty *both* groups had in distinguishing sad and neutral faces, as shown by low *d'* values. For each group, the lowest *d'* was in response to sad faces; the value for neutral (S) was also significantly lower than neutral (H). Children as young as 7 are quite adept in detecting exemplars of facial expressions, but *only* when these expressions are intense. For example, children are less sensitive than adults to typical expressions of sadness. The confusion associated with recognizing a low intensity sad face could bias children to perceive the expression as neutral (Gao & Maurer, 2009). Sad and neutral faces are also considered less arousing than the other expressions (e.g., fearful) (Dennis et al., 2009). Similar emotion recognition difficulties have been reported in developmental disorders like autism (Yerys, Kenworthy, Jankowski, Strang, & Wallace, 2013). Immaturities in younger children's sensitivity to sad and neutral (S) faces may explain the associated *d'* prime values, as well as high commission and omission errors in response to sad and neutral (S) faces.

4.2 Withholding Responses to Positive Social Cues

Data reported here corroborated an earlier study showing a reduced ability in DCD to inhibit responses to emotionally rewarding cues. In general, the DCD group made significantly more commission errors when the no-go stimulus was a positive social cue.

Earlier we argued that the mechanism underlying this issue was likely to involve neural integration of inhibitory control and emotion processing systems (Rahimi-Golkhandan, Steenbergen et al., 2014). Disruption to the coupling between these systems would compromise affective decision making (Kohls, Peltzer, Herpertz-Dahlmann, & Konrad, 2009) and self-regulation (Hare & Casey, 2005). Intriguingly, problems with day-to day planning and organisation are persistent issues in DCD, with implications for adaptive behavior in adolescence and early adulthood (Tal Saban, Ornoy, & Parush, 2014).

Evidence linking motor skill difficulties and EF is based not only on behavioral data but also recent work into the neurobiological mechanisms of motor and cognitive development in both typically and atypically developing children (Johnson, 2011; Koziol, Budding, Chidekel, 2011; Zwicker, Missiuna, Harris, & Boyd, 2011). It is estimated that about half of the children diagnosed with DCD also show reduced EF (Sugden, Kirby, & Dunford, 2008; Willcutt, & Pennington, 2000), particularly deficits of inhibitory control (Wilson et al., 2013). These deficits are evident across a range of tasks and paradigms: inhibition of attentional shifts to compelling but invalid cues (Mandich, Buckolz, & Polatajko, 2002; Wilson, Maruff, & McKenzie, 1997), non-verbal inhibition of motor responses on the Verbal Inhibition, Motor Inhibition (VIMI; Henry, Messer, & Nash, 2012) task (Leonard, Bernardi, Hill, & Henry, 2015), as well as anti-reach movements (Ruddock et al., 2015). Indeed, a direct relationship between the severity of DCD symptoms and deficits of inhibitory control has been suggested (Mandich et al., 2002). Critically, our recent work has shown that EF deficits in DCD are not confined to 'cold' aspects of cognitive control, but also affect performance in task contexts that have a strong emotional/motivational valence. In the current study, for instance, deficits of inhibitory control in the DCD group, as operationalized by commission errors on an emotional go/no-go task, was stimulus-specific:

relatively high commission errors were confined to compelling, positively-valenced cues (i.e., happy faces).

4.3 The Possible Neural Underpinnings of EF Deficits in DCD

Results of the current study can be reconciled within a current neurodevelopmental framework—the theory of *interactive specialization* (Johnson & Munakata, 2005). This theory suggests overlap in the development of motor and cognitive functions (Diamond, 2000) and the possibility that disruptions in one domain can have far reaching consequences for another. Of the six major white matter networks (WMN) in the brain, two consistent hubs from early childhood to adulthood include regions across parietal and frontal lobes (Chen, Liu, Gross, & Beaulieu, 2013). A maturational lag or disruption of the fronto-parietal network, which underlies adaptive cognitive control (Sripada, Kessler, & Angsdadt, 2014), may compromise the development of not only EF but other processes arising from specialization within and interaction between WMNs. More specifically, disruption of the fronto-parietal network would undermine inhibitory control, with implications for motor development and supervisory attentional control (Sripada et al., 2014).

