UNIVERSITY OF SOUTHAMPTON

FACULTY OF SOCIAL, HUMAN AND MATHEMATICAL SCIENCES

Psychology

The Neuropsychology of Conduct Disorder: the impact of comorbid Attention-Deficit/Hyperactivity Disorder

by

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Thesis for the degree of Doctor of Philosophy

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ABSTRACT

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THE NEUROPSYCHOLOGY OF CONDUCT DISORDER: THE IMPACT OF COMORBID ATTENTION-DEFICIT/HYPERACTIVITY DISORDER

Nadia Peppa

Conduct Disorder (CD) is characterised by a persistent and pervasive pattern of aggressive and antisocial behaviour that violates the rights of others or in which major age-appropriate societal norms are violated. Researchers have argued that this pattern of sensation-seeking behaviour stems from a higher threshold for emotional stimulation in children and adolescents with CD compared to typically-developing individuals. In addition, studies have found a reduced sensitivity to reward which interferes with the learning of appropriate behaviours. CD and Attention-Deficit/Hyperactivity Disorder (ADHD) are considered to be highly comorbid both in clinical and community samples. Research has indicated that individuals with CD+ADHD have poor social functioning, poor socio-economic outcomes later in life and are likely to drop-out or be kicked out of educational institutions. However, neuroscience research has not properly addressed the issues surrounding the effects of comorbid ADHD on cognitive and emotional processing in CD. Understanding these effects will allow us to develop more sophisticated causal pathways, which in the longer term may aid clinicians to administer treatments tailored specifically to patients' individual needs.

The present study investigated the effects of comorbid ADHD on the clinical and neuropsychological profiles of adolescents with CD, by comparing groups of adolescents with CD-only (CD-ADHD; n = 23), comorbid CD+ADHD (n = 28) and a typically-developing control group (TDC; n = 30). We used a range of clinical and questionnaire-based assessments, as well as a series of behavioural tasks that examined Executive Functions (EFs), facial emotion recognition and perspective-taking. We found that CD was independently associated with impairments in impulsive choice ("hot" EFs), whereas comorbid ADHD was associated with impairments in interference control and working memory ("cool" EFs). Furthermore, we found that a broad pattern of facial emotion recognition deficits was limited to those individuals with CD+ADHD. Our study was also the first study to explicitly investigate the impact of comorbid ADHD on perspective-taking in CD. Our results do not provide evidence for a deficit in perspective-taking in adolescents with CD, regardless of ADHD comorbidity. Considered together, these results provide some insight on the impact of ADHD on the neuropsychology of CD and can be useful to both researchers and clinicians when considering new research designs and clinical interventions.

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DECLARATION OF AUTHORSHIP

I, Konstantina Peppa, declare that this thesis and the work presented in it are my own and has been generated by me as the result of my own original research.

The neuropsychology of Conduct Disorder: the impact of comorbid Attention-Deficit/Hyperactivity Disorder

I confirm that:

1. This work was done wholly or mainly while in candidature for a research degree at this University;
2. Where any part of this thesis has previously been submitted for a degree or any other qualification at this University or any other institution, this has been clearly stated;
3. Where I have consulted the published work of others, this is always clearly attributed;
4. Where I have quoted from the work of others, the source is always given. With the exception of such quotations, this thesis is entirely my own work;
5. I have acknowledged all main sources of help;
6. Where the thesis is based on work done by myself jointly with others, I have made clear exactly what was done by others and what I have contributed myself;
7. Parts of this work have been published before submission as:

Signed: K Peppa

Date: 31/07/2017

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This thesis is dedicated to my first cousin Maria, who was killed in a motorbike accident just before I embark on my PhD journey, on the 13th of July 2011. She was 17 years old.

Until we meet again Maraki.

Abbreviations

ACC	Accuracy
ADHD	Attention-Deficit/Hyperactivity Disorder
ADHD-C	Attention-Deficit/Hyperactivity Disorder – Combined type
ADHD-H/I	Attention-Deficit/Hyperactivity Disorder – Hyperactive/Impulsive type
ADHD-I	Attention-Deficit/Hyperactivity Disorder – Inattentive type
ANOVA	Analysis of Variance
APA	American Psychiatric Association
AQ	Autism-Spectrum Quotient
ASD	Autism-Spectrum Disorder
BFRT	Benton Facial Recognition Test
BIS	Barratt Impulsiveness Scale
BIS/BAS	Behavioural Inhibition/Activation Scales
CAMHS	Child and Adolescent Mental Health Services
CD	Conduct Disorder
COM	Commission errors
CU	Callous-Unemotional
DBD	Disruptive Behaviour Disorders
DeFT	Delay Frustration Task
DSM	Diagnostic and Statistical Manual of Mental Disorders
DV	Dependent Variable
EF	Executive Function
FFS	Fight/Flight System
GAD	Generalised Anxiety Disorder
GP	General Practitioner
IC	Interference Control
ICU	Inventory of Callous-Unemotional Traits
IQ	Intelligence Quotient
IV	Independent Variable
K-SADS-PL	Kiddie-Schedule for Affective Disorders and Schizophrenia – Present & Lifetime
LL	Larger Later
MACC	Mean Accuracy
MDD	Major Depressive Disorder
MIDA	Maudsley Index of Delay Aversion
MRT	Mean Reaction Time
NES	Neighbourhood Environment Scale
NHS	National Health System
NICE	National Institute for Health and Care Excellence
OCD	Obsessive Compulsive Disorder
ODD	Oppositional Defiant Disorder
OM	Omission errors
PRU	Pupil Referral Unit
PTSD	Posttraumatic Stress Disorder
RI	Response Inhibition

RT	Reaction Time
SD	Standard Deviation
SE	Standard Error
SES	Socio-Economic Status
SS	Smaller Sooner
STAI	State-Trait-Anxiety Inventory
SWM	Spatial Working Memory
TD	Temporal Discounting
TDC	Typically-Developing Controls
ToM	Theory of Mind
VIM	Violence Inhibition Mechanism
VSWM	Visuo-Spatial Working Memory
WASI	Wechsler Abbreviated Scale of Intelligence
WM	Working Memory
YOS	Youth Offending Service
YOT	Youth Offending Team
YPI	Youth Psychopathic Traits Inventory

Chapter 1 Conduct disorder – key terms and concepts

1.1 General Introduction

Conduct disorder (CD) is characterised by a persistent and pervasive pattern of aggressive and antisocial behaviour that violates the rights of others or in which major age-appropriate societal norms are violated (APA, 1994). The term Disruptive Behaviour Disorders (DBD) refers to a group of externalising disorders consisting of CD and Oppositional Defiant Disorder (ODD). ODD is characterised by a pattern of recurrent, defiant, disobedient and hostile behaviour with onset in childhood or adolescence. In DSM-IV (APA, 1994), the existence of a diagnostic hierarchy between ODD and CD meant that ODD could not be diagnosed in the presence of CD as the latter was seen as a more serious and impairing disorder. As Angold, Costello, & Erkanli (1999) note, this hierarchy has resulted in the use of combined ODD/CD groups in many empirical studies. However, research has indicated that the majority of children with ODD will not develop CD in their lifetimes (Rowe, Maughan, Pickles, Costello, & Angold, 2002). Furthermore, Nock, Kazdin, Hiripi, and Kessler (2006) provided evidence that ODD should be regarded as a distinct disorder that is somewhat independent of CD.

CD often presents clinically along with Attention-Deficit/Hyperactivity Disorder (ADHD), another common disorder with a childhood onset. These three externalising disorders, i.e. ODD, CD and ADHD, are considered to be the most common disorders for which children and adolescents are referred to Child and Adolescent Mental Health Services (CAMHS; Green et al., 2005). The same authors reported that DBDs occur in 7% of 11-15 year olds in the UK while Nock et al. (2006) reported that the lifetime prevalence of CD in the USA was 9.5%. The worldwide estimated prevalence of ADHD for children and adolescents has been reported to be between 5.9% to 7.1% (Willcutt, 2012). In terms of the comorbidity between the two disorders, the prevalence rate of ADHD in delinquent adolescents and young adults ranges from 4% to 72% (mean prevalence 26% across studies; Vermeiren, 2003).

Given the overlap between CD and ADHD, as well as the evidenced prevalence, it is vital to understand the behavioural and neuropsychological profile of individuals with CD and those with CD and comorbid ADHD. To examine this important issue, this thesis will compare groups of adolescents with CD alone (CD-ADHD), CD with comorbid ADHD (CD+ADHD) and typically-developing adolescents across a number of measures. These are the groups'

demographic, clinical and personality characteristics, and their performance on a number of neuropsychological tasks which examine several cognitive domains.

Chapter 1 will focus on defining the key terms and concepts relevant to this thesis and will introduce the reader to the issue of comorbidity in CD. Chapter 2 will deal with the neuropsychological deficits associated with CD and ADHD and will also present the cognitive domains of interest. Chapter 3 will describe the general methods and procedures followed in this thesis, together with an analysis and discussion of the demographic, clinical and personality characteristics of our sample. The empirical chapters will examine deficits in "cool" Executive Functions (Chapter 4), deficits in "hot" Executive Functions (Chapter 5), emotion processing deficits (Chapter 6) and deficits in Theory of Mind (Chapter 7). This thesis will conclude with a general discussion (Chapter 8).

1.1.1 Operational definition of comorbidity

The term comorbidity was introduced in medicine by Feinstein (1970) as the co-occurrence, either over time or concurrently, of two separate disorders within the same individual. It has been suggested that comorbidity within the medical model of disease however, implies common underlying disease entity (Lilienfeld, Waldman, & Israel, 1994). Within a developmental psychopathology context, the notion of comorbidity is applied differently. The co-occurrence of disorders is being researched within a dynamic environment of complex interactions between risk and resilience variables from a number of domains e.g. cognitive, behavioural, and genetic. Rather than focusing on one common underlying disease entity, within the context of developmental psychopathology, developmental disorders are being researched with a focus on shared and distinct etiological processes (Drabick & Kendall, 2010). In the 1990s the study of comorbidity was referred to as an important challenge that mental health professionals and researchers had to face (Kendall & Clarkin, 1992). Data from the Dunedin Longitudinal study (Anderson, Williams, McGee, & Silva, 1987) indicated that comorbidity between different disorders occurred much more often: a. than would be expected by chance; and b. than it could be accounted for by the rate of occurrence of each individual disorder separately in the general population (Caron & Rutter, 1991).

The importance of studying comorbidity can be summed up in one sentence: its presence, if ignored, can distort research findings and clinical diagnoses. This is because the overlapping symptoms may mask the true underlying cause of the comorbid disorder. However, as Rutter,

(1997) highlighted in his pivotal paper about the different concepts of comorbidity, "if properly dealt with, it also provides important research opportunities to test hypotheses about causal mechanisms." (p. 265). Indeed, the study of comorbidity in neurodevelopmental disorders provides an important platform to explore different psychopathological and/or neuropsychological patterns of disorder manifestation and to clarify the role of possible mediating mechanisms. Thus, knowing more about the role of comorbidity in psychiatric disorders, researchers and clinicians will be in a better position to diagnose and treat individuals and manage disorders in a more effective way.

For the purposes of this thesis the terms "comorbid" or "comorbidity" will be used to describe individuals with a diagnosis of two or more psychiatric disorders and the literature examining the overlap of distinct psychiatric disorders across individuals, respectively.

1.1.2 A note on heterogeneity

Heterogeneity has been identified as a significant contributor to the difficulties with diagnosing and treating developmental disorders (Sonuga-Barke, 2016). Sonuga-Barke stressed the importance of distinguishing between two forms of heterogeneity. Surface heterogeneity refers to the variability of the expression of symptoms and how these relate to overlapping conditions. In contrast, deep or hidden heterogeneity refers to the fact that individuals with the same diagnostic profile may have completely different developmental profiles and underlying problems. This issue is quite important, bearing in mind that the Diagnostic and Statistical Manual of Mental Disorders uses a very rigid, categorical classification system for diagnosis of developmental disorders like CD and ADHD.

1.2 Conduct Disorder

1.2.1 Diagnosis

Conduct Disorder (CD) is a disruptive behaviour disorder characterised by a persistent and pervasive pattern of aggressive and antisocial behaviour that violates the rights of others or in which major age-appropriate societal norms are violated (DSM-5; APA, 2013). Symptoms of CD fall into four broad categories: aggression to people and animals, destruction of property,

deceitfulness or theft, and serious violations of rules. For an individual to be diagnosed with CD, three out of fifteen criteria must be met, they have to cause clinically significant impairment and a differential diagnosis must be ruled out. As the categories of the symptoms are so broad, there is a lot of heterogeneity within CD: two individuals with CD may very well share no common symptom.

1.2.2 Subtyping

Early research in CD has indicated the heterogeneity of the disorder. Frick and Ellis (1999) argued that there is little consensus as to which is the best way to subtype CD children in order to tackle this issue. Research in CD has frequently been distinguished between two types of CD: the childhood- or early-onset (EO) type, where the onset of at least one symptom occurs prior to the age of 10, and the adolescent or late-onset (AO) type, where there is an absence of symptoms before the age of 10. This subtyping was influenced by Moffitt's developmental taxonomic theory (Moffitt, 1993). According to Moffitt, childhood-onset CD has a neurodevelopmental basis, while adolescent-onset CD reflects social imitation of peers. However, recent research has shown that both subtypes present neuroanatomical and neurophysiological abnormalities (Fairchild et al., 2011; Passamonti et al., 2010) and that adolescent-onset CD may also be a neurodevelopmental disorder (Fairchild et al., 2013, 2016).

A different approach towards understanding CD has been to define subtypes according to the presence or absence of psychopathic features, or callous-unemotional (CU) traits in CD youth. CU traits refer to a specific affective and interpersonal style that is characteristic of a subgroup of children with severe conduct problems (Frick et al., 2003). The behaviour of these children is characterised by a failure to show empathy, a relative lack of guilt and the use of others for their own gain. However, it should be highlighted that because of the issue of heterogeneity in CD samples, a more dimensional approach should be taken in terms of explaining the manifestation of the disorder.

The importance of CU traits in correctly subtyping CD is also reflected in the latest edition of the DSM (DSM-5; APA, 2013), in which the specifier of CD with a CU presentation ('with limited prosocial emotions') is included. To qualify for this specifier, a child must have displayed two of the following four symptoms over a period of 12 months: lack of remorse or guilt, lack of empathy, being unconcerned about performance, and shallow or deficient affect.

The introduction of this specifier finds support in studies that have shown the relative stability of CU traits across childhood (Frick & Dickens, 2006) and higher heritability estimates for CD with high CU traits versus low CU traits (Viding, Jones, Paul, Moffitt, & Plomin, 2008).

1.2.3 Prevalence

Green et al., (2005) reported that CD occurs in 7% of young people aged 11-15 years in the UK. In a large survey-based, retrospective study of adults, Merikangas et al. (2010) reported that the lifetime prevalence of CD in the USA was 9.5%. However, the prevalence of CD has been reported to vary between studies – range 2%-15% (Lahey, Miller, Gordon, & Riley, 1999). This is mainly due to the use of different terminologies and classification systems.

1.2.4 Cost

Farris, Nicholson, Borkowski, & Whitman (2011), report that antisocial behaviour is associated with financial and emotional burden to individuals, families, schools and communities. The Fast Track Project (Foster & Jones, 2005) followed more than 600 children at risk for behavioural problems over a period of seven years. The public cost of CD for that period was found to be more than $40000. The costs were calculated from youths' involvement with the juvenile justice system, child services and special education courses. It was shown that public costs were steadily increasing over the course of the seven-year period and that, on average, the annual public cost of CD was $6735. In the UK, Romeo, Knapp and Scott (2006) investigated the costs incurred by children aged 3-8 years old with antisocial behaviour. Findings revealed that children received help from multiple agencies including the NHS and public sector services and that the mean annual cost per child was approx. £6000. In addition, the results indicated that the greater cost burden was borne by the family.

1.2.5 Aetiology of CD

The internal mechanisms involved in the causal models of CD tend not to occur in isolation. Rather, they form a transactional model where deficits in social cognition, biopsychosocial and neuropsychological systems, impulse control, emotion regulation and parent-child attachment covary significantly. In addition, gene-environment (GxE) interactions play a huge role in this transactional model. Weeland, Overbeek, de Castro, and Matthys (2015) proposed a theoretical model of the underlying mechanisms of GxE interaction (see Figure 1.1). In this conceptual model, vulnerability traits can act as moderators between GxE

interaction and behavioural outcomes. The authors propose three mechanisms underlying GxE in externalising behaviours: serotonin-related emotional reactivity, dopamine-related reward sensitivity and serotonin-related punishment sensitivity. While the genotype is responsible for strengthening or weakening a behavioural outcome, these traits act as mediators between the interactions with the environment and the externalising behaviour (Weeland, Overbeek, de Castro & Matthys, 2015).

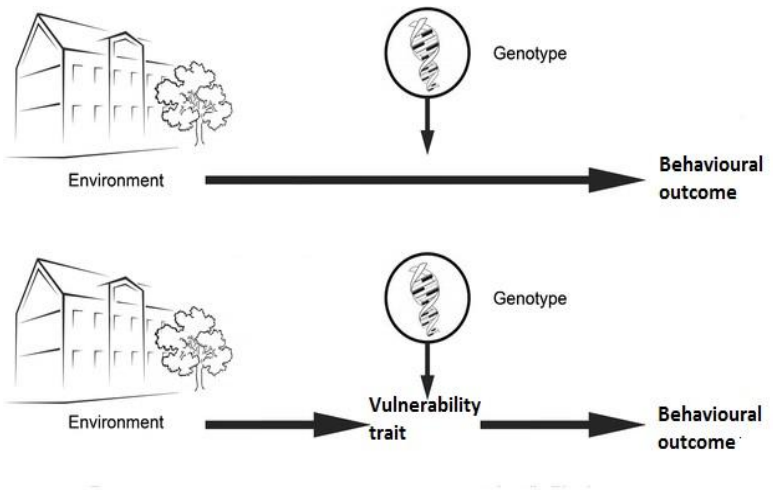

Figure 1.1. Underlying mechanisms of gene-environment interactions and externalising behaviour. The top line shows how the individual's genotype moderates the relationship between the environment and the outcome. The bottom line specifies the key mechanism via which this happens i.e. vulnerability trait. Adapted from "Underlying mechanisms of gene-environment interactions in externalising behaviour: a systematic review and search for theoretical mechanisms" by J. Weeland, G. Overbeek, B.O. de Castro and W. Matthys, 2015, Journal of Clinical Child and Family Psychology Review, 18(4), p.427. Copyright 2015, by Springer.

Based on the previous description, the behavioural expression of CD can also be explained by two main notions in developmental psychopathology. The two notions are equifinality and multifinality. Equifinality refers to a paradigm where many different developmental paths may lead to the same behavioural outcome. Multifinality suggests that the same risk factor can lead to a plethora of different outcomes (Beauchaine & McNulty, 2013).

1.3 Attention-Deficit/ Hyperactivity Disorder

1.3.1 Diagnosis

Attention-Deficit/Hyperactivity Disorder (ADHD) is the most common externalising disorder for which children and adolescents are referred to Child and Adolescent Mental Health Services (CAMHS; NICE, 2013). Until recently, ADHD was included under the category of Disruptive Behaviour Disorders (DBDs) together with CD and ODD (APA, 1994). In the latest version of the DSM (DSM-5; APA, 2013), ADHD is included under the neurodevelopmental disorders category, alongside intellectual disability (Intellectual Developmental Disorder), communication disorders, autism spectrum disorders, specific learning disorders, and motor disorders.