Recent fMRI studies have linked executive control deficits in DCD to inefficient activation of the inhibition network comprising middle frontal, anterior cingulate (ACC) and inferior parietal cortices (Querne et al., 2008), and visuomotor deficits to hypoactivation of parietal regions (Kashiwagi & Tamai, 2013). Maturation of WMNs spanning frontal and parietal sites are also associated with the development of cognitive control in typical children (Casey, Galvan, & Hare, 2005), and response inhibition, more specifically. One possibility is that inhibitory control deficits in DCD may disrupt performance on both traditional tests of (cold) EF and hot EF tasks.

Moreover, WM and inhibition have an interactive relationship such that activation of mental representations relevant to a particular goal-directed task is accompanied by automatic

inhibition of irrelevant stimuli and actions (Diamond, 2002; Roberts & Pennington, 1996). Both of these processes are subserved by overlapping neural networks (i.e., DL-PFC, ACC) (Braver et al., 2001; Owen, 2000). Situations that are high in response uncertainty or that present numerous distractors or extraneous stimuli, constitute a high load on EF—this is problematic for children with DCD.

Cognitive re-appraisal strategies have an important role in suppressing or regulating (automatic) responses to highly salient stimuli—those with a strong appetitive or motivational component. For example, mental readying or rehearsal can help prime preferred responses, even in the face of compelling cues (Williams, Bargh, Nocera, & Gray, 2009). These aspects of the cognitive control of action mature with development of DL-PFC (selective attention, WM), and VL-PFC (inhibition) (Ochsner et al., 2012).

4.4 Implications for Motor Learning and Control in DCD

In DCD, delayed maturation of executive control networks (spanning PFC and its reciprocal connections to posterior association cortex) may explain broad-based difficulties in the planning, execution, and control of action. More precisely, hypoactivity of the attentional network in DCD including the DL-PFC (Querne et al., 2008) would compromise the initial stages of motor learning and action planning (Brown-Lum & Zwicker, 2015). Further, reduced prefrontal involvement in action planning may contribute to the high comorbidity of attentional problems with DCD (Deng et al., 2014), with consequences for both motor learning and selective inhibition of responses. Our current findings suggest that a reduced ability to inhibit responses to emotional cues may bias the child's attention in ways that do not facilitate skill learning. To illustrate, certain movement contexts, especially novel or difficult ones, may be experienced as aversive by children with DCD, especially if their prior learning experiences have been negative. Conversely, passive or less physically-intense activities may be more appealing or enable the child to avoid negative emotions (like the

embarrassment that might be felt when attempting a new motor skill). It is important to think about the process of learning and motor behavior as something that is inexorably entwined with the experience of emotion. Indeed, most real-world problems enlist a blend of hot and cold EF when action must be planned and implemented with respect to a goal that holds some motivational/emotional significance (Zelazo, 2015).

4.5 Implications for Intervention

Interventions designed to improve motor skill in children with DCD are more likely to be effective when reasonable adjustments are made for co-development of EF (Reinert, Po'e, & Barkin, 2013). Indeed, a recent review by Diamond (2013) shows that the strongest effect of cognitive training across a range of disorders is shown for children with the most severe EF deficits. This finding and our data underscore the importance of assessing EF in DCD as a standard protocol and then designing interventions that target both motor and cognitive skills, hopefully mitigating the adverse consequences of each (Gonzalez et al., 2014). In the very least, motor interventions may need to modified in delivery to accommodate the reduced level of EF in many children with DCD.

One important aspect of training is the *mode of delivery* for task instructions. Action simulation (observation plus imagery) using video-based modeling has been shown to be equally effective as traditional physical therapy in promoting skill acquisition in DCD (Wilson, Thomas, & Maruff, 2002). By comparison, verbal instructions may not yield the same training effects in DCD as those seen in typically developing children, at least with respect to motor imagery (Williams, Thomas, Maruff, & Wilson, 2008). While the reasons for this effect remain unclear, it is possible that task instructions that place excessive demands on WM and/or attention may disadvantage the child with DCD (Wilson et al., 2013). Among other strategies, part-whole learning, providing more opportunities for rehearsal, and increasing task complexity in a more gradual manner – compared with the rate

used for TD children – may also help scaffold the learning process for children with DCD, compensating for reduced EF and a generally slower rate of learning.

Interestingly, preferred approaches to EF training in children involve aspects of physical activity and skill training (Diamond, 2013). Such blended approaches are likely to be particularly effective for children with DCD given the overlap between emotional regulation, cognitive control and movement skill; blended approaches afford ways of breaking down mediation factors that lie between DCD and its psychosocial consequences (Cairney, Rigoli, & Piek, 2013). Task-specific training (like martial arts or dance) presents a potentially powerful means of modifying not only cognitive control, but also the risks posed by inactivity and reduced movement skill. Participation in such training requires that child offset the immediate (emotional) challenge of learning novel tasks in a novel context, by the experience of a different set of valued outcomes (e.g., enhanced participation, the immediate joys of success, enhanced movement capability and improved self-efficacy). Indeed, interventions are more effective through an integrated approach that aims to improve happiness, physical ability, self-efficacy, and feelings of social belonging and support (Diamond, 2012).

4.6 Limitations and Future Research

The heterogeneity of performance within the DCD group on the go/no-go task has previously been reported on related cognitive and motor tasks (Green, Chambers, & Sugden, 2008), particularly when DCD samples include children with other comorbid conditions, either explicitly or in an uncontrolled manner. In our study, we recruited children without any previous or existing neurological or psychiatric conditions, including other developmental disorders such as ADHD. This raises the possibility that there might be subtypes of DCD based on their motor and cognitive profile – even when no comorbid developmental disorder exists. While a sub-type analysis was beyond the scope of our study, follow-up studies are

encouraged to consider such an analysis, and to extend sampling over a broader age range. Finally, our interpretation of hot EF deficits in DCD was based on behavioral data generated from experimental tasks like the HDT and go/no-go task. Needed now are morphological studies into the development and connectivity of major WMNs in DCD, as well as controlled fMRI evaluations using parametric techniques (Wilson et al., 2013). Together, this work will clarify what appear to be some intriguing points of differentiation in the development of neurocognitive systems in DCD and TD.

4.7 Conclusion

The current study shows that EF deficits in DCD are not confined to 'cold' aspects of cognitive control, but also disrupt functioning in 'hot', affective contexts. Disrupted coupling between cognitive control and emotion processing networks, such as fronto-parietal and fronto-striatal networks, may contribute to reduced inhibitory control in DCD. More specifically, emotionally significant and rewarding stimuli constitute a higher load on inhibitory control than neutral or negative stimuli, making it more difficult for children with DCD to modulate their approach tendencies. Reduced inhibitory control in emotionally-laden situations, involving both motor and non-motor activities, would undermine action planning, execution and control, and potentially disrupts self-regulation in DCD; such deficits have been reported in recent studies (e.g., Tal Saban et al., 2014). We suggest interventions that address both motor skills and the co-development of EF are more likely to facilitate skill learning and cognitive control in DCD, with transfer to organization and execution of daily activities.