Even though the changes in the diagnostic criteria have been characterised as subtle (Epstein & Loren, 2013), they reflect the increased knowledge among clinicians and researchers about the nature of ADHD; they also provide a higher percentage of reliable diagnosis.
There are eighteen diagnostic symptoms, which form two dimensions: inattention and hyperactivity/impulsivity, and three subtypes: inattentive, hyperactive/impulsive, and combined. There are five (A-E) ADHD diagnostic criteria categories. The latest DSM revisions include changes to every diagnostic criterion. Criterion A which stipulates the diagnostic symptoms, remained unchanged with the exception of the reduction of symptoms in order for a diagnosis to be made in individuals older than 17: the individual needs to meet five, rather than six, symptoms in either dimension. This reflects the recognition of ADHD as a lifetime disorder and its associated clinical impairment (Mannuzza et al., 2011), despite a general drop in observable symptoms with increasing age. Criterion B changed from age of symptom onset before age 7 to symptom onset before age 12. This change reflects the evidence which show that there are no meaningful differences in functioning or outcomes in children who are symptomatic before age 7 and those who display symptoms at an older age (Barkley & Biederman, 1997). Criterion C simplified the role of pervasiveness by changing the sentence “evidence of impairment” to “evidence of symptoms in two or more settings”. This makes the symptoms easier to quantify as the use of the term “impairment” is more subjective and less measurable (Epstein & Loren, 2013). Criterion D substituted the need for “clinically significant” functional impairments with impairments that “reduce the quality of social, academic or occupational functioning”. Finally, Criterion E allows for a comorbid

diagnosis of ADHD and ASD. Again this change recognises consistent research findings that children with ASD can also have comorbid ADHD (Ronald et al., 2008; Simonoff et al., 2008).

1.3.2 Subtyping

In DSM-5 (APA, 2013), the three subtypes mentioned above are introduced as presentation specifiers. The specifiers reflect the individual's current clinical profile rather than a distinct subtype. This change is reinforced by studies showing the subtypes' instability over time (Lahey et al., 1999) and their limited contribution to the explanation of heterogeneity within ADHD samples.

1.3.3 Prevalence

Polanczyk, de Lima, Horta, Biederman, and Rohde (2007) completed the most comprehensive meta-analysis of the worldwide prevalence of ADHD. It was estimated to be 5.29%. One major difficulty that the authors faced was the changing diagnostic criteria of ADHD across the years. In a meta-analytic review by Willcutt (2012), 86 studies of children and adolescents and 11 studies of adults with ADHD were included. Acknowledging that the estimates of prevalence varied widely between studies, mainly because of the differences in the diagnostic criteria, Willcutt reported an estimated prevalence ranging from 5.9% to 7.1% for children and adolescents, and 5% for adults. In the most recent systematic review and meta-regression analysis by Polanczyk, Willcutt, Salum, Kieling, and Rohde (2014) it has been documented that there is no evidence to suggest an increase in the number of children with ADHD within community samples.

1.3.4 Cost

The cost of the management of child and adolescent ADHD in the UK is approximately £66 million per year (King et al., 2006). Respectively, the total cost of crime in England and Wales is currently about £75 billion a year, of which £22.5 billion is attributed to crimes committed by individuals with severe antisocial behaviour (Sainsbury Centre for Mental Health, 2009). Taking into account that the prevalence rate of ADHD in delinquent adolescents and young adults ranges between 4% to 72% (Vermeiren, 2003) it becomes apparent why the research in the overlap between CD and ADHD is beneficial not only to the individual, but to the society as a whole.

1.3.5 Aetiology of ADHD

The issue of the heterogeneity of ADHD at the behavioural level has been a contributing factor to the difficulties establishing its aetiology, and has also complicated the diagnosis and treatment of the disorder. The aetiology of ADHD is complex and it involves a genetic component and diverse neurobiological alterations (Banaschewski et al., 2017). Given this complexity, it has been argued that the manifestation of the disorder should be explained in more dimensional terms (Kraemer et al, 2004) in order for the full range of the severity of symptoms to be captured (Wahlstedt, Thorell, & Bohlin, 2009). This point is reflected in the diagnostic criteria changes mentioned above, i.e., with the introduction of current clinical profiles instead of subtypes.

1.4 CD and ADHD comorbidity

1.4.1 Diagnostic criteria

Neurodevelopmental disorders like ADHD are thought to be the result of structural changes in the brain, which have their roots in early developmental alterations and are associated with a variety of developmental delays (Frick & Nigg, 2012; Van Herwegen, Riby, & Farren, 2015). In comparison, CD is classified in DSM-5 as a behavioural disorder, which is determined by a range of biological and environmental factors and social conditions (Murrihy, Kidman, & Ollendick, 2010). DSM-5 (APA, 2013) now stipulates that ODD can be diagnosed independently of CD. This very important distinction will allow researchers to look for similarities and differences between the two disorders. This distinction is also supported by past research, where boys with ADHD+ODD were no more likely to develop criminal behaviour than boys with ODD alone (Lahey & Lober, 1997; Lilienfeld & Waldman, 1990). In addition, it may be that ADHD has been identified incorrectly as a possible precursor to CD due to its frequent comorbidity with ODD (Van Lier, van Der Ende, Koot, & Verhulst, 2007).

1.4.2 Prevalence

Biederman et al. (1991) reported that ADHD and CD co-occurred in 30% to 50% of all the cases reported in both epidemiological and clinical samples. Heritability estimates for ADHD and CD have been found to range between 0.27 and 0.78 (Slutske et al., 1997). Research has also shown that there is evidence for heritability of the comorbid condition ADHD+CD (Nadder et al., 2002; Anney et al, 2008). ODD is present in around 50% of children with

ADHD, with anger and hostility as the main symptoms, while CD is present in around 20% of children with ADHD and is linked to multiple forms of hostile, aggressive or delinquent behaviour (Loeber et al., 2009; Taurines et al., 2010). However, it should be noted that children with ADHD+CD have worse outcomes than children with ADHD+ODD, mainly due to the severity of the symptomatology and the pattern of offending behaviour (Mannuzza et al., 2004).

1.4.2 Genetics of CD and ADHD

The six most studied candidate genes in CD and ADHD are the following: monoamine oxidase A (MAOA), the dopamine receptors D4 (DRD4) and D2 (DRD2), the dopamine transporter 1 (DAT1), the 5' serotonin transporter linked polymorphic region (5-HTTLPR) and the catechol-O-methyltransferase (COMT) (Weeland, Overbeek, de Castro and Matthys, 2015). The high percentage of overlap between the two disorders indicates that there must be some degree of shared vulnerability between the two disorders (the issue of comorbidity will be discussed in depth in Chapter 2).

Beauchaine and McNulty (2013) argue that many studies (Durston 2013; Rubia et al., 2009; Shannon et al., 2009) show a consistent pattern of mesolimbic dopamine (DA) dysfunction as a neurobiological substrate of inherited vulnerability in externalising disorders, e.g., ADHD, CD and ODD. For a detailed account of the significant gene associations between CD and ADHD please see De Young et al. (2010).

Comings et al. (2000) studied the role of dopamine, serotonin and noradrenaline genes in ADHD, CD and ODD. The findings indicated that adrenergic genes played a greater role in ADHD expression than either dopaminergic or serotonergic genes combined. In addition, examination of the overlap between ADHD, CD and ODD, showed that the overall percentage of the variance in genes for ODD increased to a level approaching that for ADHD; the CD level remained low. The authors concluded that their findings support the notion that ODD shares genes with ADHD but utilises different genotypes for some genes while CD shares fewer genes with ADHD, and uses different genes rather than different genotypes of the same genes.

1.4.3 Comorbidity models and study designs

The first study to test a range of comorbidity hypotheses about the comorbidity between ADHD and CD was by Rhee et al. (2008). The authors used the model fitting approach of Neale and Kendler (1995). They summarised the different comorbidity models in the following categories: chance, alternate forms, six multiformity models, three independent disorders models, and four correlated liabilities models. Their sample consisted of 110 monozygotic twin pairs and 181 dizygotic twin pairs with data for both ADHD and CD diagnoses available. The multifactorial analysis indicated the correlated risk factors model as the model that best fit the data, suggesting that there were shared genetic and environmental influences between ADHD and CD. The three independent disorders model did not fit the data well.

In a behavioural genetic study by Nadder et al. (2002), a twin method was used to investigate the aetiological relationship between ADHD and CD. The results indicated that the ADHD+CD comorbidity was more likely to be attributed to a shared genetic liability, rather than environmental influences independent of ADHD. This overlap between the genetic influences of ADHD and CD supports a correlated risk factors model of comorbidity. On the other hand, family prevalence studies give their support to an independent disorders comorbidity model (Faraone et al., 1997). Furthermore, Banaschewski et al. (2003) investigated the impact of the psychopathological ADHD+CD comorbidity on brain electrical correlates. They used a cued continuous performance test in samples of children aged 8 to 14 years. Results indicated that children with the ADHD combined type suffered from a more general deficit and rejected the hypothesis that the comorbid condition represented an additive co-occurrence of the two disorders (correlated risk factors model). Instead, they suggested that the comorbid condition represented a separate condition, which differed from samples of children with ADHD or CD only.

Apart from ADHD and ODD, CD has been found to be comorbid with a number of disorders, including reading disorders (Maughan et al., 1996), anxiety (Shanahan et al., 2014) depression (Schepman et al., 2014), sleep problems (Shanahan et al., 2014) and substance use disorder (Sung et al, 2014). Additionally, research suggests that comorbid CD and anxiety have a complex etiological interaction. Specifically, when anxiety is developed alone in childhood, it is found to serve as a protective factor towards future antisocial behaviour, whereas children who develop CD first are at increased risk of developing anxiety (Loeber et al., 2000).

Even though our focus is not on ADHD per se, it is worth mentioning that ADHD has been found to be highly comorbid with a number of other disorders. Wilens et al. (2002) found a 50% comorbidity between ADHD and mood disorders in a clinical sample of school aged children with ADHD. In addition, they found a 33% comorbidity between ADHD and anxiety disorders. Tourette syndrome has also been found to be highly comorbid with ADHD. It has been reported that that an estimated 60-70% of children who were clinically diagnosed with Tourette syndrome also met the diagnostic criteria for ADHD (Swain et al., 2007). In addition, it is estimated that 20% to 40% of children with dyslexia also have comorbid ADHD (Williams and Lind, 2013; Tarver, Daley and Sayal, 2014).

1.5 Conclusion

The aim of this chapter was to introduce the reader to CD, ADHD and the notion of comorbidity. The issue of heterogeneity and its importance to psychopathology was also highlighted. It is clear that both disorders cover a wide range of symptoms that often overlap with each other. This makes the diagnostic process difficult and the selection of treatments challenging. Here, it should be noted that the psychopharmacological treatments currently used for children with both ADHD and CD have limited long-term effectiveness on CD outcomes (Langley et al., 2010; Molina et al., 2009). Understanding the nature of comorbidity and its causes has the potential to inform treatment planning and assist with the development of new interventions. Chapter 2 will focus on the neuropsychological deficits in CD and comorbid ADHD.

Chapter 2 Conduct disorder and comorbid attention-deficit/hyperactivity disorder –neuropsychological deficits

The aim of this chapter is to review the neuropsychological deficits in CD and CD+ADHD within the different cognitive domains of interest that this thesis will examine.

2.1 Neuropsychological deficits

Neuropsychological methods have been widely used to understand the cognitive deficits in CD and ADHD. Willcutt et al. (2005) argue that the only way to understand the neuropsychological correlates of comorbid conditions is to directly compare the pure groups and the comorbid group on the same measures. We can understand the importance of the proposed study design from this: if individuals with the comorbid condition show shared neuropsychological deficits that are common to both pure disorders, this may lead to a better understanding of the underlying processes that lead to the comorbid disorder. In contrast, if individuals with comorbid conditions show unique deficits relative to those with pure conditions, this may indicate that the comorbid condition is a separate disorder with a distinct aetiology. However, even though this is the best design to employ when comorbidity models are investigated, our thesis focuses on considering how comorbid ADHD confounds our understanding of the neuropsychology of CD. This has yet to be established.

2.2.1 Executive Functions

Executive functions (EF) incorporate meta-cognitive processes that enable efficient planning, execution verification and regulation of goal-directed behaviour, with the frontal cortex and its subcortical connections serving as its major neural correlates (Oosterlaan et al., 2005). Research has indicated that there are three core EFs: inhibition (or inhibitory control including self-control and interference control), working memory (WM) and cognitive flexibility (also called set shifting or mental flexibility) (Miyake et al., 2000). For a recent review on EFs, see Diamond (2013). Higher order EFs, such as Theory of Mind, problem solving and planning are built on the aforementioned core EFs (Lunt et al., 2012).

Deficits in EFs are documented in a number of disorders including CD (Fairchild et al., 2009), depression (Taylor-Tavares et al., 2007), obsessive compulsive disorder (Penades et al., 2007)

and schizophrenia (Barch, 2005). Historically, ADHD has been considered to be a disorder of executive dysfunction (Barkley, 1997), where the problematic behaviour is a result of a deficit in inhibitory control. However, as already mentioned in the previous section, research has shown that ADHD is a complex heterogeneous disorder and children with ADHD may present deficits in more than one cognitive domain (Solanto et al., 2001; Sonuga-Barke et al., 2003; Nigg et al., 2005). Studies have also shown that independent of ODD/CD, ADHD is associated with deficits in EF and vice-versa (Thorell & Wåhlstedt, 2006). However, even though EF deficits have been documented in CD, it is yet to be determined whether these deficits are related to antisocial behaviour or to comorbid conditions with similar deficits (Blair et al., 2006).

Previous research in EF deficits in CD has yielded mixed findings (Oosterlaan, Logan, & Sergeant, 1998; Morgan & Lilienfeld, 2000; Rubia, 2011). Performance deficits in EF tasks have been linked to high trait aggressiveness (Krämer, Kopyciok, Richter, Rodriguez-Fornells, & Münte, 2011), as well as to high levels of physical aggression (Barker et al., 2011). Studies have either grouped CD participants with children with externalising behaviour problems (Schoemaker, Mulder, Deković, & Matthys, 2013) or with children with other disruptive behaviour disorders (DBDs), such as ODD (Schoemaker et al., 2012). The results have indicated that children with DBDs have shown both response inhibition and WM deficits. However, some of the tasks used in these studies included reward–related EF tasks, which other researchers have considered to be measuring more specific inhibition deficits, i.e. "hot" EFs (Rubia, 2011). This variability in findings may be due to two factors. Firstly, DBDs are not a homogeneous category. For example, ODD is not characterised by delinquent behaviour. This means that when studies group together children with both milder and more severe forms of this spectrum of disorders, the sample becomes increasingly heterogeneous. Secondly, the role of ADHD comorbidity in CD samples is not always explicitly investigated and controlled for.

As the term EF is an umbrella term which includes many different cognitive purposes, theorists have proposed a distinction between "cool" and "hot" EFs. The former involve cognitive functions like attention, working memory, planning and inhibition, while the latter involve motivation control, emotion regulation, reward and punishment (Zelazo & Müller, 2002). Recent research asserts that children and adolescents with CD appear to have more difficulties in "hot" EFs and tasks that involve gambling paradigms or stimulus-response

contingency reversal tasks, while children and adolescents with ADHD have more difficulties in "cool" EFs and tasks that involve inhibitory control, interference control working memory and cognitive flexibility (Rubia, 2011). However, as discussed in the previous paragraph the findings have been mixed. The present thesis will focus on specific "cool" and "hot" EFs in order to examine whether CD is involved more in one type of EFs and less in the other.

2.2.1.1 "Cool" EFs

Interference Control

The general class of inhibition processes can refer to two related constructs: response inhibition (RI) and interference control (IC; Nigg, 2000). Response inhibition refers to one's ability to control their behaviour, emotions and attention so they can override a strong internal or external urge for immediate action, and instead perform the most appropriate action suited to the situation (Diamond, 2013). IC refers to an individual's ability to resolve response conflict by filtering out information that is irrelevant to optimal task performance (Wöstmann et al., 2013). Many studies have identified the necessity to treat RI and IC as different inhibition constructs rather than treating them as a single unified construct (Friedman & Miyake, 2004; Nigg, 2000). In this thesis we will investigate IC as a dissociable component of inhibition.

ADHD participants have been found to have impaired performance in tasks that measure inhibition, compared to controls (Sergeant, Geurts, & Oosterlaan, 2002; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). Six meta-analyses have investigated the effects of ADHD on IC (Frazier, Demaree, & Youngstrom, 2004; Hervey, Epstein, & Curry, 2004; Homack & Riccio, 2004; Lansbergen, Kenemans, & Van Engeland, 2007; Schwartz & Verhaeghen, 2008; Van Mourik, Oosterlaan, & Sergeant, 2005). However, the results have been mixed (Cohen's d range: 0.15-0.54). This variability in effect sizes (from weak to moderate) may be due to differences in quantifying interference scores, sample sizes, inclusion/exclusion criteria and controlling for other comorbid conditions.

By contrast, previous research employing an alternative IC paradigm that employs shape stimuli, the Eriksen-Flanker task, has typically found a larger interference effect in children with ADHD with longer RTs and more errors on incongruent trials, compared with controls, suggesting impaired IC (for a review, see Mullane, Corkum, Klein, & McLaughlin, 2009). Furthermore, Mullane et al. also highlighted that 9 out of the 12 studies in their review

provided little information regarding the impact of comorbid ODD/CD on the performance of IC tasks. Subsequently, the authors concluded that a key direction for future research should be to directly compare ADHD children with and without comorbid ODD/CD.

The majority of the studies included in the aforementioned meta-analyses used the Stroop (Colour-Word) task, during which participants were asked to identify the colour of a word, while inhibiting the automatic response to read the word. Even though the Stroop task is generally considered to be a reliable measure of IC (Van Mourik et al., 2009), it also relies heavily on other functions such as overall word reading ability and word naming efficiency (Tannock, Martinussen, & Frijters, 2000; Van Mourik et al., 2005). Given that children with ADHD show deficits in both of these abilities (Tannock et al., 2000; Van Mourik et al., 2005; Willcutt et al., 2005), the use of the Stroop task in ADHD samples may confound results. Indeed, a meta-analysis using the Stroop Task found little evidence for impaired IC in ADHD (Van Mourik et al., 2005; Willcutt et al., 2005).

Working Memory

WM refers to a complex, limited-capacity, cognitive system, which is used for the simultaneous short-term storage and processing of information (Baddeley, 1992; Smith & Jonides, 1999). This allows everyday information to be temporarily stored, rehearsed and manipulated so that we can use it in guiding our behaviour. This cognitive system consists of two main subsystems: the phonological and the visuospatial; and a cognitive attentional controller named the "central executive" which is responsible for their smooth coordination (Wager & Smith, 2003). For the purposes of this thesis we will focus on visuo-spatial WM (VSWM). Research has shown that WM task performance in normative samples of school children is associated with teacher-reported externalising behavioural problems (Aronen, Vuontela, Steenari, Salmi, & Carlson, 2005).

Results from studies that look at WM performance in adolescents with DBDs are mixed, with some showing deficits and others showing no differences (Van Goozen et al., 2004; Youngwirth, Harvey, Gates, Hashim, & Friedman-Weieneth, 2007). In a more recent study by Shoemaker et al. (2012) children with comorbid ADHD+DBD were not found to have significant performance differences in WM tasks, compared to controls. However, a study looking at EF deficits in young offenders found WM impairments (Syngelaki, Moore, Savage, Fairchild, & Van Goozen, 2009). This finding is very interesting as the overlap between

young offenders and those with DBDs is quite high: 90% of persistent juvenile offenders have been found to have had CD in childhood (Farrington, 2005). Conflicting findings in studies that investigate WM deficits in CD can be explained in terms of not taking into account comorbid conditions like ADHD, and using community-based samples with less severe symptomatology than clinical samples.