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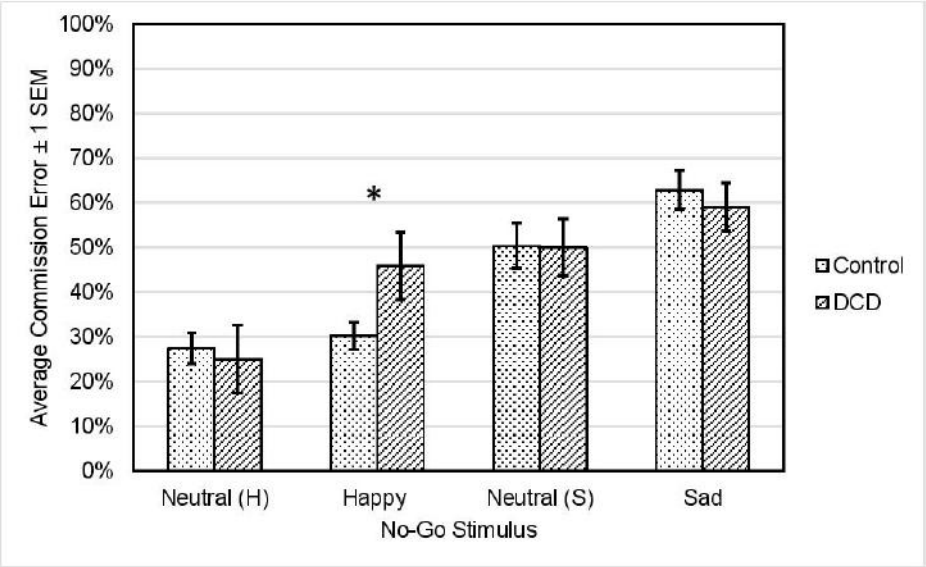

Figure 1. Mean (±SE) commission errors to no-go targets in the DCD and control groups (*$p < .05$)

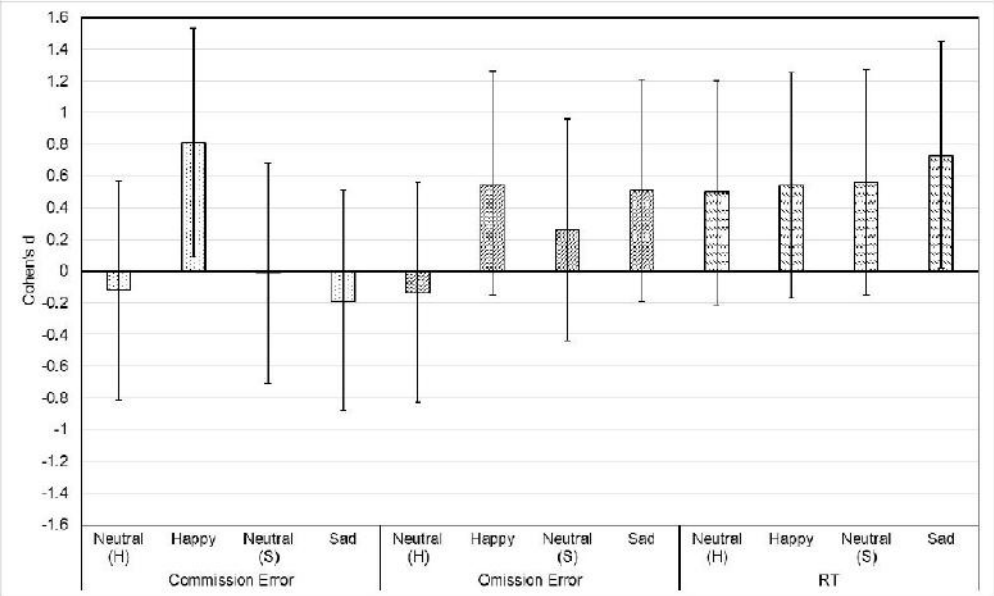

Figure 2. Effect sizes (Cohen's d ± 95% CI) for group differences. Positive values indicate more commission and omission errors and slower RT for children with DCD.

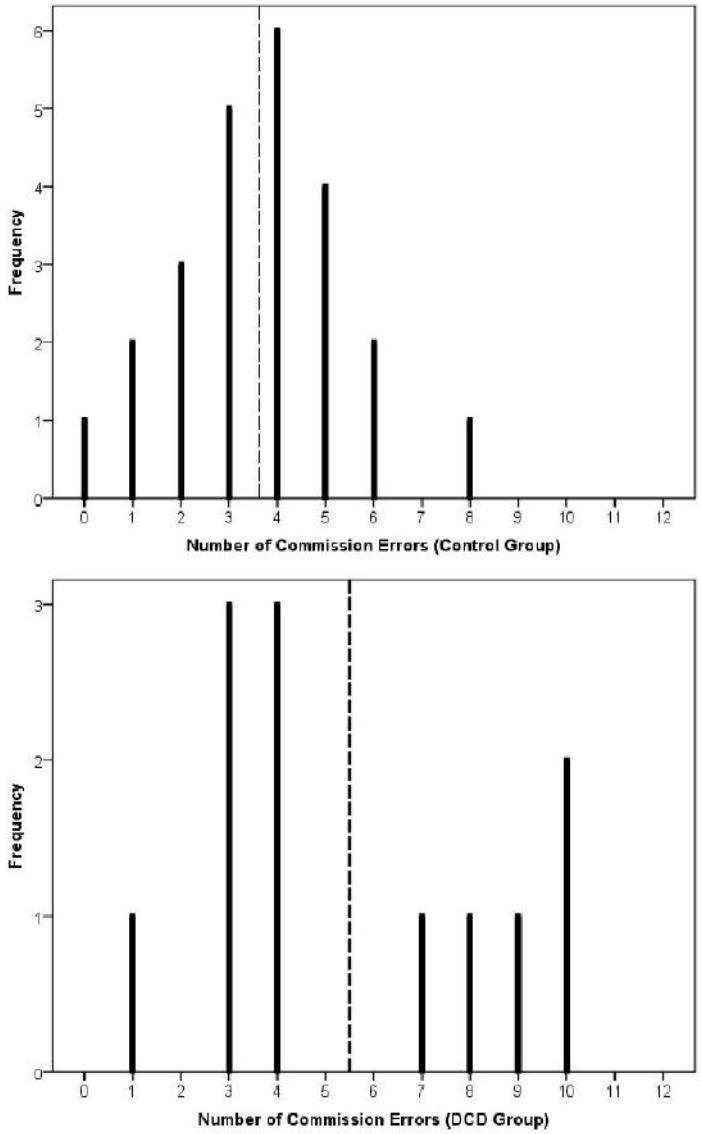

Figure 3. Frequency distribution of commission errors to happy no-go targets. The *dotted line* represents group average. Maximum possible number of commission errors was 12 (trials).