WM has been considered a central construct in theories of EF deficits in ADHD. While some previous research investigating WM deficits in ADHD has shown mixed findings (Martinussen & Tannock, 2006; Pennington & Ozonoff, 1996; Scheres et al., 2004; Sonuga-Barke, Dalen, Daley, & Remington, 2002), the literature has provided strong evidence of WM deficits in children with ADHD, particularly SWM (for a review see Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005). Failure to obtain more homogeneous results among the different studies may be due to two issues: a) WM is a complex construct with different components whose underlying processes are not clear yet; and b) the relationship between IC and WM has not been clearly defined. Brocki et al. (2008) report that that these constructs are distinct, yet related. However, there are few published studies looking at SWM task performance in ADHD (Karatekin, Bingham, & White, 2009; Strand et al., 2012) and even fewer in CD (Oosterlaan, Scheres, & Sergeant, 2005; Saarinen, Fontell, Vuontela, Carlson, & Aronen, 2015; Van Goozen et al., 2004).

One of the most common SWM experimental paradigms is the "N-back" task. In this task, participants are presented with a sequence of items that are either spatial or non-spatial in nature. They are requested to respond to items that are identical to the item 0, 1 or 2 trials back in the sequence. As the number of trials increases, so does the load placed on the participants in order to store and manipulate information in their WM. A common finding among studies is that clinical participants and controls show worse performance scores as the n number of trials increases, i.e. 2-back rather than 1-back (Saarinen et al., 2015).

Two points should be made regarding the inconsistencies in the findings of WM tasks. The first is that heterogeneous samples show more variability in their deficit patterns and may indicate a bigger overlap in symptoms because of the initial heterogeneity. The second is the assessment of a WM deficit at different developmental levels. WM ability has been found to vary across different ages, and research has also indicated different developmental trajectories between spatial and non-spatial tasks (Van Leijenhorst, Crone, & Van der Molen,

2007). As Schleepen and Jonkam (2009) noted, apparent WM impairments may simply reflect a developmental delay in a particular group. Therefore, researchers should be careful not to attribute WM deficits to a possible immaturity of the executive control processes, as individuals with these delays may ultimately catch up with individuals in their age cohort.

2.2.1.2 "Hot" EFs

Identifying the presence of comorbid ADHD is very important as children with CD and children with ADHD are hypothesised to also exhibit different psychophysiological responses to reinforcement. This is illustrated by the theoretical framework of Gray (1982; 1985) in which three interrelated brain systems are responsible for behaviour. The first one is named the Fight/Flight System (FFS) and is activated by unconditioned aversive stimuli. These stimuli elicit unconditioned defensive aggression and escape behaviour. The second system is known as the Behavioural Activation System (BAS). This system is activated by cues of reward and non-punishment, and initiates behavioural approach. In the third system, known as the Behavioural Inhibition System (BIS), ongoing behaviour is inhibited in response to cues for punishment and non-reward, and passive avoidance is initiated. Gray highlighted that the BIS and BAS are two independent systems that operate in an antagonistic fashion: activation of one inhibits the activation of the other.

Based on Gray's theoretical framework, Quay (1993, 1997) hypothesised that these two systems contributed differentially to the development of ADHD and CD. An underactive BIS is responsible for ADHD, whereas a dominant BAS over BIS is responsible for CD. Specifically, when cues for reward and punishment are simultaneously present, both systems are activated, but children with CD are over-responsive to cues of reward. In contrast, when presented with punishment cues, children with ADHD are less responsive to these cues and signals of non-reward. To explain the comorbid condition, ADHD+CD, Quay suggested that the BIS was less able to inhibit the ongoing BAS activity than in CD alone, because in the comorbid condition not only was the BAS dominant over BIS, but the BIS was also persistently underactive. In other words – they are both hypersensitive to reward and hyposensitive to punishment. This is an additive model in which individuals with CD+ADHD have a combination of the deficits observed in each of the pure conditions.

Delay Aversion & Delay-related Frustration

Douglas and Parry (1983) were amongst the first to report reward sensitivity in hyperactive children. Indeed, several theoretical paradigms have linked ADHD to an atypical sensitivity to reward, punishment and reinforcement contingencies (Sagvolden & Sergeant, 1998; Sergeant, 2000; Sonuga-Barke, 2002). The dual pathway model of ADHD (Sonuga-Barke, 2002) explained the neuropsychological heterogeneity within the disorder in terms of dissociable cognitive and motivational deficits, each affecting some, but not other, individuals. The motivational deficit was associated with an aversion to delayed reinforcement and it was linked to alterations in reward mechanisms. In the Delay Aversion model (DA; Sonuga-Barke, 2003), individuals with ADHD find delays, and cues which signal delay, aversive. As a consequence, the imposition of fixed delays leads to frustration, and individuals attempt to modify the waiting period in order to reduce the aversiveness of the delay. So, when given the opportunity to make a choice and avoid a delay, ADHD individuals will have a preference for smaller sooner (SS) over larger later (LL) rewards (Solanto, Abikoff, Sonuga-Barke et al., 2001). The impact of reinforcement contingencies on ADHD have also been reviewed by Luman, Oosterlaan and Sergeant (2005). While the review includes 22 studies and a total of 1181 children, findings are rather inconclusive as to why children with ADHD prefer immediate rewards and seem less sensitive to reinforcement. The key element of this review is that out of the 22 studies only two of them accounted for the comorbidity between ADHD, ODD and CD, by including a separate group of children with ODD/CD alone (Oosterlaan & Sergeant, 1998; Scheres et al, 2001). All of the remaining studies did not take this comorbidity into account and listed it as a possible confounding variable.

2.2.2 Emotion recognition

In CD, emotion processing deficits have been at the forefront of research studies that investigate the developmental pathways of antisocial behaviour. The most prominent theoretical model is that developed by Blair (1995, 2001), namely the Violence Inhibition Mechanism model (VIM). Blair suggested that the development of antisocial, aggressive behaviour stems from a deficient facial affect processing system. Specifically, children and adolescents with antisocial behaviour have difficulties recognising social cues of distress i.e. facial expressions of fear and sadness. There is a plethora of studies which indicate that children and adolescents with CD, but also young offenders, exhibit deficits in facial emotion

recognition tasks (Bowen, Morgan, Moore, & van Goozen, 2014; Fairchild et al., 2009; Fairchild, Stobbe, van Goozen, Calder, & Goodyer, 2010). In specific, research has indicated that children and adolescents with CD have a more generalised facial emotion recognition deficit which also includes facial expressions of happiness and surprise (Short, Sonuga-Barke, Adams, & Fairchild, 2016; Sully, Sonuga-Barke, & Fairchild, 2015a). In ADHD, impaired emotion recognition is associated with behavioural problems already in children who attend preschool (Chronaki et al., 2015). In addition, deficits in facial emotion recognition in children and adolescents with ADHD have been found to be consistent among studies (Aspan et al., 2014; Boakes et al., 2008). However, the issue with the aforementioned studies is that they consistently overlook the impact of comorbidity on their findings either by excluding the participants with the comorbid diagnosis from the statistical analysis or by simply acknowledging as a contributing factor without establishing the magnitude of impact.

2.2.3 Theory of Mind – Perspective taking

Theory of Mind (ToM), or mentalising as it is often referred to, is the ability to make inferences about the mental states of others such as intentions and beliefs. This ability lies within the domain of social cognition. Social cognition involves the following functions: encoding of social cues, perception of emotions, ToM, empathy and humour processing (Uekermann et al., 2010). ToM starts to develop quite early in childhood. Infants as young as 18 months begin to master how to direct their parent's attention to an object. In addition, by the age of four children can successfully complete false belief tasks, which involve understanding that others' mental representations may differ from your own and that they may be false (Wimmer & Perner, 1983). ToM is considered to have two levels: level 1 refers to visual perspective taking and children around two years old start to develop that ability, i.e. to understand which objects can or cannot be seen by someone with a different perspective, and level 2 refers to perspective taking which requires the understanding that people may have different viewpoints on the same object based on different visual perceptions (Dumontheil, Kuster, Apperly and Blakemore, 2010).

ToM deficits have been a major focus of research in Autistic Spectrum Disorders (ASD). However, far fewer studies have tested for the presence of social cognition deficits in children with CD and ADHD. Nijmeijer et al (2008) reported a social dysfunction in ADHD but concluded that the deficits could be explained by comorbid Pervasive Developmental Disorders. Studies of ToM in ADHD have yielded conflicting findings with some reporting

intact ToM task performance (Dyck et al., 2001), while others have shown that children with ADHD fail false belief tasks (Buitelaar et al., 1999). Overall there is little evidence that CD is associated with ToM deficits (Dunno, Parker, Gilmour, & Skuse, 2010; Ha, Sharp, & Goodyer, 2011), but more studies are needed using more age-appropriate and sensitive tasks in order to fully investigate the presence of ToM deficits in CD.

2.2 Gaps in the Literature

The high rate of co-occurrence between CD and ADHD highlights the importance of understanding the impact of comorbidity. In particular, it is important to characterise the impact that ADHD has on CD, as this has implications for clinical diagnosis and treatment options (Bakker, Greven, Buitelaar & Glennon, 2017). However, this overlap also complicates the study of the neuropsychology of CD as the overlapping ADHD symptoms may mask the true relationship between CD and neuropsychological performance.

From a neuropsychological perspective, CD has been associated with deficits in emotion processing and in "hot" executive functions with motivation elements, which are mediated by ventromedial and orbitofrontal limbic neural networks (Rubia, 2011). ADHD has also been associated with deficits in both "hot" and "cool" processes, i.e. working memory, sustained attention and inhibition, which are mediated by frontostriatal and frontoparietal neural networks. In addition, several theoretical paradigms have linked ADHD to an atypical sensitivity to reward, punishment and reinforcement contingencies (Sagvolden & Sergeant, 1998; Sergeant, 2000; Sonuga-Barke, 2002). Research has associated this motivational deficit in ADHD with an aversion to delayed reinforcement (Sonuga-Barke, 2003). However, this theoretical paradigm has not yet been investigated in CD.

In the light of this potential overlap, a review of the previous literature highlights two methodological limitations in studies of CD neuropsychology. First, many studies have looked at DBDs as a whole rather than assessing the impact of CD separately – combining those with a full diagnosis of CD with those with the milder diagnosis of ODD – but have used this evidence base to draw their conclusions about CD specifically (Shoemaker et al., 2012; 2013). Second, many studies have not controlled for ADHD when studying CD or have controlled for it through the post-hoc use of statistical methods rather than as an explicit part of the study design and participant screening process. The majority of the studies looking at neurocognitive deficits in CD have not accounted for the comorbidity with ADHD

(Pennington and Ozonoff, 1996; Morgan and Lilienfeld, 2000; Toupin, Dery et al., 2000). This has led to many inconsistencies among the findings of different studies and the reported deficits (Hummer et al., 2011; Barnett et al., 2009). This problem may be exacerbated by the presence of undiagnosed ADHD in CD populations (Sergeant et al., 2002).

In our attempt to address the aforementioned limitations, we originally designed a study that involved both neuropsychological and psychophysiological (EEG) tasks. This meant that the same individuals would have to attend two separate testing sessions on two different days. Furthermore, the original study design included the recruitment of a "pure" ADHD group. A total of 140 individuals were approached and screened (please see chapter 3 for details on sample recruitment). Identifying "pure" cases of ADHD proved to be quite challenging. The recruitment pace for that group was very slow i.e. 10 eligible cases in 12 months. Due to time constraints, a decision was made to drop the "pure" ADHD group and focus on the other two clinical groups i.e. CD and CD+ADHD. Out of the 81 participants that ended up taking part in the study, 69 also completed the psychophysiological testing session. However, delays in EEG analysis made it impossible to include any meaningful findings within the specific timeline. Therefore, the present piece of research focused on the impact of ADHD comorbidity on the neuropsychological phenotype of CD, which is often overlooked even though there is a clear link between the two disorders.

2.3 Thesis Aims and Outline

The main aim of this thesis is to examine the impact of comorbid ADHD on the neuropsychology of CD. It will attempt to characterise the clinical and neuropsychological profile of adolescents with CD alone (CD-ADHD) and comorbid ADHD (CD+ADHD) as compared to a group of typically-developing controls (TDC).

Given the heterogeneity and high levels of comorbidity between CD and ADHD (see Chapters 1and 2), the first objective was to characterise the clinical and personality profiles of individuals with CD-ADHD and CD+ADHD (Chapter 3). Following this, I examined the effects of CD and ADHD on the performance of selected behavioural tasks in the following neuropsychological domains of interest: "cool" EFs (Chapter 4), "hot" EFs (Chapter 5), emotion processing (Chapter 6) and theory of mind-perspective taking (Chapter 7).

In Chapter 8, I will present a summary and discussion of the findings, how they extend existing literature and their implications for future research.

Chapter 3 General Methods and Sample Characteristics

3.1 Introduction

The goal of this thesis is to test in what way the presence of comorbid ADHD influences the neuropsychological profile associated with CD. In Chapter 2 we presented the neuropsychological domains that this thesis will investigate and the rationale for their inclusion. This chapter will describe the clinical assessments, questionnaire measures and recruitment and testing procedures used in the study. In addition, the demographic, clinical and personality characteristics of the sample will also be described, analysed and discussed. The neuropsychological testing battery will also be mentioned briefly. However, detailed information about each of the behavioural tasks will be presented in the corresponding empirical chapters that follow.

3.2 Participants

Eighty-one male adolescents aged between 11-18 years old, participated in the study. Participants were allocated to one of three groups: typically developing controls (TDC), adolescents with Conduct Disorder alone (CD; CD-ADHD) and adolescents with comorbid CD and Attention-Deficit/ Hyperactivity Disorder (ADHD; CD+ADHD). The TDC group was recruited from mainstream schools and colleges in the Hampshire area. Adolescents in the two clinical groups were recruited from Youth Offending Services (YOSs) and Pupil Referral Units (PRUs) across Hampshire.

The two main disorders, CD and ADHD, and other mental health disorders were assessed using the Kiddie Schedule for Affective Disorders and Schizophrenia-Present and Lifetime version (K-SADS-PL; Kaufman et al., 1997). In total, fifty-one adolescents with CD were recruited – twenty-three in the CD-ADHD group and twenty-eight in the CD+ADHD group. The TDC group was comprised of thirty adolescents. One participant in the TDC group met the diagnostic criteria for General Anxiety Disorder (GAD), two participants in the CD+ADHD group also met the diagnostic criteria for GAD and one participant had a high number of Major Depressive Disorder (MDD) symptoms, but not enough to reach a full diagnosis. Participants who were diagnosed with ADHD were also classified according to the subtype of ADHD, i.e. ADHD-Inattentive (ADHD-I), ADHD-Hyperactive/Impulsive (ADHD-H/I) or ADHD-Combined type (ADHD-C). In addition, a further thirty-one adolescents who expressed an interest to participate in the study were not included due to

eligibility issues (please see below). All potential participants were initially approached either through an invitation letter (schools, colleges, Pupil Referral Units) or via flyers handed to them by their caseworkers (Youth Offending Services). Once interest in the study was expressed, either by a reply to the invitation letter or a referral by a caseworker, the researcher contacted the participant and an introductory meeting was scheduled. No participants were taking psychotropic medication at the time of testing and all participants were native English speakers. In addition, participants who were receiving pharmacological treatment for their ADHD symptoms stopped taking their stimulant medication for at least 24 hours before the testing session took place (two participants in the CD+ADHD group).

3.2.1 Exclusion Criteria

Participants were excluded from the study if they had: i) an IQ estimate of <75 (assessed using the vocabulary and matrix reasoning subtests of the Wechsler Abbreviated Scale of Intelligence – WASI; Wechsler, 1999), and ii) a psychiatric condition i.e. a psychotic disorder or pervasive developmental disorder which was either disclosed in the initial interview or identified using the Autism Quotient questionnaire (AQ; Baron-Cohen, Wheelwright, Skinner, Martin & Clubley, 2001).

3.3 Diagnostic Assessment

3.3.1 Kiddie Schedule for Affective Disorders and Schizophrenia-Present and Lifetime version interview (K-SADS-PL)

The K-SADS-PL (Kaufman et al., 1997) is a semi-structured, diagnostic interview designed to assess for current and past DSM-IV disorders (American Psychiatric Association, 1994) in children and adolescents. In this thesis, we used the K-SADS-PL to assess symptoms of the following disorders: CD, ODD, ADHD, GAD, Obsessive Compulsive Disorder (OCD), MDD, Alcohol and Substance Abuse, and Post-Traumatic Stress Disorder (PTSD). In cases where the adolescent was found to meet the criteria for either CD or ADHD but had not received a formal clinical diagnosis from a mental health professional, a diagnosis was not provided to the family by the researchers. If the participants were found to meet the diagnostic criteria for depression, anxiety or any other psychiatric disorder, it was suggested that they should seek help from their GP (please see Appendix for the full interview schedule and precise wording).

The participant and their parent/carer (whenever available) were interviewed separately. For confidentiality reasons, the interviews were administered in separate rooms by trained researchers. The interview consisted of two parts. The first part covered screening questions related to the following disorders: MDD, GAD, OCD, PTSD, alcohol and substance use disorders, and ODD. The screening questions map onto the cardinal symptoms of each disorder, such as low mood or anhedonia in major depressive disorder. The second part covered the supplementary symptoms of the disorders and was used in cases where the primary symptom criteria were endorsed during the screening interview. The K-SADS-PL has been found to allow a reliable and valid dimensional assessment of externalising symptoms in children and adolescents (Jans et al., 2009). On the scoring sheet, symptoms were assessed on a 0-3 scale: 0 = no information, 1 = not present, 2 = subthreshold and 3 = definitely present. For a symptom to be "definitely present", it required a rating of 3 by either the adolescent or the parent/carer. Even though the "or" rule, i.e. a symptom is counted as definitely present if reported by either informant, has been found to produce higher rates of diagnoses (Costello, 2015), it has also been found to produce reasonable validity of diagnoses (Angold et al., 2012).

3.4 Demographic Measures

3.4.1 Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999)

The two-subtest version of the WASI (Wechsler, 1999) was used to obtain an estimate of the participants' full scale IQ. This specific version consists of the vocabulary and the matrix-reasoning subtests that measure Verbal and Performance IQ, respectively. Participants were requested to explain the meaning of words that were read aloud by the researcher and shown in a booklet, and they were also asked to find the missing piece of a matrix by choosing one out of five possible options. To obtain the estimate of the participant's full-scale IQ, the summed T-scores of both subtests were converted into IQ scores, using age-normed data.

3.4.2 Neighbourhood Environmental Scale (NES; Crum et al., 1996)

The NES (Crum et al., 1996) is a self-reported measure and consists of 18 true/false items relating to neighbourhood poverty, safety and antisocial behaviour in the participant's community. It serves as a proxy measure of socio-economic status. True items count as one while false items count as zero. The higher the total score, the higher the levels of

neighbourhood disadvantage. As previously mentioned, the NES was used as a proxy measure for socio-economic status (SES). In the current sample, the NES' internal consistency was good ($\alpha = 0.81$).

3.5 Clinical Measures

In order to investigate the clinical profile of our population, we collected a range of clinical measures. We focused particularly on the influence of Callous-Unemotional (CU) traits on the performance of the neuropsychological tasks as previous research (please see Chapter 1) has highlighted the relationship between CU traits and externalising disorders.

3.5.1 Autism-Spectrum Quotient (AQ; Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001)

The AQ (Baron-Cohen et al., 2001) was used to assess the presence of autistic-spectrum traits in our sample. The AQ is a 50-item self-report questionnaire with sentences covering social skills, communication, imagination and attention to detail. The participant is required to rate each item on a scale of 1 to 4, with 1 being "definitely agree" and 4 "definitely disagree". The rating is then converted to a score of 0 = trait absent or 1 = trait present. The total score can range from 0 to 50; the higher the score, the higher the level of autistic-spectrum traits. Scores of ≥ 32 indicate clinically significant levels of autistic traits. The internal consistency of the AQ in the present sample was excellent ($\alpha = 0.95$). None of the participants in the study scored 32 or more, therefore no one was excluded from the study.