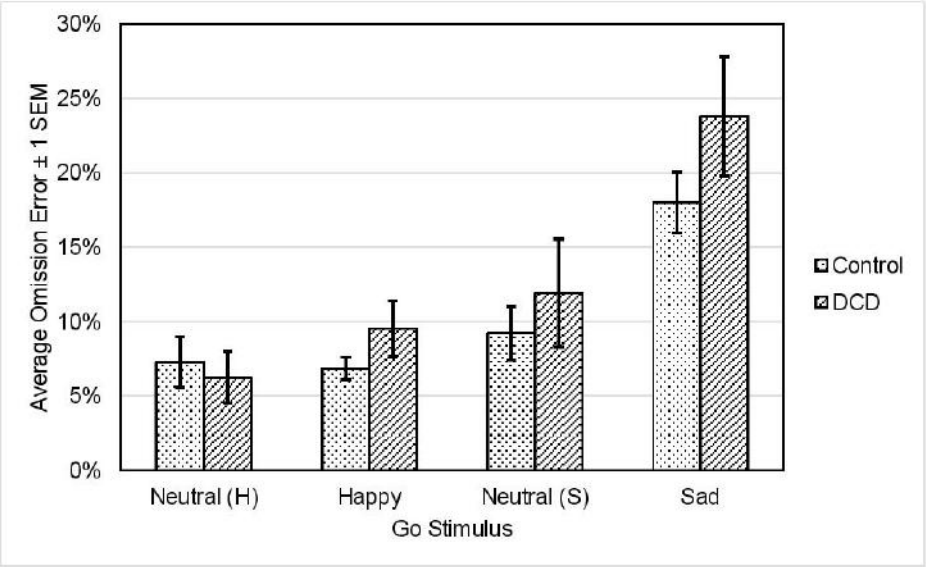

Figure 4. Mean (±SE) omission errors to go targets in the DCD and control groups

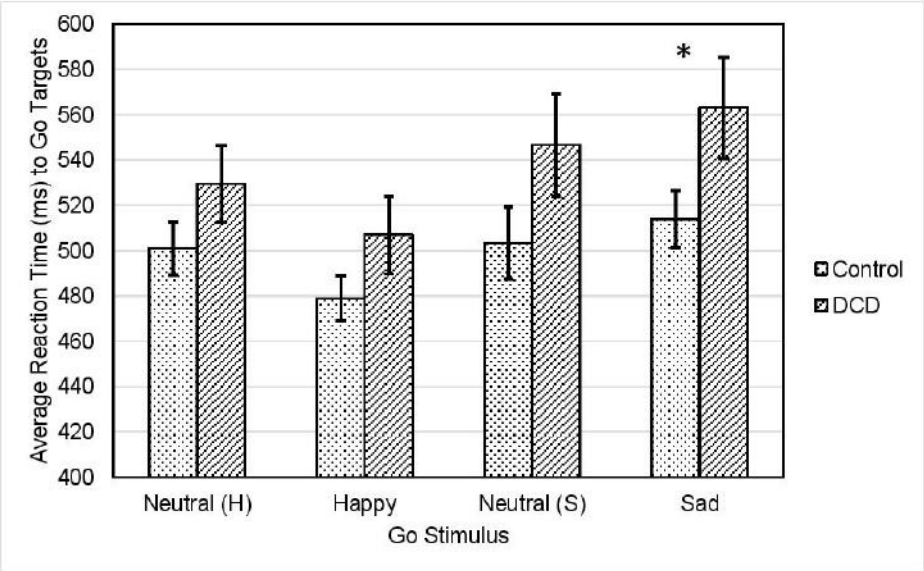

Figure 5. Mean (±SE) RT to go targets in the DCD and control groups (*$p < .05$)

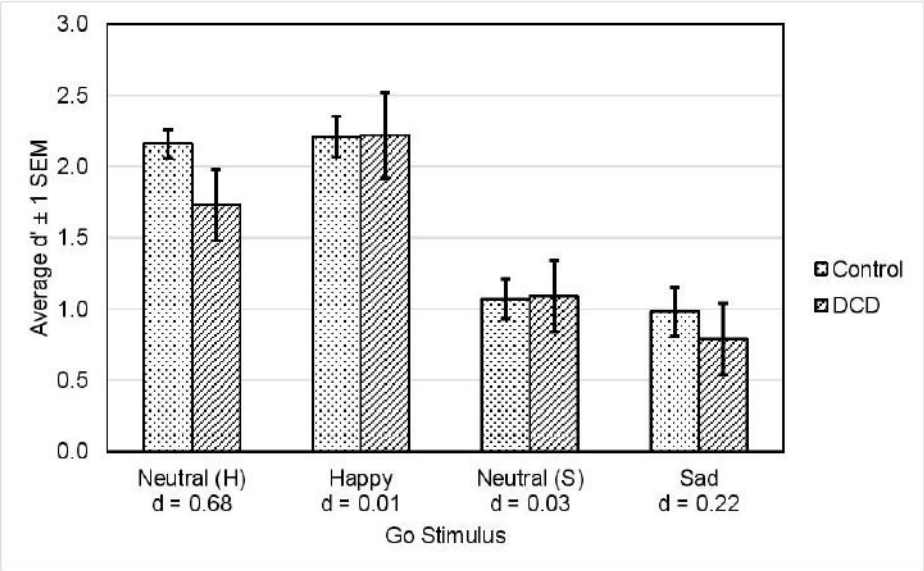

Figure 6. Mean (±SE) d' for DCD and control groups in response to the go stimuli