3.5.2 Youth Psychopathic traits Inventory (YPI; **Andershed, Kerr, Stattin, and Levander, 2002)**

The YPI (Andershed et al., 2002) is a 50-item self-report questionnaire that assesses the presence of psychopathic traits. The items are phrased as statements (e.g., "To feel guilt and regret when you have done something wrong is a waste of time", "Pretty often I act charming and nice, even with people I don't like, in order to get what I want"). Participants are asked to rate each item using a four-point scale (1 = does not apply at all; 4 = applies very well), with higher scores reflecting higher levels of psychopathic traits. Within the present sample, internal consistency for the total scale was found to be good ($\alpha = 0.89$).

3.5.3 Inventory of Callous-Unemotional Traits (ICU; **Frick, 2004)**

The ICU (Frick, 2004) is a 24-item self-report questionnaire that assesses the presence of callous-unemotional (CU) traits. It was devised based on the Antisocial Process Screening Device (APSD; Frick & Hare, 2001). The ICU consists of three subscales that measure callousness, uncaring and unemotional traits. Participants are asked to rate each item in terms of how well it describes them (0= not at all true; 3= definitely true). Total ICU scores range from 0 to 72, with higher scores reflecting higher levels of CU traits. The internal consistency of the total scale in the present sample was found to be good ($\alpha = 0.84$).

3.5.4 State-Trait-Anxiety Inventory (STAI; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983)

The STAI (Spielberger et al., 1983) is a 40-item questionnaire that measure levels of state (situation-centred) and trait (stable, personality-centred) anxiety. It is comprised of two separate scales; state and trait, which each contain 20 items. Participants are asked to rate the items after thinking about how they feel right now (state) and how they feel in general (trait). Items are rated on a four-point scale (1= not at all; 4= very much so); higher scores indicate higher levels of anxiety. The internal consistencies of both scales in the present sample were excellent (State $\alpha = 0.91$; Trait $\alpha = 0.90$).

3.6 Personality Measures

We used two measures of personality characteristics in order to investigate the personality profile of our sample. We specifically focused on impulsivity given the direct connection with ADHD.

3.6.1 Barratt Impulsiveness Scale-11 (BIS-11; Patton, Stanford, & Barratt, 1995)

The BIS-11 (Patton et al., 1995) is a 30-item self-report scale that assesses the construct of impulsivity. It consists of three subscales: attentional impulsiveness (e.g. “I concentrate easily” – reverse-scored), motor impulsiveness (e.g. “I act on the spur of the moment”), and non-planning impulsiveness (e.g. “I save regularly” – reverse-scored). Participants are asked to rate each item using a four-point scale (1= never, 2= sometimes, 3=often, 4= always). The internal consistency of the total scale in the present sample was good ($\alpha = 0.87$).

3.6.2 The Behavioural Inhibition/Activation Scales (BIS/BAS; Carver & White, 1994)

The BIS/BAS scales are based on Gray's (1987) theoretical model about two motivational brain systems: behavioural inhibition (BIS) and behavioural activation (BAS). The BIS/BAS

scales provide an index of the sensitivity of these two systems in a given individual – i.e. the sensitivity of the reward and punishment processing systems in the brain. The scales contain 24 self-report items which are rated on a four-point scale (1 = very true; 4 = very false). Higher BIS and/or BAS scores indicate a higher predisposition towards avoidance or approach behaviours, respectively. Internal consistency was found to be acceptable for the BIS scale (α =0.77) and good for the BAS scale (α= 0.84).

3.7 Neuropsychological Test Battery

3.7.1 "Cool" Executive Functions

3.7.1.1 The Eriksen-Flanker task - Interference Control (Broyd, Helps, & Sonuga-Barke, 2011)

Flanker tasks measure the ability to inhibit responses to irrelevant, interfering stimuli (response conflict). For the purposes of this thesis, we used an Eriksen-Flanker task to assess interference control. Participants were given a response box and were presented with a horizontal array of 5 stimuli consisting of left- and right-pointing arrows and equals signs. They were instructed to focus on and respond only to the central or middle stimulus in each array. The central stimulus was always an arrow. If the central arrow was left-pointing, participants had to press the left button. If the central arrow was right-pointing, they had to press the right button. The interference emerged when all the other arrows in the array were positioned congruent or incongruent with the central arrow. Participants were asked to respond as quickly and as accurately as possible.

3.7.1.2 The N-Back Task (Shackman et al., 2006)

The N-Back task assessed Spatial Working Memory (SWM) capacity. Participants were presented with a sequence of stimuli (letters) and were asked to remember the position of a square that appeared on the screen. The square was transparent, meaning that the letters in the background were visible. Both the letters in the background and the position of the square kept changing. Participants were asked to press a button on a response box when the position of the square matched the one that appeared N trials earlier.

3.7.2 "Hot" Executive Functions

3.7.2.1 The Maudsley Index of Delay Aversion Task – MIDA (Kuntsi et al., 2001)

The MIDA is a computer-based choice delay task that measures preferences towards smaller/sooner (SS) over larger/later (LL) rewards. This task was presented as a space game, in which the participants had to shoot down enemy spaceships to win points. If they opted for the SS choice, they would receive 10 p after a 2 sec delay, whereas if they selected the LL choice, they would receive 20 p after a 30 sec delay.

3.7.2.2 The Delay Frustration Task (DeFT) (Bitsakou, Antrop, Wieserma, & Sonuga-Barke, 2006)

The DeFT measures frustration caused by the unexpected imposition of delay in a response sequence and involves a series of simple maths questions presented on a computer screen. Each question was presented separately and was accompanied by four possible solutions. Participants were required to select the correct answer by pressing a button on a response box. On most trials as soon as the participant pressed the button, the program moved onto the next trial. However, on a minority of the trials access to the next maths question was unexpectedly interrupted and delayed by 20sec. In addition, 8 distractor trials were included, where the delay period varied from 3 to 10 seconds. On the experimental and distractor trials the response button was 'inactive' during the delay period and any responses made by the participant were therefore ineffective at accessing the subsequent trial. At the end of the delay period the response box was 'reactivated' and the first response became effective in allowing the participant to move on to the next trial.

3.7.3 Emotion Processing

3.7.3.1 The Emotion-Hexagon Task (Calder et al., 1996)

The Emotion-Hexagon task measures accuracy of facial emotional expression recognition. Participants were asked to label morphed facial expression continua spanning the following six expression pairs: happiness-surprise, surprise-fear, fear-sadness, sadness-disgust, disgust-anger, and anger-happiness. The photographs of the faces were in black and white. Participants were asked to name the correct expression by selecting the corresponding emotion label, i.e. happiness, surprise, fear, sadness, disgust, and anger.

3.7.4 Perspective Taking-Theory of Mind

3.7.4.1 The Director's Task (Dumontheil. Apperly, & Blakemore, 2010)

The Director's Task measures the ability to understand a situation from another person's perspective. Participants viewed a 4x4 grid which contained various objects in different grey slots and they were instructed by a "director" to move certain objects around the grid. Certain slots in the grid were occluded, thus the director could see some, but not all, of the objects that were visible to the participant. The instructions required the participant to use the information about the director's perspective to interpret the task instructions. In this thesis we also used a control No-Director condition, in which participants were instructed to ignore objects in the grey slots. The critical difference between the two conditions was that, in the Director condition, participants were instructed to take into account which objects the Director could and could not see (i.e., another's perspective), whereas in the No-Director condition, participants simply had to take account of the colour of the slot the object was in.

3.8 Procedure and testing session order

The initial meeting took place either at participants' homes or at the University of Southampton. During this first session the participants and, where available, their parents/carers were informed about the aims of the study and what participation in the study involved. Both the adolescents and their parents/carers were informed orally and in writing (see Appendix). During this initial meeting and throughout the course of the study, participants and their parents/carers were reminded that they were: a) under no obligation to take part in the study, and b) free to withdraw their consent at any time.

If the adolescents agreed to take part in the study, they were asked to give written informed consent. If the young person was aged below 16, he was asked to give his assent while his parent/carer gave their consent. If the young person was aged 16+ he was able to give consent by signing the form himself. In the latter case, the capacity to consent in participants aged 16+ was assessed in accordance to the guidelines set by the General Medical Council and the British Psychological Society. In accordance with those guidelines, the researchers kept in mind that "at 16 a young person can be presumed to have the capacity to consent" and we made sure that all relevant information about the study and the testing session was provided and thoroughly discussed before consent was taken. Only after we made sure that the participants had understood, and considered the information that was given, did we assume

that the young person had the capacity to consent. In situations where the capacity to consent may have been compromised due to the use of substances, e.g. drugs, alcohol, we were obliged to explain to the young person that we were unable to proceed either with the initial screening interview or with the testing session. For this reason we placed a strong emphasis on the need for sobriety when booking the appointments. On two occasions, we had to ask the participant to reschedule their testing session as they appeared to be intoxicated when they arrived at the University.

When the inclusion criteria were met, the participant was invited to take part in the neuropsychological testing session at the University of Southampton. On the agreed date, the participant arrived at the university and was met by the researcher. Figure 1 shows the testing order of the neuropsychological tasks that were administered and the questionnaires that were completed. The 2-Back task was the most cognitively demanding task in the neuropsychological battery, so it was decided that we would administer this task first in the overall battery regardless of the testing order they were allocated to. Participants were given an ID number and depending whether it was an odd or even number, they followed the corresponding testing order (Figure 1). The questionnaires were completed in two blocks: a) 1-4: STAI-T, STAI-S, ICU, BIS-11 and b) 5-8: YPI, AQ, NES, BIS/BAS (Please note: all the materials used in the study can be found in Appendix A). The testing session lasted 2-2.5 hours, depending on the number of breaks that the participant took. The computerised tasks in this study were programmed using either Presentation (Neurobehavioural systems, Inc., San Francisco, CA) or E-Prime, Version 2.0 (www.pstnet.com/eprime.cfm) and were displayed on a 17" CRT monitor. Behavioural responses were registered on a Cedrus response pad (Cedrus Corporation, San Pedro, CA). After the end of the session, participants were debriefed orally and were also asked to sign a Debrief Statement. They received their own copy of this document. They were also reimbursed for their participation in the study.

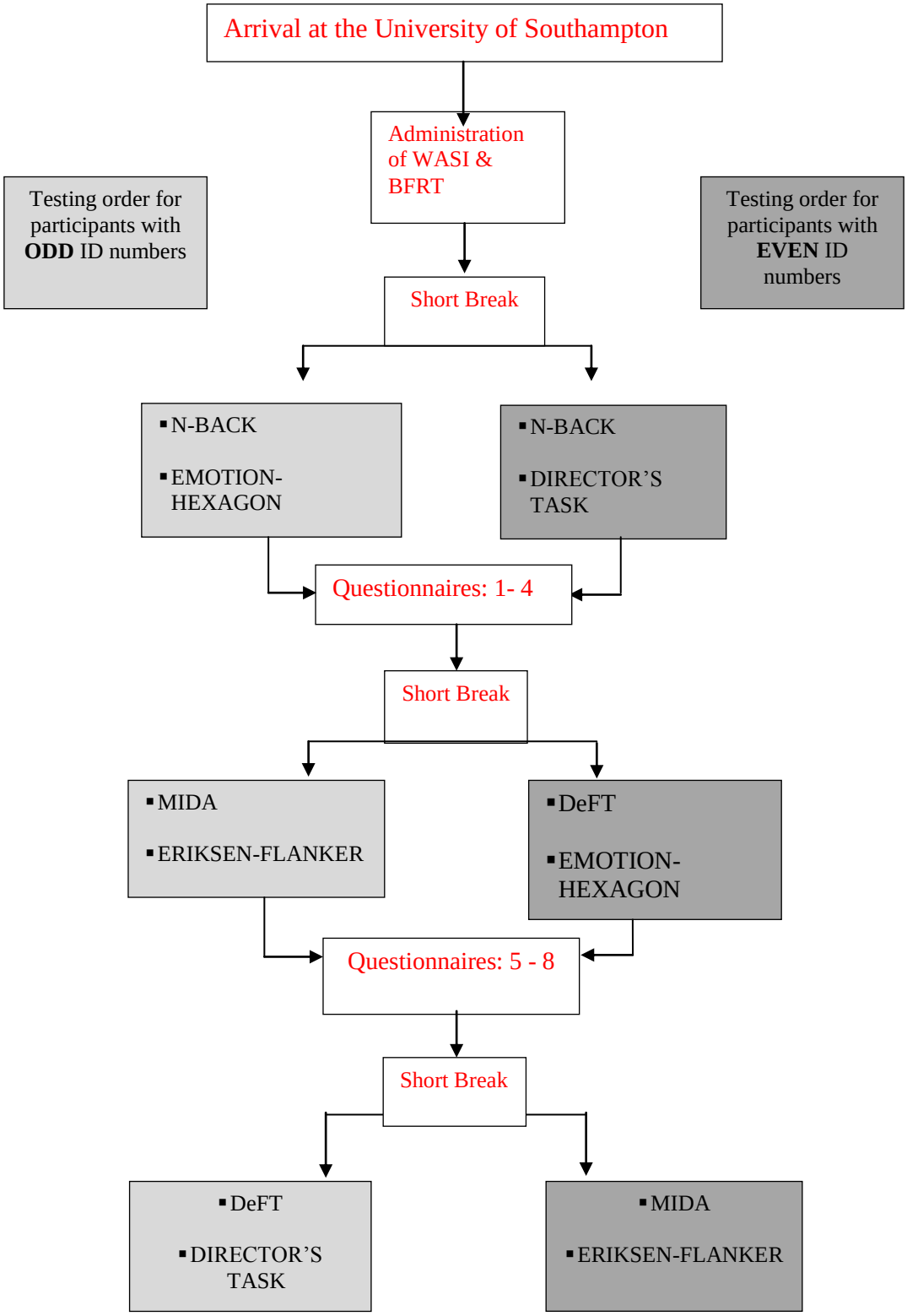

Figure 3.1. Testing order of the neuropsychological tasks and self-reported questionnaires. The left side demonstrates the order for the participants with odd numbered subject IDs, while the right side shows that of the participants with even numbered subject IDs.

3.9 Ethics

This study was approved by the University of Southampton Ethics Committee and Research Governance Office (ERGO ID: 9747), Southampton City Council, Hampshire County Council and the NHS National Research Ethics Committee – Hampshire B (ref no: 14/SC/0020). It should be noted that there were several occasions where a parent/carer was not available for the initial interview. On these occasions we still conducted the interview with the young person, whilst making sure that the informed consent process was thoroughly followed. In addition, for security reasons, two researchers always attended the screening interview, even if we knew that there was no parent/carer available. Participants, and when available their parent/carer, were reimbursed for the initial interview and thanked for their time even if they did not meet the inclusion criteria.

3.10 Power and Data Analysis

Power calculations conducted in G* power 3.1.9 (Faul, Erdfelder, Lang & Buchner, 2007), indicated that a total sample size of 60 would be required to detect significant main effects and interactions. The calculations were based on a medium effect size, an alpha level of 0.05, and a power of 0.80.

In order to explore the profile of our sample and assess the differences between the three groups, we conducted separate one-way analysis of variance (ANOVA) for each of the demographic, clinical and personality measures. Group was the dependent variable with three levels (TDC, CD-ADHD, CD+ADHD). Post-hoc Bonferroni tests were conducted where significant main differences were found.

3.11 Results

3.11.1 Group comparisons

3.11.1.1 Demographic characteristics

The demographic characteristics of the sample are presented in Table 3.1. The groups did not differ in terms of age (p=.10). The TDC group had significantly higher IQs than the CD-ADHD and CD+ADHD groups whereas the two clinical groups had similar IQs. Lastly, CD-ADHD and CD+ADHD reported a higher level of neighbourhood disadvantage than the TDC group.

Table 3.1 Demographic characteristics of the sample

Demographic characteristics	TDC[a] (n=30) Mean (SD)	CD-ADHD[b] (n=23) Mean (SD)	CD+ADHD[c] (n=28) Mean (SD)	Comparisons
Age	16.42 (1.22)	17.02 (1.15)	16.29 (1.39)	ns
Estimated IQ	105.10 (9.37)	93.78 (7.32)	94.71 (7.36)	a>b,c**
NES	4.14 (1.60) [±]	8.09 (3.75)	9.14 (3.58)	b,c>a**

**p<.001, [±] n=29 for this cell, ns=not significant, TDC=typically developing controls, IQ=intelligence quotient, NES=neighbourhood environment scale, CD=conduct disorder, ADHD=attention-deficit/hyperactivity disorder

3.10.1.2 Clinical characteristics

Data on the clinical characteristics of the sample are presented in Table 3.2. As expected, the CD-ADHD and the CD+ADHD groups had significantly more CD symptoms than the TDC group. In addition, the CD+ADHD had significantly more ADHD symptoms than the CD-ADHD and TDC groups. In terms of psychopathic and CU traits, the CD+ADHD group reported significantly higher levels than both the CD-ADHD and the TDC group. The TDC group had significantly lower levels of autistic traits than the CD-ADHD and CD+ADHD groups. The two CD groups had almost the same level. Here it should be noted that even though the clinical groups scored higher in the AQ than the TDC group, the average score for each group was well below the cut-off point of 32. Subsequently no participant was excluded

from the analyses. Lastly, there were no significant differences in state and trait anxiety between the three groups. The CD+ADHD group reported higher scores on both measures; however this main effect of group trend did not reach statistical significance (State: p=.07; Trait: p=.06).

Table 3.2 Clinical characteristics of the sample

Clinical characteristics	TDC[a] (n=30)	CD-ADHD[b] (n=23)	CD+ADHD[c] (n=28)	Comparisons
	Mean (SD)	Mean (SD)	Mean (SD)	
CD symptoms	0.37(0.72)	6.52(2.63)	7.46(2.84)	b,c>a***
ADHD-I symptoms	0.77(1.28)	1.96(1.78)	7.11(1.40)	c>a,b***
ADHD-H/I symptoms	0.47(0.78)	2.09(1.93)	6.59(1.99)	c>a,b***
AQ	15.30 (6.17)	19.70 (6.34)	21.00 (5.80) ±±	b>a*,c>a**
ICU	21.93 (7.39)	26.39 (8.18)	34.12 (9.54) ±±	c>a***, c>b**
YPI	101.03 (15.35) ±	112.07 (17.04)	124.88 (15.37) ±±	c>a***,c>b*
STAI-S	32.00 (8.73)	32.13 (9.84)	37.54 (11.32)	ns
STAI-T	37.40 (10.56)	40.35 (11.59)	44.39 (11.41)	ns

***p<.001, **p<.01, *p<.05. ±n=29, ±±n=26, ns=not significant , TDC=typically developing controls, CD=conduct disorder, ADHD-I=attention-deficit/hyperactivity disorder-inattention , ADHD-H/I=attention-deficit/hyperactivity disorder-inattention-hyperactivity/impulsivity, STAI-S = State-Trait Anxiety Inventory-State version, STAI-T = State-Trait Anxiety Inventory-Trait version, ICU=Inventory of Callous Unemotional traits, AQ = Autism-Spectrum Quotient, YPI=Youth Psychopathic traits Inventory.

3.11.1.3 Personality characteristics

Data on the personality characteristics of the sample are presented in Table 3.3. As measured using the Barratt Impulsiveness questionnaire (BIS-11), the CD+ADHD group had significantly higher levels of impulsivity than the TDC and CD-ADHD groups. There was a significant difference in the BAS drive subscale with the two clinical groups scoring higher than the TDC group. The CD-ADHD group scored significantly higher than the CD+ADHD group in the BAS reward responsiveness subscale. No group differences were observed between the three groups in the BAS fun subscale (p=.08) and or the BIS measure (p=.55).

Table 3.3 Personality characteristics of the sample

Personality characteristics	TDC[a] (n=30)	CD-ADHD[b] (n=23)	CD+ADHD[c] (n=28)	Comparisons
	Mean (SD)	Mean (SD)	Mean (SD)	
BIS-11	64.27 (10.72)	69.48 (9.46)	76.92 (9.17) ±±	c>a,b**
BAS Fun	11.20 (2.01)	12.26 (1.39)	12.39 (2.79)	ns
BAS Drive	9.77 (2.21)	12.30 (2.48)	11.89 (2.81)	b,c>a**
BAS Reward	16.33 (2.12)	17.57 (1.83)	15.04 (3.70)	b>c**
BIS	18.23 (4.02)	18.17 (3.87)	17.11 (4.30)	ns

**p<.01. ±±n=26, ns=not significant , TDC=typically developing controls, CD=conduct disorder, ADHD=attention-deficit/hyperactivity disorder, BIS-11=Barratt Impulsiveness Scale, BAS Drive = Behavioural Activation System-Drive subscale, BAS-Fun= Behavioural Activation System-Fun Seeking subscale, BAS Reward = Behavioural Activation System-Reward Responsiveness, BIS = Behavioural Inhibition System

3.12 Discussion

In this chapter we presented the clinical assessments, questionnaire measures and recruitment and testing procedures used in this thesis. We also reported the demographic, clinical and personality characteristics of the sample and conducted statistical analyses to investigate group differences in these variables. Here we summarise and discuss the key findings first by highlighting the differences between the TDC group and the CD as a whole, and then by looking at the impact of comorbid ADHD specifically.

3.12.1 TDC versus CD

In line with previous studies in the area of antisocial behaviour and conduct problems (Farrington, 2005a), we found that our CD group had significantly lower IQs than the TDC group. This finding, even though not surprising, highlights the need for further research on the educational needs of children and adolescents with externalising disorders. Furthermore, future research should also investigate the aspects of cognitive ability that can be strengthened and improved, and how individuals with CD can better engage with the educational system. The latter is important as research has indicated the high dropout rates from mainstream education within this population (Baker, 2013).

The CD group differed from the typically developing adolescents in terms of reported neighbourhood disadvantage. Even though the measure we used is only a proxy for socioeconomic status, the findings are in line with the notion that antisocial behaviour and conduct problems tend to be concentrated in areas with low socioeconomic status populations (Piotrowska, Stride, Croft, & Rowe, 2015). Even though no participant was excluded from the study and data analyses because of an ASD diagnosis, adolescents with CD reported significantly higher levels of autistic traits than the adolescents in the TDC group. Furthermore, in terms of CU and psychopathic traits, the CD group scored significantly higher than the TDC group.

There were no significant group differences in state or trait anxiety levels. This was somewhat surprising as research has shown high levels of comorbidity between CD and anxiety disorders (Short et al., 2016). However, our sample consisted solely of male participants and studies have reported that anxiety is more strongly concentrated within the female population, whereas males tend to dominate in terms of externalising disorders

(Nordström et al., 2013). No group differences were found in the BIS or the BAS fun subscale.

3.12.2 TDC versus CD-/+ADHD

The two groups CD-ADHD and CD+ADHD did not differ in terms of IQ, neighbourhood disadvantage and autistic traits. The latter highlights the association between CD, ADHD and autism and supports research that investigates common pathways between these three disorders (Mulligan et al., 2009; Simonoff et al., 2008). This finding meant that ADHD had no additional impact on the profile of CD adolescents.

Results from the psychopathic and CU traits measures indicated significantly elevated levels in the CD+ADHD group. The comorbid group was significantly different not only from the TDC group but also from the CD-ADHD group. Furthermore, the CD-ADHD group reported similar levels of these traits to the TDC group. This finding was somewhat unexpected, as research has consistently linked CD with high levels of CU traits (Viding, Frick, & Plomin, 2007). Our study implicates ADHD as a factor that exacerbates the presence of psychopathic and CU traits in adolescents with CD.

As expected, the CD+ADHD group reported higher impulsivity levels than the other two groups. The CD-ADHD group reported similar impulsivity levels to the TDC group. This was somewhat surprising, as CD has been linked to impulsivity independently of ADHD (Moeller, Barratt, Dougherty, Schmitz, & Swann, 2001).

The CD-ADHD and CD+ADHD groups reported significantly higher scores on the drive subscale of the BAS than the TDC group. This is somewhat to be expected as CD has been associated with risk-taking behaviours (Byrd, Loeber, & Pardini, 2014); the specific subscale measures the tendency to pursue reward and the persistence to achieve goals. Lastly, there was a significant difference in the reported BAS reward responsiveness subscale, but post-hoc tests showed that this was only between the CD-ADHD and CD+ADHD groups with the former group scoring significantly higher. The specific subscale measures the anticipation of and positive response to reward (Van den Berg et al., 2011).

3.13 Summary

Taking into consideration the above findings, we can conclude that the groups in this thesis have different behavioural profiles. CD participants differ from TDC participants in terms of IQ, neighbourhood disadvantage, autistic traits, and impulsivity; whereas adolescents with CD+ADHD were specifically elevated in impulsivity and CU traits. In the next four empirical chapters we investigate the neuropsychological profile of CD and the impact of comorbid ADHD in our chosen cognitive domains.

Chapter 4 “Cool” Executive Functions in Adolescents with conduct disorder with and without comorbid attention-deficit/hyperactivity disorder

4.1 Introduction

The goal of this thesis is to test in what way the presence of comorbid ADHD influences the neuropsychological profile associated with CD. In this Chapter, we focus on the first of the three neuropsychological areas of interest that this thesis examines – “cool” Executive Functions (EFs). As explained in Chapter 2, EF is an umbrella term which covers a spectrum of cognitive processes that characterise an individual’s ability to monitor, self-regulate and adjust behaviour to external stimuli. We also differentiated between “cool” and “hot” EFs. Here we present an empirical examination of the aforementioned processes in relation to the following “cool” sub-domains within EF: Response Inhibition, more specifically interference control (IC) and Working Memory (WM), more specifically Visuo-Spatial WM (VSWM). These constructs were defined, and their relationship to CD, with and without comorbid ADHD, was reviewed in Chapter 2. To summarise this work, it has been found that the relationship between CD, IC and VWM is inconsistent, with some studies identifying deficits and others not, and it was also noted that many of those studies have not controlled for ADHD comorbidity (Morgan & Lilienfeld, 2000; Oosterlaan, Logan, & Sergeant, 1998; Rubia, 2011). Indeed, when ADHD symptoms have been controlled, the effects of CD on IC and VWM are often weaker (Blair, 2015; Mairead Dolan & Park, 2002; Pennington & Ozonoff, 1996). This is in line with the literature showing a consistent, negative effect of ADHD on both IC and WM (Cortese et al., 2015; Sergeant, Geurts, & Oosterlaan, 2002; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005).

We employed two commonly used tasks to measure our “cool” EF constructs. VSWM was measured using the N-Back task (Shackman et al., 2006) and IC was measured using the Eriksen-Flanker task (Broyd, Helps, & Sonuga-Barke, 2011). Previous research with CD using the N-Back task has been rare. There are only a few published studies looking at VSWM task performance in ADHD (Karatekin, Bingham, & White, 2009; Strand et al., 2012) and even fewer relating to CD (Oosterlaan, Scheres, & Sergeant, 2005; Saarinen, Fontell, Vuontela, Carlson, & Aronen, 2015; Van Goozen et al., 2004). Studies using the Eriksen-Flanker task have consistently found that participants with ADHD display IC deficits (Frazier, Demaree, & Youngstrom, 2004; Hervey, Epstein, & Curry, 2004; Homack & Riccio, 2004; Lansbergen, Kenemans, & Van Engeland, 2007; Schwartz & Verhaeghen, 2008; Van Mourik,

Oosterlaan, & Sergeant, 2005). The findings have been inconsistent with regard to CD, with some studies reporting deficits, while others found no clear deficits after controlling for the effects of ADHD (Barnett, Maruff, & Vance, 2009; Sarkis, Sarkis, Marshall, & Archer, 2005).

Taking into account the results of these studies, we hypothesised that: a) the CD group as a whole (including participants with comorbid ADHD) will show deficits in both IC and VSWM and b) when the CD group is divided into those with and without comorbid ADHD, the deficits in these domains will be restricted to the comorbid group, i.e. CD+ADHD.

Our study addressed a number of methodological limitations present in previous studies: a) our sample consisted of a very-well screened CD clinical population which was also divided in adolescents with “pure” CD and adolescents with comorbid CD+ADHD, b) our study included two well-defined, well-validated cool executive function tasks targeting interference control and VSWM only minimally involving motivational or affective processes, and c) we used a VSWM task without a verbal element as visuospatial skills are more stable across adolescence than verbal skills; for example, the influence of verbal WM on cognitive functioning has been found to increase with age (Van de Weijer-Bergsma, Kroesbergen, & Van Luit, 2015).

4.2 Method

4.2.1 Sample

For a detailed description of the sample please see Chapter 3.

4.2.2 Ethics Statement

Please see relevant section in Chapter 3.

4.2.3 Experimental Tasks

Eriksen-Flanker task – Interference Control

A modified version of the Eriksen-Flanker task was employed (Broyd, Helps, & Sonuga-Barke, 2011). Participants were seated in front of a computer screen and were given the response pad. They were then presented with a horizontal array of five stimuli consisting of a combination of left and/or right-pointing arrows and equals signs. They were asked to focus on, and respond only to, the central target in the array, which was always an arrow. Participants had to press the left button if the central arrow was pointing to the left and the

right button if the central arrow was pointing to the right. The task consisted of 204 trials. The different trial types included: control (==<== or ==>==), congruent (<<<<< or >>>>>) and incongruent (<< > << or >> < >>) stimulus conditions, which were each presented an equal number of times (68 trials per condition). Each trial began with a fixation cross (+) presented at the centre of the screen for 500ms. After the fixation, the horizontal array of stimuli was presented for 200ms, followed by a black screen presented for 2800ms (Figure 4.1). The inter-trial interval was 3500ms.We calculated the mean reaction time (MRT) and the mean accuracy level (MACC; correct response= 1, incorrect response= 0) for the congruent and incongruent trials. MACC was calculated as the number of correct responses divided by the total number of presentations. The primary dependent variable (DV) for this task was the Interference Score, which is the magnitude of the additional inhibitory burden placed on the participants by the incongruent flankers. The Interference Scores were calculated as the difference in MRT/MACC between the congruent and incongruent conditions.

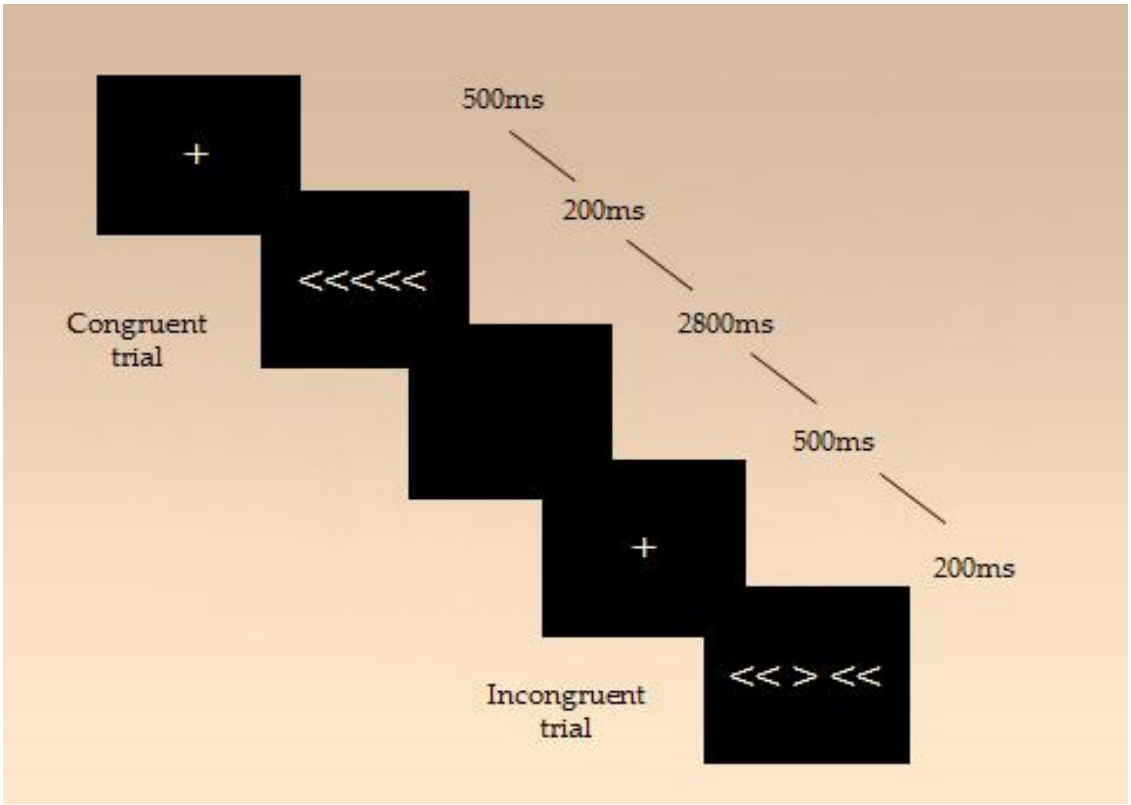

Figure 4.1. Schematic representation of the modified Eriksen-Flanker task.

N-Back task – Visual Working Memory (VWM)

We used an N-Back task (Shackman et al., 2006) with cognitive loads of 1 and 2, i.e. both 1-Back and 2-Back versions were used. Both tasks consisted of a series of trials presented on a computer screen and they were identical in all respects apart from the cognitive load. Participants completed the 2-Back task first and the 1-Back task second. At the start of each trial, a square box (9.5cm x 9.5cm) filled with a random jumble of different lower and upper case letters was presented on the screen (Figure 4.2 depicts the 2-Back trial sequence). Inside this box, another smaller square box (4.5cm x 4.5cm) appeared, framing a random assortment of lower case letters, and was presented for 500ms. After this period, the smaller box disappeared from the screen and participants had 3500ms to respond or not until the next trial. The inter-trial interval was 4000ms. The position of the smaller box changed from trial to trial in one of six locations within the big box. Participants were asked to press a button on the response pad only when the position of the small box appeared in the same location as the one that preceded it - in the case of the 1-Back task these are separated by 1 trial and in the case of the 2-Back task by 2 trials. Both tasks included a block of 18 practice trials and an experimental block of 74 trials, of which 18 trials were targets (where the small box appeared in the same location as one/two trials previously). The DVs were the percentage of omission (OM) errors, i.e. when participants did not press the button when they should have done so, and the percentage of commission (COM) errors, i.e., when participants pressed the button when they should not have. The percentage of Total Error Rate was also calculated by averaging the OM and COM errors for each group, to provide an index of overall task performance.

As mentioned above, the number of matched trials (which failure to identify resulted to OM errors) and mismatched trials (which failure to identify resulted to COM errors) differed in the 2-Back task (18 vs. 54 trials). This may have assisted the formation of a probabilistic guessing strategy. However, the task was set-up in such a way that a) the matched and mismatched trials occurred equally often and b) repeated presentation of a specific pattern on the trials intervening between initial presentation (trial 0) and response (trial 2) occurred rather infrequently. This is in line with previous research (Hadwin & Richards, 2016; Shackman et al., 2006) and ensured that successful performance, i.e. correct identification of the matched trials, was based more on memory and less on random guessing.

Figure 4.2. Schematic trial sequence of the 2-Back Visual Working Memory task that was used in the study. The top row depicts a trial sequence where the participants are required to press the button after the presentation of the last box (if they fail to do so, this is an *omission* error). The bottom row depicts a sequence where the participants should not press the button (if they do so, this is a *commission* error).

4.2.4 Procedure

Please see Chapter 3.

4.2.5 Data Preparation

For the Eriksen-Flanker task, we removed all incorrect trials (973 out of 15551) and trials in which the response time was shorter than 150ms (a further 61 trials) (Whelan, 2008). We then removed any trials that were either less than or longer than 3SDs each participant’s MRT (2510 trials). By applying a 3SD cut off we minimised the impact of longer responses on the mean (please see Whelan, 2008). After these steps, we calculated the means and standard deviations per group for each condition.

For the N-Back task, in the trials with a cognitive load of 2 we only used 72 out of 74 trials for the analysis, as the first two trials did not contain a target. From those 72 trials, the participants only had to press the button on 18 trials (probe in same position). They were not supposed to press the button in 54 of the trials (different position). We calculated the number of correct responses in the same condition (out of a possible 18) and the number of accurate responses in the different condition (out of a possible 54). After that we calculated the mean of these two values to obtain the overall accuracy rate per participant. We followed the same procedure for the 1-Back task with the exception of including 73 trials in the analysis.

4.2.6 Data Analysis

Statistical analysis was performed using the Statistical Package for the Social Sciences, Version 21 (SPSS Inc., Chicago, IL). Effect sizes are reported as *eta-squared* (η^2; small≥.02, medium≥.13, large≥.26) and *partial eta-squared* (η_p^2; small≥.01, medium≥ .06, large ≥.14) (Cohen, 1988b). For the analysis we followed a two-stage process. During the first stage, we compared task performance between the typically developing controls (TDC) (n=30) and all the participants with a CD diagnosis (n=51). In the second stage, we divided the CD participants into two groups: CD-ADHD (n=23) and CD+ADHD (n=28).

Data from the Eriksen-Flanker task - MRT and MACC on congruent trials, and MRT and MACC on incongruent trials - and the Interference Scores for both MRT and MACC, were analysed using univariate analysis of variance (ANOVA). We also analysed the MRT on the control trials to check for the possibility of facilitation effects. Data for the N-Back task were analysed using a number of different statistical approaches. As explained in Chapter 3, eighty-one participants (30 TDC and 51 with CD) completed just the 2-Back task, while a sub-group of that sample (n=58) completed both the 1 and 2-back tasks (22 TDC and 36 with CD). We therefore ran two analyses. First, we ran analyses for the entire sample to examine group differences on the 2-Back task using ANOVA and then for the reduced sample for whom 1-Back task data were available. We then completed a separate analysis including those with both 1- and 2-Back task data (n=58) using repeated measures ANOVA with task difficulty (1-Back versus 2-Back) as a within-subject factor. As mentioned above, the first trial in the 1-Back condition and the first two trials in the 2-Back condition required no WM processing and were therefore not included in the analysis. All analyses were completed both with and without including IQ as a covariate. For the analysis comparing the TDC, CD-ADHD and CD+ADHD groups, planned comparisons were also performed to identify the

source of main effects that were observed. The relation between the two EF tasks was examined by computing correlations between the Interference Score for MRT, MACC (Eriksen-Flanker) and OM and COM errors (2-Back task).

4.3 Results

In Table 4.1 we have summarised the data for the CD and TDC groups, together with group comparisons, across all outcome measures. When we split the CD group into those with and without comorbid ADHD (CD+ADHD and CD-ADHD), the task performance data changed accordingly. Table 4.2 summarises the data from the three groups across all outcome measures.

Table 4.1. A comparison of Task Performance between TDC and CD groups.

	TDC[a] (n=30)	**CD[b] (n=51)**	**Comparison**
	Mean (SD)	**Mean (SD)**	
IQ estimate	105.10 (9.37)	94.29 (7.28)	a>b, p<.001
Interference Control			
$MRT^{control}$	426.36 (54.76)	443.75 (74.34)	ns, p=.268
MRT^{congr}	422.39 (59.52)	438.72 (72.00)	ns, p=.298
$MRT^{incongr}$	512.30 (66.58)	550.53 (115.30)	ns, p=.101
Interference (MRT)	89.91 (32.79)	111.81 (58.96)	ns, p=.065
$MACC^{congr}$	0.99 (0.01)	0.96 (0.10)	ns, p=.078
$MACC^{incongr}$	0.94 (0.08)	0.84 (0.18)	a>b, p<.01
Interference (MACC)	0.05 (0.07)	0.12 (0.12)	b>a, p=.006
Visual Working Memory			
1-BACK	(n=22)	(n=36)	
OM %	56.57 (45.70)	59.41 (43.40)	ns, p=.813
COM%	5.45 (5.91)	10.05 (10.60)	ns, p=.068
Total errors (%)	31.01 (22.29)	34.73 (22.77)	ns, p=.539
2-BACK	(n=30)	(n=51)	
OM %	69.29 (38.60)	72.33 (29.60)	ns, p =.692
COM%	17.90 (13.63)	25.16 (17.03)	b>a, p=.050
Total errors (%)	43.59 (19.73)	48.74 (16.07)	ns, p=.205

Note. MRT is measured in milliseconds (ms). TDC=Typically Developing Controls, CD=Conduct Disorder, IQ=Intelligence Quotient, MRTcontrol=Mean Reaction Time control condition, MRTcongr=Mean Reaction Time congruent condition, MRTincongr=Mean Reaction Time incongruent condition, MACCcongr=Mean Accuracy congruent condition, MACCincongr=Mean Accuracy incongruent condition, OM=Omission errors, COM=Commission errors, ns=not significant.

Table 4.2. A comparison of Task Performance between TDC and CD groups with and without ADHD.

	TDC[a] (n=30)	CD-ADHD[b] (n=23)	CD+ADHD[c] (n=28)	Comparison
	Mean (SD)	**Mean (SD)**	**Mean (SD)**	
IQ estimate	105.10 (9.37)	93.78 (7.36)	94.71 (7.36)	a>b,c, p<.001
Interference Control				
$MRT^{control}$	426.36 (54.76)	460.03 (79.50)	430.38 (68.36)	ns, p=.162
MRT^{congr}	422.39 (59.52)	446.89 (73.74)	432.00 (71.18)	ns, p=.431
$MRT^{incongr}$	512.30 (66.58)	567.84 (114.44)	536.30 (116.12)	ns, p=.141
Interference (MRT)	89.91 (32.79)	120.95 (53.81)	104.30 (62.84)	ns, p=.095
$MACC^{congr}$	0.99 (0.01)	0.98 (0.03)	0.94 (0.13)	a>b,c , p=.023
$MACC^{incongr}$	0.94 (0.08)	0.90 (0.09)	0.79 (0.21)	a,b>c, p<.001
Interference(MACC)	0.05 (0.07)	0.09 (0.14)	0.15 (0.14)	c>a,b, p=.003
Visual Working Memory				
1-BACK	(n=22)	(n=16)	(n=20)	
OM %	56.57 (45.70)	54.86 (45.72)	63.06 (42.28)	ns, p=.837
COM%	5.45 (5.91)	11.25 (13.53)	9.09 (7.76)	ns, p=.150
Total errors (%)	31.01 (22.29)	33.06 (22.68)	36.07 (22.43)	ns, p=.766
2-BACK	(n=30)	(n=23)	(n=28)	
OM %	69.29 (38.60)	64.97 (30.83)	78.37 (27.65)	ns, p=.330
COM%	17.90 (13.63)	21.18 (14.98)	28.44 (18.15)	c>a, p=.039
Total errors (%)	43.59 (19.73)	43.07 (13.67)	53.41 (16.4362)	c>a,b , p=.048

Note. MRT is measured in milliseconds (ms). TDC=Typically Developing Controls, CD=Conduct Disorder, ADHD=Attention Deficit Hyperactivity Disorder, CD-ADHD= Conduct Disorder without Attention Deficit Hyperactivity Disorder, CD+ADHD= Conduct Disorder with Attention Deficit Hyperactivity Disorder, IQ=Intelligence Quotient, MRTcontrol=Mean Reaction Time control condition, MRTcongr=Mean Reaction Time congruent condition, MRTincongr=Mean Reaction Time incongruent condition, MACCcongr=Mean Accuracy, congruent condition, MACCincongr=Mean Accuracy, incongruent condition, OM=Omission errors, COM=Commission errors.

4.3.1 Eriksen-Flanker task

4.3.1.1 TDC versus CD

There were effects of group on the MACC for the incongruent trials [F (1, 79) = 8.60, p=.004, η^2 = 0.09] and on the MACC Interference score [F (1, 79) = 7.97, p=.006, η^2 = 0.09]. The group effects for the interference score were driven by the discrepancy between the congruent and the incongruent trials. The CD group were much more affected by the congruency manipulation than the TDC group. There were no group effects on the MRT for the congruent [F (1, 79) = 1.09, p=.29, η^2 = 0.01] or the incongruent trials [F (1, 79) = 2.75, p=.10, η^2 = 0.03], while there were trends towards significant group differences for the MACC in the congruent trials [F (1, 79) = 3.19, p=.07, η^2 = 0.40] and the MRT Interference Score [F (1, 79) = 3.49, p=.06, η^2 = 0.04]; both groups were slower on incongruent trials but effect of congruency was disproportionately greater in the CD group.

Results obtained when controlling for IQ: The group difference in the MACC Interference score was evident even after controlling for IQ differences between the groups [F (1, 78) =4.35, p=.04, η_p^2=0.053], while the group effect on MACC for Incongruent trials was reduced to a strong trend [F (1, 78) =3.44, p=.06, η_p^2=0.042].

4.3.1.2 TDC versus CD-/+ADHD

There were group effects on the MACC for both the congruent [F (2, 78) = 3.94, p=.023, η^2 = 0.09] and incongruent [F (2, 78) = 8.39, p<.001, η^2 = 0.18] trials and for the MACC Interference score [F (2, 78) = 6.45, p=.003, η^2 = 0.14]. Post-hoc comparisons between the three groups, indicated that the CD+ADHD group showed poorer interference control compared to both the CD-ADHD [F (2, 78) = 4.58, p=.036] and TDC [F (2, 78) = 12.67, p<.001] groups; the CD+ADHD group made more errors than the other two groups both in the congruent [vs TDC: F (2, 78) = 6.96, p=.01; vs CD-ADHD: F (2, 78) = 4.75, p=.04] and incongruent trials [vs TDC: F (2, 78) = 1.85, p<.001; vs CD-ADHD: F (2, 78) = 7.45, p=.008]. This deficit was evident even after IQ was entered as a covariate in the analysis for the MACC Interference Score [F (2, 77) = 4.59, p=.013, η^2 = 0.11]. In addition, the main analysis indicated that there were no group effects on the MRT Interference Score [F (2, 78) = 2.43, p=.09, η^2 = 0.05], and no group effects on the MRTs for the congruent [F (2, 78) = 0.85, p=.431, η^2 = 0.02] and incongruent [F (2, 78) = 2.01, p=.141, η^2 = 0.05] trials.

4.3.2 N-Back task

4.3.2.1 TDC versus CD

There was a group effect on COM errors made during the 2-Back task [F (1, 79) =3.96, p=.05, η^2=0.05], with the CD group committing more errors than the TDC group. This suggests a difficulty in engaging working memory processes sufficiently in order to complete the task. There was a trend towards a group effect on COM errors made during the 1-Back task [F (1, 56) =3.46, p=.06, η^2 = 0.01], which may reflect the smaller sample size for this variant of the task. There were no group effects on OM errors made during the 1-Back task [F (1,56)=0.06, p=.81, η^2 = 0.001], the 1-Back Error Rate [F (1,56)=0.38, p=.54, η^2 = 0.01], OM errors made during the 2-Back task [F (1,79)=0.16, p=.69, η^2 = 0.002] or the 2-Back Error Rate [F (1,79)=1.63, p=.21, η^2 = 0.02] (please see figure 4.3).

Results obtained when controlling for IQ: when entering IQ as a covariate in the analysis, the group difference on COM errors in the 2-Back task was no longer significant [F (1, 78) =1.14, p=.29, η^2 = 0.01]. This suggests that this group difference may have been partly driven by differences in IQ.

When repeated-measures ANOVA was run in the sub-sample that completed the tasks at both cognitive loads (1 and 2-Back; n = 58), with task difficulty included as a within-subject variable, there was no main effect of group on task performance [F (1, 56) = 0.45, p=.503, η_p^2= .008], a main effect of task difficulty on overall task performance [F(1,56)=62.21, p<.001, η_p^2= .526], but no interaction between group and task difficulty [F (1, 56) = 0.04, p=.841, η_p^2= .001].

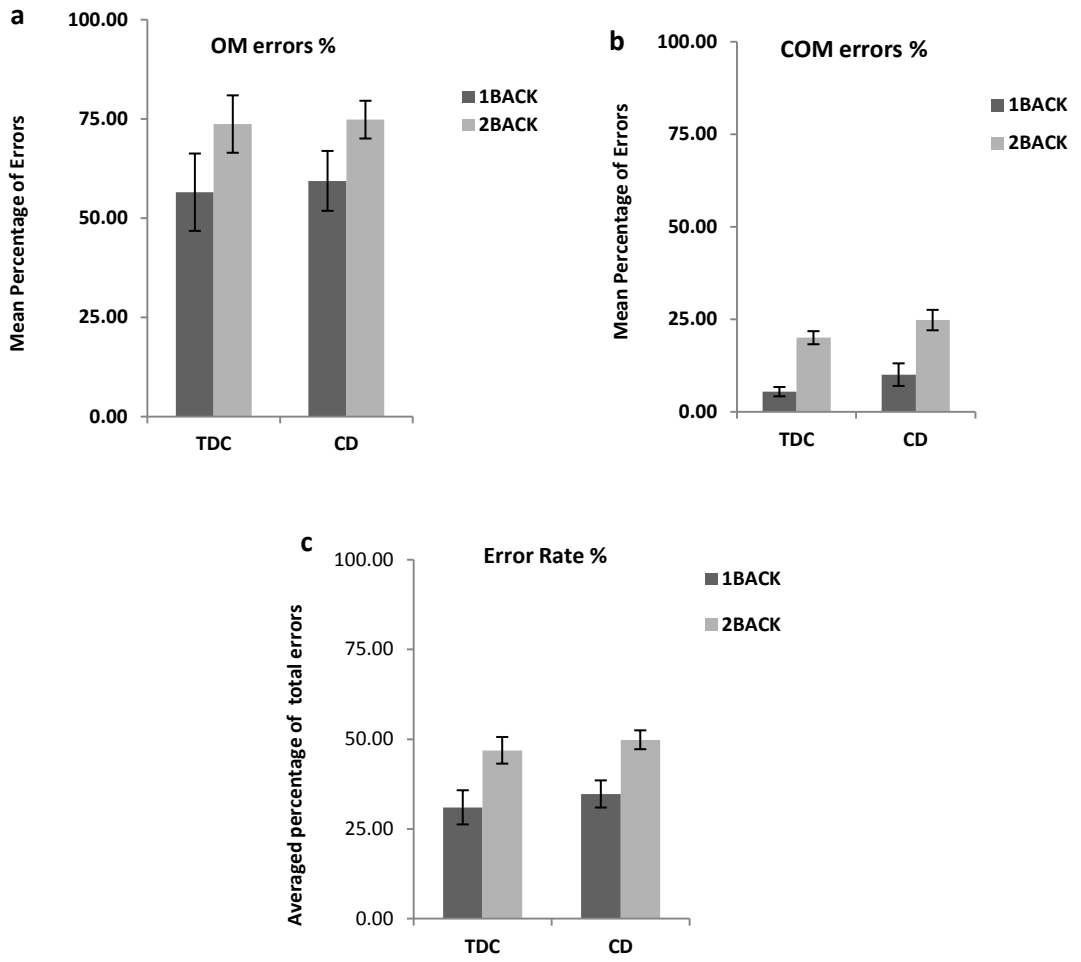

Figure 4.3. (a) Effect of task load on omission errors during the visual working memory task; (b) Effect of task load on commission errors; (c) Total error rates across error types at the two cognitive loads, by group. (The bars show mean values whereas the error bars show ±1 standard error of the mean. TDC=Typically Developing Controls, CD=Conduct Disorder, OM%=percentage of Omission errors, COM%=percentage of commission errors).

4.3.2.2 TDC versus CD-/+ADHD

There were main effects of group on 2-Back COM errors [F (2, 78) = 3.374, p=.039, η^2 = 0.08] and 2-Back total error rates [F (2, 78) = 3.155, p=.048, η^2 = 0.075]. Post-hoc comparisons revealed that the CD+ADHD group committed significantly more COM errors than the TDC group [F (2, 78) = 2.56, p=.013], but not the CD-ADHD group [F (2, 78) = 1.64, p=.104]. When considering 2-Back total error rates, the CD+ADHD group committed significantly more errors than both the TDC [F (2, 78) = 2.184, p=.032] and CD-ADHD groups [F (2, 78) = 2.143, p=.035].

There were no significant group differences on 1-Back OM errors [F (2, 55) = 0.178, p=.0837, η^2 = 0.006], 1-Back COM errors [F (2, 55) = 1.962, p=.150, η^2 = 0.067], 1-Back total error rate [F (2, 55) = 0.268, p=.766, η^2 = 0.01] or 2-Back OM errors [F (2, 78) = 1.125, p=.330, η^2 = 0.028] (please see figure 4.4).

However, when we controlled for IQ, the group effect for 2-Back COM errors was no longer significant [F (2, 77) = 2.04, p =.138, η_p^2= .050]. This suggests that group differences in COM errors could be partly explained by IQ differences between the groups. For the 2-Back total error rate, the analysis revealed a group difference at a trend level [F (2, 77) = 2.640, p =.078, η_p^2= .064], with the CD+ADHD group showing significantly more total errors than the CD-ADHD group [F (2, 77) = 2.170, p =.033], but not the TDC group [F (2, 77) = 1.56, p =.124].

When a repeated measures ANOVA was run on the data from the sub-sample that completed the N-Back task at both cognitive loads (n = 58), with task difficulty included as a within-subjects factor, there was no main effect of group on total error rate [F (2, 55) = 0.602, p=.551, η_p^2= .021]. We found a main effect of task difficulty with participants finding the n=2 load more difficult than the 1=load and a main effect of error type with participants making more omission than commission errors. We found no significant interaction between group and task difficulty [F (2, 55) = 0.46, p=.632, η_p^2= .017], error type and group [F (2, 55) = 0.14, p=.869, η_p^2= .005] and task difficulty and error type [F (1, 55) = 0.15, p=.695, η_p^2= .003].

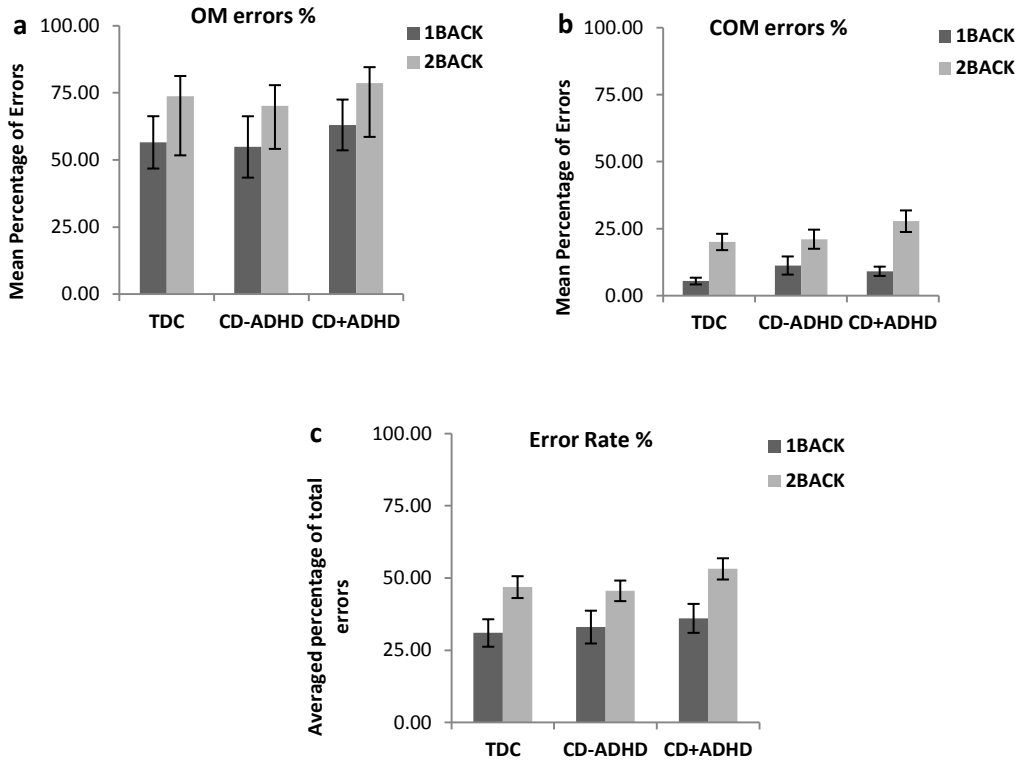

Figure 4.4. (a) Effect of task load on omission errors; (b) Effect of task load on commission errors; (c) Percentage of total error rate between groups in the two task loads (The bars demonstrate mean values whereas the error bars show ±1 standard error of the mean. CD=Conduct Disorder, ADHD=Attention Deficit/ Hyperactivity-Disorder, OM%=percentage of Omission errors, COM%=percentage of commission errors).

4.3.3. Relationship between the outcome measures from the "cool" EF tasks

There was a positive correlation between the Interference Score for MACC in the Eriksen-Flanker task and COM errors in the 2-Back task (please see table 4.3). This implies that the higher the interference score, the more the false positives, i.e. COM errors, the person makes. Poor interference control was associated with poor working memory – suggesting the existence of a coherent underlying "cool" EF construct. In addition, as already implied by the above results, there was a negative correlation between IQ and COM errors. This suggests that the lower the IQ, the more COM errors participants are making.

Table 4.3. Correlation Matrix of the "cool" EF tasks' outcome measures

	1	2	3	4
1. IQ				
2. OM Errors % -2-Back	-.035			
3. COM Errors % - 2-Back	-.220*	-.108		
4. Interference Score-MRT	-.106	-.028	.033	
5. Interference Score-MACC	.210	.077	.306**	.145

Note. 1-Back task outcome variables have not been included in this table because 1-Back data were only available for a subset of the sample. **p<.01, *p<.05, MRT= Mean Reaction Time, MACC= Mean Accuracy, OM=Omission, COM= Commission, IQ=intelligence quotient

4.4 Discussion

We used an interference control (IC) task and a visual working memory (VWM) task to investigate whether adolescents with CD show EF deficits compared to typically developing controls (TDC). We also divided the CD group into those with and those without comorbid ADHD to investigate the impact of ADHD comorbidity on the neuropsychological profile associated with CD. We identified four main findings which we discuss below.

First, in general, the CD group when considered as a whole had poorer “cool” EF than controls. This effect was most clear for the Eriksen-Flanker task data where results indicated a significant difference in the interference score for accuracy (ACC), with the CD group displaying a higher interference score than the TDC group. This indicates lower interference control in the former group. This was also confirmed by the subsequent analysis for the Mean Accuracy (MACC) measure, where the CD group was less accurate than the controls for the incongruent trials. There was no difference between the TDC and the CD group in the interference scores for RT. The situation was less clear on the N-Back task. When the larger sample that completed just the 2-Back task was considered there was a significant effect for the errors of commission. However, this effect was lost when the restricted sample with data from both the 1- and 2-Back versions of the task were considered.

Second, when the CD group was divided into those with and those without a diagnosis of ADHD, it became clear that to a large extent the “cool” EF deficits in the CD group as a whole were driven by the individuals with comorbid ADHD. For the Eriksen-Flanker task, results revealed that the CD+ADHD group was significantly less accurate than both the TCD and the CD-ADHD groups. This means that adolescents with CD+ADHD found it more difficult to suppress their actions, i.e., they could not recruit adequate inhibitory control on the incongruent trials. This led to disinhibited responses which made them commit more mistakes. One interesting finding is that even though the CD-ADHD group was the slowest of the three groups, this group was still more accurate than the CD+ADHD group. A similar, though weaker, pattern was seen for visual working memory. For the 2-Back task results obtained when comparing the three groups indicated that the CD+ADHD group had a higher total error rate and committed more false positive errors (i.e. COM errors) than the control group. In contrast, the CD-ADHD group performed in a similar way to the control group. The 2-Back task is considered to be a task with a high cognitive demand; when there is a high

load of information to remember, individuals find it more difficult to store and manipulate information that needs to be constantly updated. So the fact that adolescents with CD-ADHD did not show impairment in VWM could imply the use of a cognitive strategy – perhaps the CD-ADHD participants had a cognitive deficit but were able to adopt a different strategy from the typically developing participants which allowed them to compensate and show seemingly intact performance. Research has shown that individuals with high WM capacity are able to adopt a successful strategy in order to overcome interference (Long & Prat, 2002).

Third, we found that IC ability and WM capacity were positively correlated; the more limited the capacity of WM is, the lower the ability to accurately distinguish between contrasting information. This relationship also suggests that the Eriksen-Flanker task and the 2-Bask task tap cognitive processes that are partly overlapping (Casey, Giedd, & Thomas, 2000). This claim is also supported by previous research on IC and WM (Stins, Polderman, Boomsma, & de Geus, 2005a), where IC was shown to be mediated by WM. Inadequate ability to handle sources of cognitive conflict is influenced by WM capacity; the weaker the capacity to process and hold information, the lower the ability to control sources of distraction. In this study, these issues were particularly evident in the CD+ADHD group.

Fourth, as the groups differed in IQ we included this variable as a covariate in the analyses. The group differences in the Eriksen-Flanker task remained significant even after controlling for possible influences of IQ on task performance. The CD+ADHD group performed the worst. However, for the 2-Back task when the effect of IQ was controlled, some of the group differences were reduced to non-significant levels. Specifically, there was no difference between the TDC and the CD+ADHD group in task performance. This implies that the initial results may have been driven, at least in part, by group differences general cognitive ability. Even though previous studies have indicated that WM and IQ are related, but not identical (Conway, Kane, & Engle, 2003), we cannot be certain whether the WM deficit in the CD+ADHD group was due to the effect of ADHD on task performance or the low IQ of this group. The effect of IQ in studies of CD needs to be carefully examined. CD has been robustly and repeatedly linked to low IQ (Farrington, 2005b) and researchers have highlighted that “hand-picking” CD participants with normal or relatively high IQs so they are IQ-matched to controls, may reduce the construct validity of CD (Short, Sonuga-Barke, Adams, & Fairchild, 2016).

Overall, these findings help to clarify the relative contributions of CD and ADHD to “cool” executive function deficits observed in children with CD. Previous research has yielded mixed results, although in general when ADHD symptoms are controlled, the effects attributable to CD reduce in size. Here, the effects of accounting for ADHD were quite marked – with the non-comorbid CD group performing in a very similar way to controls.

Strengths and Limitations

Our study had several strengths relative to previous studies. The adolescents (n=51) that were included in the CD group were a more homogeneous group than the mixed CD and ODD groups that have largely been used in previous research; all the participants that were in the CD group had received a full CD diagnosis – which was not the case in most previous studies. It may be that when EF deficits are found in children and adolescents with conduct problems, ODD is the disorder that contributes more than CD to the observed deficit. Although it should be noted that even though all the participants in the clinical group had CD diagnosis they could also have comorbid ODD. In relation to the performance of the CD+ADHD group in the IC task, results indicated performance impairments that were particularly marked for accuracy rather than reaction time. This confirms our initial hypothesis that the CD+ADHD group would perform worse than the other two groups, and lends support to the line of research that claims ADHD to be the main contributor to the “cool” EF deficits observed in the comorbid group. In addition, our study examined two well-validated “cool” EF domains in the same sample and its sample size allows for meaningful group comparisons. Lastly, another strong point is the fact that the sample was homogeneous with regard to gender, as only male adolescents were included.

On the other hand, the results of this study should be interpreted in the light of the following limitations. First, in the Eriksen-Flanker task, we observed ceiling effects in all three groups for the accuracy in the congruent condition. This suggests that the groups found this condition quite easy and did not have a problem choosing the correct response when the flanker stimuli were congruent. However, this did not prevent us from demonstrating group differences in the incongruent condition.

For the N-Back task, it should be noted that the majority of the studies using cognitive loads of 0, 1 and 2, in order to assess the gradual impairment that is caused by the escalating cognitive load. In our study, a load of 0 was not included because of testing time restrictions, so it was not possible to show the aforementioned escalation effects. In addition, as the

participant numbers were low for the 1-Back task, we were unable to detect any significant differences between the groups. However, the controls performed better in the 1-Back than in the 2-Back condition of the task, while the CD group exhibited a similar response pattern at both load conditions. Lastly, although we were interested in WM in general, our use of a visuospatial WM task may mean our results do not apply to WM more broadly. However, research has shown that all WM tasks have been found to activate a shared network of brain areas, in addition to domain-specific verbal and visuospatial WM sub-systems (Alloway, Gathercole, & Pickering, 2006).

4.5 Summary

In this study, we compared CD adolescents with and without ADHD in terms of neuropsychological performance on “cool” EF tasks, to examine the impact of ADHD comorbidity on the neuropsychological profile of CD. We found that, in general, such deficits, where they existed, were driven almost exclusively, by the existence of comorbid ADHD. Our study contributes to the literature and is differentiated from previous research on the area in two main ways: a) the adolescents in the clinical groups were all diagnosed with CD and b) the presence of ADHD comorbidity within the sample was carefully assessed and included as a factor of interest. Taken together, results from the “cool” EF tasks evaluating interference control and visuo-spatial working memory indicate that adolescents with CD do not differ from controls in these processes unless they also meet diagnostic criteria for comorbid ADHD. These results are consistent with models of ADHD/CD comorbidity that highlight the additive role of the two conditions and studies that emphasise that “cool” EF deficits are restricted to children and adolescents with CD and comorbid ADHD, rather than those with pure CD. In the next chapter, we will explore similar issues with regard to “hot”, motivational aspects of executive function.

Chapter 5 “Hot” Executive Functions in adolescents with conduct disorder with and without comorbid attention-deficit/hyperactivity disorder: Delay-related decision making

5.1 Introduction

In Chapter 2, we described the way in which CD and ADHD have both been associated with sub-optimal decision making, while in Chapter 4 we focused on “cool” EFs. In this chapter, we examine “hot” EFs (please see chapter 2 for the distinction between “cool” and “hot” aspects of executive functions). We employed two behavioural tasks, one investigating delay-related choice and the other delay-related frustration. Data from these tasks will be presented and performance differences between the different groups will be discussed. Our goal, similar to chapter 4, is first to examine whether adolescents with CD show deficits in delay-related decision-making and then to investigate the extent to which any group differences are related to co-occurring ADHD.

Both CD and ADHD share a common, core behavioural trait: impulsivity. Among the several definitions that describe impulsivity (please see review by Dalley & Robbins, 2017), Sagvolden, Aase, Johansen, and Russell (2005) describe it as a preference for immediate rewards over larger but delayed rewards. This index of impulsiveness (as described by Schweitzer & Sulzer-Azaroff, 1995) has also been one of the most robust motivational markers of ADHD (Luman, Oosterlaan, & Sergeant, 2005; Sonuga-Barke, Sergeant, Nigg, & Willcutt, 2008). Research on motivational processing in CD has been focused mostly on: a) risk-taking using gambling paradigms like the Risky Choice Task and Iowa Gambling Task (Byrd et al., 2014; Fairchild et al., 2009; Sully et al., 2015a; Syngelaki, Moore, Savage, Fairchild, & Van Goozen, 2009) and b) temporal discounting (TD) of reward tasks (Fanti, Kimonis, Hadjicharalambous, & Steinberg, 2016; White et al., 2014). This heightened time preference -favouring of the present over the future-is a consistent finding across the CD literature and has been attributed to deficits in striatal and prefrontal cortex function (Ballard & Knutson, 2009; Critchfield & Kollins, 2001) (also see Sonuga-Barke, 2014 for an alternative hypothesis). In these types of delay-related paradigms, TD is measured by assessing participants’ preferences between an immediate reward and a delayed reward of greater value (e.g., £10 today or £15 in a week’s time). The amount of reward varies according to the variation in the time delay (measured in days or weeks) (Mitchell, 1999).

White et al. 2014 used a TD paradigm and compared task performance between typically-developing adolescents and adolescents with CD. The task that was used involved varying values of immediate reward ($0 to $10.50 in varying $0.50 increments) and a larger reward, held at a constant value ($10). The receipt of the larger reward was delayed by different time intervals (0, 7, 30, 90, 180, 360 days). Results indicated that adolescents with CD chose significantly smaller amounts of immediate reward rather than the larger future rewards; they exhibited increased TD impulsivity compared to controls. However, the specific study had one important limitation: the CD group consisted of twenty-one adolescents, five of whom had comorbid ADHD. Furthermore, the CD sample included both males and females. This complicates the interpretation of the findings further, as research has shown that the pathophysiological basis of CD may be, at least to some extent, sex-specific (Smaragdi et al., 2017).

Despite the evidence of altered decision making in CD, only a small number of studies have investigated delay-related decision making in CD. We employed two tasks which have been used widely to measure “hot” EF constructs. Impulsive choice was measured using the Maudsley Index of Delay Aversion task (MIDA; Kuntsi, Stevenson, Oosterlaan, & Sonuga-Barke, 2001) and delay-related frustration was measured using the Delay Frustration task (DeFT; Bitsakou, Antrop, Wieserma, & Sonuga-Barke, 2006). Impulsive choice has been investigated thoroughly in ADHD but not in CD. In ADHD, it has been explained as a reduced tolerance for delay (Sonuga-Barke, Taylor, Sembi, & Smith, 1992). In brief, children with ADHD prefer immediate rewards because they are primarily more concerned with reducing the overall delay time than maximising the final reward (please see chapter 2 for a detailed review of this concept). The DeFT task has not been used in this clinical population before. Moreover, most of the existing studies have not explicitly examined the impact of ADHD comorbidity on their CD-related findings (Dolan & Park, 2002; Morgan & Lilienfeld, 2000; Toupin, Déry, Pauzé, Mercier, & Fortin, 2000). On the other hand, studies of impulsive choice tasks in hyperactive and non-hyperactive children have concluded that group differences can be fully explained by comorbid disruptive behaviour disorders (DBDs), i.e. CD and/or ODD (Kuntsi, Oosterlaan, & Stevenson, 2001)

The present study is the first to examine delay-related decision making and frustration responses to the imposition of delay in a sample of adolescents with “pure” CD. We hypothesised that a) the CD group as a whole will experience greater levels of impulsive choice and delay-related frustration compared to the TDC group, but that b) once the CD

group is split into CD-ADHD and CD+ADHD subgroups, deficits in impulsive choice and ability to tolerate delay will be significantly more pronounced in the CD+ADHD group than the CD-ADHD and TCD groups. Our hypothesis is based on the finding that both ADHD and CD have been independently associated with deficits in “hot” EF processes (Rubia, 2011).

Our study extends previous research in the area of “hot” EFs in the following ways: a) by focusing specifically on delay-related choice and frustration using two behavioural paradigms which combine emotion regulation and decision-making processes; and b) by increasing the homogeneity of the CD group by only recruiting participants with full CD diagnoses rather than a mixed-group of youths with CD and ODD.

5.2 Method

5.2.1 Sample

Please see Chapter 3.

5.2.2 Ethics Statement

Please see Chapter 3.

5.2.3 Experimental Tasks

The Maudsley Index of Childhood Delay Aversion task (MIDA)

The MIDA (Kuntsi et al., 2001) task was designed to assess impulsive choice by quantifying the extent to which an individual chooses a small reward of less value that is available immediately, than a large reward of greater value that is available after a short delay. The MIDA was designed to have two conditions – one with and one without post-reward delay to examine the impact of total delay on reward choice to examine the delay aversion hypothesis. However, in this study only the condition with no-post reward delay was included.

The task was presented as a space game (figure 5.1), in which the participants had to shoot down enemy spaceships to win points. Participants were seated in front of a computer screen and were asked to use the computer mouse as their “weapon” - once the participant clicked the mouse, bombs were fired from their spaceship and the enemy spaceship was destroyed. The task consisted of twenty trials or “missions”. For each mission the participant was given

a choice between a small immediate reward, i.e. shooting down a single enemy spaceship (SS; 1 point – 10p), which involved a 2sec pre-reward delay, and a large delayed reward, i.e. shooting down two enemy spaceships (LL; 2 points – 20p), which involved a 30sec pre-reward delay. Choosing the SS reward meant that the game moved on to the next trial and the overall task length was reduced. Participants were instructed that they would be allowed only one shot per mission. The aim of the task was to win as many points as possible, which would result in a greater monetary reward. The DV in this task was the percentage of choices that the impulsive choice was selected (SS%). Participants were given two practice trials, one for each reward choice.

Figure 5.1. Maudsley’s Index of Childhood Delay Aversion task, presented in the format of a space game. Participants had control over a spaceship which they had to defend by shooting down enemy spaceships and winning points.

Delay Frustration Task (DeFT)

The DeFT (Bitsakou, Antrop, Wieserma, & Sonuga-Barke, 2006) was designed to assess the degree of frustration experienced by individuals when unexpected and unavoidable delay is introduced into a simple task. The task involved a series of simple maths questions presented on a computer screen (figure 5.2). Each question was presented separately, with each question accompanied by four possible solutions. Participants were given a response pad and asked to select the correct answer, i.e. A, B, C or D. On thirty-nine trials, as soon as the participant pressed the button, the program moved onto the next trial (no post-response delay condition). However, on eight trials access to the next maths question was delayed by 20sec (unexpected post-response delay condition). In addition, eight distractor trials were included, where the delay period was varied between 2 to 10 seconds. These were included to reduce the chances that participants would identify a consistent pattern of the presentation of the post-response delay trials (i.e. that they would considered to be due to a random fault). On the post-delay condition and distractor trials the response button was inactive during the delay period and any responses made by the participant were therefore ineffective at accessing the subsequent trial. At the end of the delay period the response box was reactivated and the first response became effective in allowing the participant to move on to the next maths question. The post-response delay trials were added in a pseudo-random order: for the first five trials there was no delay, and the delay trials were randomised across the remaining fifty trials. The distractor trials were not included in the analysis.

Participants were given clear instructions prior to the beginning of the task. They were “warned” that the computer had been malfunctioning and if the computer showed that it did not register their response, they needed to respond again before they could move on to the next maths question. The DV in this task was the mean total duration (MTD) of responding per second of delay on the eight delay trials. The MTD was the product of the average response frequency, i.e. the number of responses per second of delay, and the average duration of each response, i.e. the total time of response per second of delay. The MTD was logged in four time bins: 0-4 secs, 5-9 secs, 10-14 secs and 15-19 secs into the delay period. The final analysis contained only the first and last bin, to contrast the level of delay-related frustration at the beginning and at the end of the delay period. In addition, the first second (0-1sec) was excluded from the analysis, as it indexed reaction to the task and not delay-related frustration.

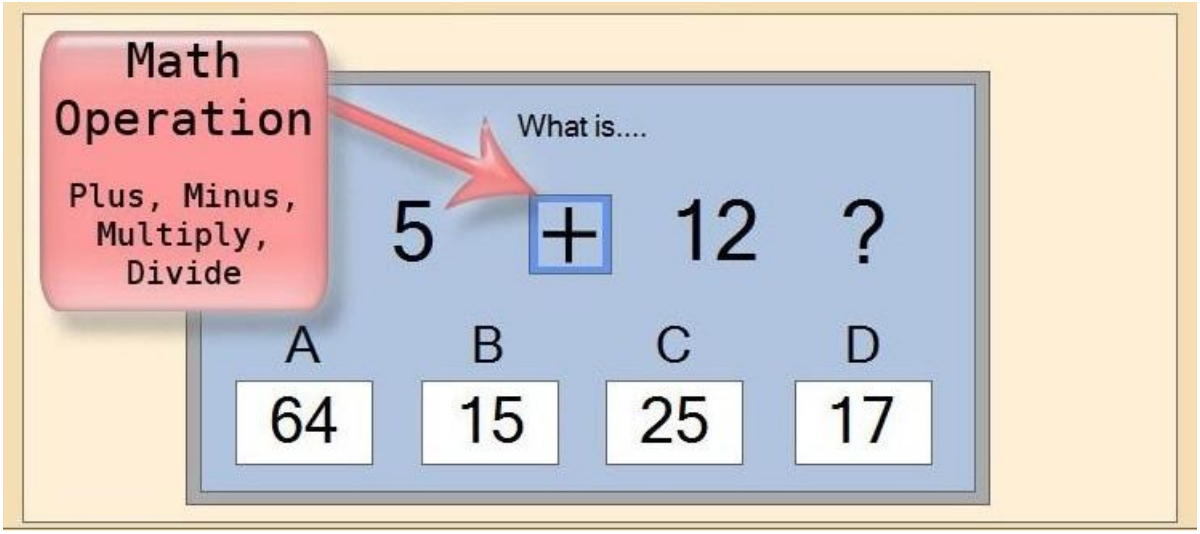

Figure 5.2. The Delay Frustration Task. The primary task involved participants to solve maths questions by selecting one out of four possible solutions.

5.2.4 Procedure

Please see Chapter 3.

5.2.5 Data Analysis

Statistical analyses were performed using the Statistical Package for the Social Sciences, Version 21 (SPSS Inc., Chicago, IL). Effect sizes are reported as partial eta-squared (η_p^2; small≥.01, medium≥ .06, large ≥.14) (Cohen, 1988a). For the analysis we followed the same two-stage process as in the previous chapter. During the first stage we compared task performance between the controls (n=30) and all the participants with a CD diagnosis (n=51). In the second stage, we divided the CD participants into two groups: CD-ADHD (n=23) and CD+ADHD (n=28). The analysis chosen for both tasks was based on previous research using either the MIDA (Marco et al., 2009), the DeFT (Bitsakou et al., 2006) or both (Bitsakou, Psychogiou, Thompson, & Sonuga-Barke, 2009). IQ was entered as a covariate in both sets of analyses. For the MIDA, task-level comparisons between the different groups were analysed using a univariate ANOVA with group as the between-subjects factor. For the DeFT, univariate ANOVAs and repeated measures ANOVA was employed to assess for group differences, with MTD (Time bins: 0-4 secs, 15-19 secs) as the within-subjects factor. A correlation analysis was employed between cognitive ability and the outcome measures of the two tasks to examine whether IQ and performance on the tasks were related

5.3 Results

In Table 5.1 we have summarised the data for the CD and TDC groups, together with group comparisons, across all outcome measures. When we split the CD group into those with and without comorbid ADHD (CD+ADHD and CD-ADHD), the task performance data changed accordingly. Table 5.2 summarises the data from the three groups across all outcome measures.

Table 5.1. A comparison of Task Performance between TDC and CD groups

	TDC[a] (n=30)	CD[b] (n=51)	Comparison
	Mean (SD)	**Mean (SD)**	
IQ estimate	105.10 (9.37)	94.29 (7.28)	a>b, p<.001
MIDA – impulsive choice			
SS%	37.53 (37.78)	54.59 (32.53)	b>a, p=.03
DeFT - delay-related frustration			
MTD 1-4secs	254.83 (305.24)	262.16 (228.54)	ns, p =.902
MTD 15-19secs	319.72 (329.59)	363.83 (300.61)	ns, p=.540

TDC=Typically Developing Controls, CD=Conduct Disorder, IQ=Intelligence Quotient, SS%=percentage of smaller sooner reward, MTD=Mean Total Duration, ns=not significant.

Table 5.2. A comparison of Task Performance between TDC and CD-/+ADHD groups

	TDC[a] (n=30)	CD-ADHD[b] (n=23)	CD+ADHD[c] (n=28)	Comparison
	Mean (SD)	**Mean (SD)**	**Mean (SD)**	
IQ estimate	105.10 (9.37)	93.78 (7.36)	94.71 (7.36)	a>b,c, p<.001
MIDA-impulsive choice				
SS%	37.53 (37.78)	57.97 (33.42)	51.82 (32.12)	b>a, p=.035
DeFT – delay-related frustration				
MTD 1- 4secs	254.83 (305.25)	260.68 (271.49)	263.38 (191.48)	ns, p=.992
MTD 15-19secs	319.72 (329.59)	348.04 (314.77)	376.81 (293.64)	ns, p=.787

TDC=Typically Developing Controls, CD=Conduct Disorder, ADHD=Attention-Deficit Hyperactivity Disorder. IQ=Intelligence Quotient, LL%=percentage of larger later reward, MTD=Mean Total Duration, ns=not significant.

5.3.1 MIDA

5.3.1.1 TDC versus CD

There was an effect of group on the choice of the SS option [F (1, 79) = 4.72, p=.03, η_p^2 = .056]. The CD group displayed more impulsive choices by choosing the SS reward more than the TDC group. However, once IQ was entered as a covariate in the analysis, the group difference reduced to a trend level and the effect size was smaller [F (1, 78) =3.30, p=.07, η_p^2 = .041].

5.3.1.2 TDC versus CD-/+ADHD

There was a trend towards a group effect on the probability of choosing the SS option [F (2, 78) =2.2.54, p=.086, η_p^2 = .061]. The CD-ADHD group chose the SS choice significantly more than the TDC group [F (2, 78) =4.60, p=.035]. The CD-ADHD and CD+ADHD groups did not differ from each other [F (2, 78) =0.40, p=.530]. No significant difference was observed between the CD+ADHD and TDC groups [F (2, 78) =2.53, p=.116].

5.3.2 DeFT

5.3.2.1 TDC versus CD

There were no group effects on task performance in the 1-4sec time bin [F (1, 79) = 0.015, p= .902, η_p^2 = .000] or the 15-19sec time bin [F (1, 79) = 0.379, p= .540, η_p^2 = .005]. The results from the repeated measures ANOVA indicated no group effects on the levels of expressed delay-related frustration [F (1, 79) = 0.168, p= .683, η_p^2 = .002]. There was also no significant TimeBin x Group interaction [F (1, 79) = 0.845, p= .361, η_p^2 = .011] (Figure 5.3).

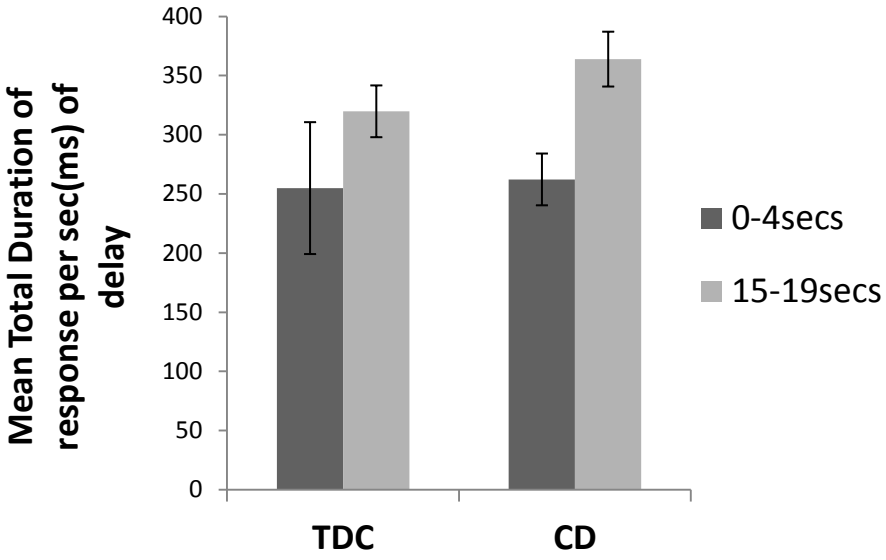

Figure 5.3. Mean Total Duration of response for the two groups at the shortest and longest delay time intervals in the DeFT task. Note. The bars demonstrate mean values whereas the error bars show ±1 standard error of the mean. CD=Conduct Disorder, DeFT=Delay Frustration Task, TDC=Typically Developing Controls.

5.3.2.2 TDC versus CD-/+ADHD

There were no group effects on task performance during the 1-4sec time bin [F (2, 78) = 0.01, p= .992, η_p^2 = .000] or the 15-19sec time bin [F (2, 78) = 0.240, p= .787, η_p^2 = .006]. The results from the repeated measures ANOVA indicated no group effects on the levels of the expressed delayed-related frustration [F (2, 78) = 0.103, p= .902, η_p^2 = .003]. There was also no significant TimeBin x Group interaction [F (2, 78) = 0.559, p= .574, η_p^2 = .014] (Figure 5.4).

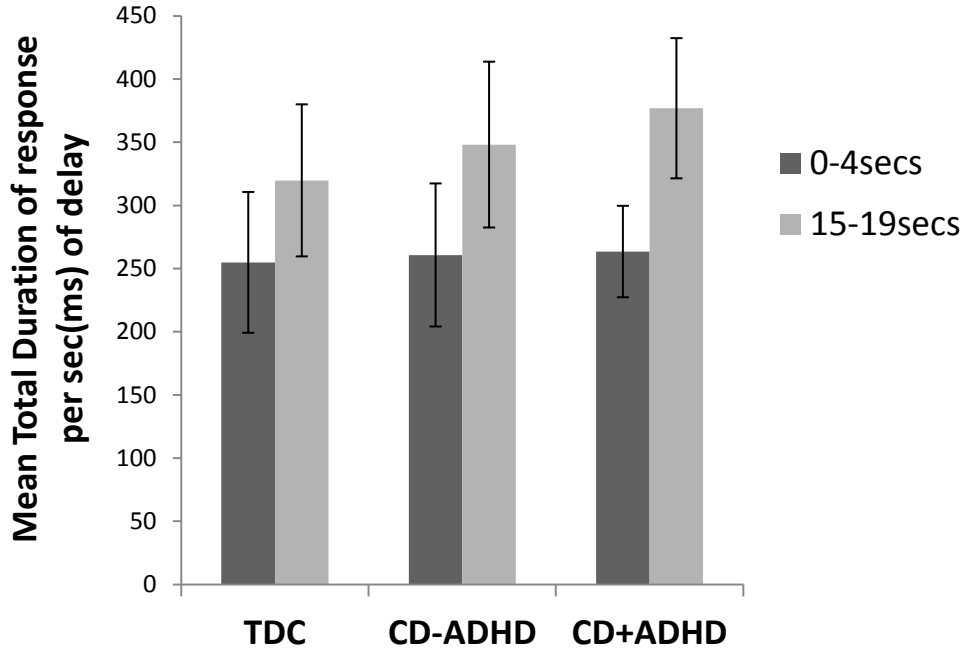

Figure 5.4. Effect of time bins on the Mean Total Duration between the three groups

Note. The bars demonstrate mean values whereas the error bars show ±1 standard error of the mean. CD=Conduct Disorder, ADHD=Attention Deficit/Hyperactivity Disorder, DeFT=Delay Frustration Task, TDC=Typically Developing Controls.

5.3.3 Relationship between the outcome measures of the two tasks

Correlational analyses showed no significant relationship between the DVs from the two tasks (table 5.3). This indicates that performance in the two tasks shared no common variance and the task variables were independent of each other. As might have been expected, there was a strong positive correlation between the MTD for the first (1-4 secs) and last (15-19 secs) time bin in the DeFT, indicating that the higher the delay-related frustration at the beginning of the delay period, the higher it was towards the end. Furthermore, IQ did not correlate with any of the outcome measures. This implies that task performance was not affected by cognitive ability.

Table 5.3. Correlation Matrix between the outcome measures of the MIDA and DeFT.

	1	2	3
1. IQ			
2. SS %	-.131		
3. MTD 1-4secs	-.122	-.004	
4. MTD 15-19secs	-.107	.029	.829**

**p<0.01, SS=probability of smaller sooner, MTD=Mean Total Duration, MIDA= Maudsley’s Index of Delay Aversion, DeFT=Delay Frustration Task, IQ=Intelligence Quotient.

5.4 Discussion

Moving on from the focus on "cool" EF in chapter four, in this chapter we used an impulsive choice task – the MIDA - and a delay-related frustration task to investigate whether adolescents with CD show "hot" EF deficits compared to typically developing controls (TDC). In order to explore the impact of comorbid ADHD, we divided the CD sample into CD-ADHD and CD+ADHD subgroups. Below, we discuss the four main findings to emerge from these analyses.

First, on the MIDA task the CD group, as a whole, showed a significantly stronger preference for the SS choice in the DA task. This is the first time that an impulsive choice task such as the MIDA has been used in a sample of adolescents with "pure" CD. The majority of the studies have employed classical Temporal Discounting (TD) tasks to investigate the impact of delay reward-related motivational processes.

Second, the results indicated that impulsive choice was not driven by ADHD - when we divided the CD group into CD-ADHD and CD+ADHD subgroups, the CD-ADHD adolescents still displayed elevated levels of SS choices. This is quite a striking finding as there is a large body of evidence linking ADHD to impulsive choice. However, our findings are consistent to those reported by Kuntsi et al. (2001), who found that differences on the MIDA task between hyperactive and non-hyperactive children were fully explained by co-occurring DBDs. However, as previously mentioned not many studies have actually controlled for comorbid CD more fully.

Third, the effects seen on the MIDA did not extend to the DEFT. The two groups displayed similar levels of frustration levels in the first time period, while the CD groups levels of responding was slightly, though non-significantly higher in the last time period. Splitting up the group into ADHD+ and ADHD- did not change the pattern of results. Based on these data it appears that frustration induced by unexpected delay is related neither to CD nor to comorbid ADHD in the context of CD. This runs counter to both the previous data on the DeFT with children with ADHD (Sonuga-Barke et al., 2010) but also the more general notion that children with ADHD+ disruptive behaviour disorders (DBDs) are centrally characterised by poor affect regulation (Nigg et al., 2004). One possible explanation for our null finding is that adolescents with CD have a higher frustration tolerance than we expected. In addition, we cannot exclude the possibility that the CD participants in this sample were unconcerned about finishing the primary activity that was interrupted (i.e., mathematics questions).

Fourth, given the different pattern of group differences for the MIDA and DeFT, it was not surprising that the outcomes from the two tasks were not correlated. This contradicts the result from the study by Bitsakou et al. (2009) where they reported a significant correlation between DeFT and MIDA performance, albeit this was small in size. However, previous studies have provided inconclusive evidence about the relationship between emotional self-regulation and delay-related decision making (i.e., on temporal discounting tasks). Critchfield and Kollins (2001) refer to TD as the decrease of subjective reward value based on the increase of delay in receiving it. The TD process can be measured by asking participants to choose between immediate rewards or delayed rewards of greater value. The Delay Aversion theory has been described as an influential explanatory model for TD in ADHD (Utsumi, Miranda, & Muszkat, 2016). In addition, some studies have tried to connect TD and emotional self-regulation, since the ability to delay gratification requires more emotional resources and a mechanism enabling an individual to look beyond the immediate situation and weigh up possible consequences of impulsive behaviour (Baumeister & Vohs, 2016; Baumeister et al., 2010). Our results indicated that even though CD adolescents display similar levels of frustration level when confronted with delay they seem to prefer instant gratification over larger delayed reward.

Fifth, the effects of IQ on task performance appeared to be minimal, as the group differences in the MIDA task persisted when adjusting for this variable. This finding is important as it highlights further the relationship between impulsive choice and CD. In the previous chapter, IQ also moderated the effect of CD and comorbid ADHD. In fact the IQ effects were larger for the Eriksen-Flanker and the N-Back tasks which were measuring "cool" EFs.

In conclusion, if we consider the findings from chapters 4 and 5 together, we can see that our results seem to support the idea that CD is related to "hot" EF deficits, whereas ADHD, at least as it presents in the context of comorbid CD, is related to deficits in "cool" EF.

Strengths and limitations

The findings of the present study should be interpreted in light of several limitations. First, group differences in IQ may be responsible, to a degree, for the difference in IC between CD and TDC groups. However, in general, CD is associated with lower IQ in many studies and group differences have been found even when the samples are IQ matched. Second, even though the MIDA showed group differences in the present sample and age group, there is a possibility that the current version is not wholly appropriate for use with older children. In

addition, participants gained only 20p per round if they chose the LL option (compared with 10p if they chose the SS). Adolescents with CD may not value the larger outcome highly enough to wait for the LL choice – i.e., the total amount was not perceived as rewarding/motivating enough to be worth the extra wait. Third, the DeFT was developed based on the idea that individuals who are delay intolerant will also exhibit elevated frustration levels caused by unexpected delays that they encounter whilst trying to complete a specific activity (Bitsakou et al., 2006). However, this is the first time that the specific task has been tested in individuals with CD, and even though it has good construct validity the DeFT task may not be suitable for this particular clinical group (or age group) in terms of how the frustration levels are measured. Nevertheless, our study provides the basis for future investigation in delay-related frustration. Future studies could video record the participants’ behaviour and code for other signs of frustration, e.g. sighing, swearing, as well as the duration of button presses.

5.5 Summary

In contrast with the findings relating to “cool” EF reported in chapter 4, we found that impulsive choice – a marker of “hot” EF problems - was driven by CD, independently of comorbid ADHD. Taking the two chapters together, our data are consistent with a model in which CD is regarded as a “hot” EF and ADHD a “cool” EF deficit disorder.

Chapter 6 Emotion recognition deficits in adolescents with conduct disorder with and without comorbid attention deficit/hyperactivity disorder

6.1 Introduction

In Chapters 4 and 5 we investigated "cold" and "hot" EF deficits in adolescents with Conduct Disorder (CD), and tested whether these were moderated by the presence of comorbid attention-deficit/hyperactivity disorder (ADHD) moderated task performance. In brief, we found that adolescents with CD displayed elevated levels of both "cold" (interference control and working memory deficits) and "hot" (impulsive choice) EF deficits compared with TDC. Furthermore, the "cold" EF deficits were related to comorbid ADHD while "hot" EF deficits were present whether or not comorbid ADHD was present. This chapter will extend the focus to social cognition in general, with a specific emphasis on trying to identify whether CD is related to deficits in the recognition of emotional facial expressions and, furthermore, whether such effects are linked specifically to the presence of comorbid ADHD.

Discriminating and recognising facial expressions play an integral role in human social interaction and socialisation. The skill to recognise emotional expressions involves a cognitive interpretation process of other individuals' expressive signal (Hoehl, 2009). Studies have shown that distress-related cues i.e. fearful expressions, inhibit antisocial behaviours (Blair, 2001), while impairments in processing these distress-related cues are quite common within antisocial populations (Nichols, 2001; Price, Gardner, & Erickson, 2004).

Marsh and Blair (2008) reviewed twenty studies that included populations characterised as psychopathic, conduct disordered, aggressive, unsocialised, abusive and criminal. The results of this meta-analysis indicated specific deficits in fear recognition in CD. In fact, the findings for a deficit in recognising negative emotions are quite robust especially for fear, in this regard (Dadds et al., 2006; Muñoz, 2009).Within children and adolescent CD populations, recent research has shown a more general pattern of emotion recognition impairments including surprise, anger and disgust (Fairchild et al., 2009, 2010; Short et al., 2016; Sully et al., 2015a) and to a lesser extent recognition impairments in sadness (Fairchild et al., 2010).

Furthermore callous-unemotional (CU) traits, commonly seen in groups with CD, (Bowen et al., 2014; Kimonis, Frick, Fazekas, & Loney, 2006; Leist & Dadds, 2009), exacerbate these problems, especially in relation to fear, (Pardini & Frick, 2013) though this contribution is less consistent in other facial expressions (Fairchild et al., 2009, 2010).

ADHD has also been linked to emotion recognition impairments in fear, disgust and surprise while the recognition of happiness, anger and sadness seems to be relatively intact (Boakes, Chapman, Houghton, & West, 2008). In a recent study by Aspan et al (2014), adolescents with ADHD were more sensitive in the recognition of disgust, worse in the recognition of fear, while they showed a tendency for impairment in the recognition of sadness. However, studies on facial emotion recognition in comorbid cases of CD and ADHD have been few in number and inconclusive even though research has suggested that emotion recognition deficits in children as young as four is linked to externalising behaviour problems (Chronaki et al., 2015).

The present study is the first study to explicitly investigate the links between adolescents diagnosed with "pure" CD and emotion recognition deficits and also whether comorbid ADHD either exacerbates or ameliorates these effects. We hypothesised that adolescents with CD would present with impaired recognition of negative emotions. Our hypothesis for the comorbid group was somewhat more speculative and is based on the idea that ADHD is connected to deficits in emotion processing. For this reason we anticipated that ADHD would exacerbate the emotion recognition problems in CD. In addition, given the strong relationship between CU traits and CD, we also performed a regression analysis to investigate the combined impact of both CU traits and ADHD on emotion recognition in CD. Little is known about the relationship between CU traits and ADHD and their combined impact on adolescents with CD, highlighting the need for more research in this area.

6.2 Method

6.2.1 Sample

Please see Chapter 3 for sample characteristics.

Specific to the current task, participants who scored below 41 on the Benton Facial Recognition Test (BFRT; Benton, Hamsher, Varney, & Spreen, 1983) were excluded from the analysis. This led to the removal of 3 TDC participants, 1 CD-ADHD participant and 2 CD+ADHD participants from the data analysis of the emotion recognition task. In total, 75 out of 81 participants' data were included in the statistical analysis.

6.2.2 Measures

6.2.2.1 Screening for Facial Identity Perception

As previously mentioned, we used the BFRT (Benton et al., 1983) to screen for basic face processing deficits. During the BFRT, participants were presented with a target face and are asked to identify the target in an array of three, and after a few pages, six faces. The faces vary in illumination (tonal variations of black and white) and/or head orientation (left/right). The total score can range from 0 to 54, with scores below 41 indicating clinically significant impairments in face recognition. In the present study, six participants (three in the control group, one in the CD group and two in the CD+ADHD group) scored lower than 41 and were excluded from the analyses of data from the facial emotion recognition task.

6.2.2.2 Facial Emotion Recognition

We used the Emotion Hexagon Task developed by Calder et al. (1996). This is a computerised task which measures the accuracy of facial emotional expression recognition. Sequences of facial expressions taken from the Ekman and Friesen (1975) facial affect series were blended across continua that spanned the following six expression pairs: happiness-surprise, surprise-fear, fear-sadness, sadness-disgust, disgust-anger and anger-happiness. Images of the two emotions were morphed across five ratios containing the following percentages: 90% - 10%, 70%-30%, 50%-50%, 30%-70%, and 10% - 90%. Figure 6.1 depicts the six expression continua in pairs.

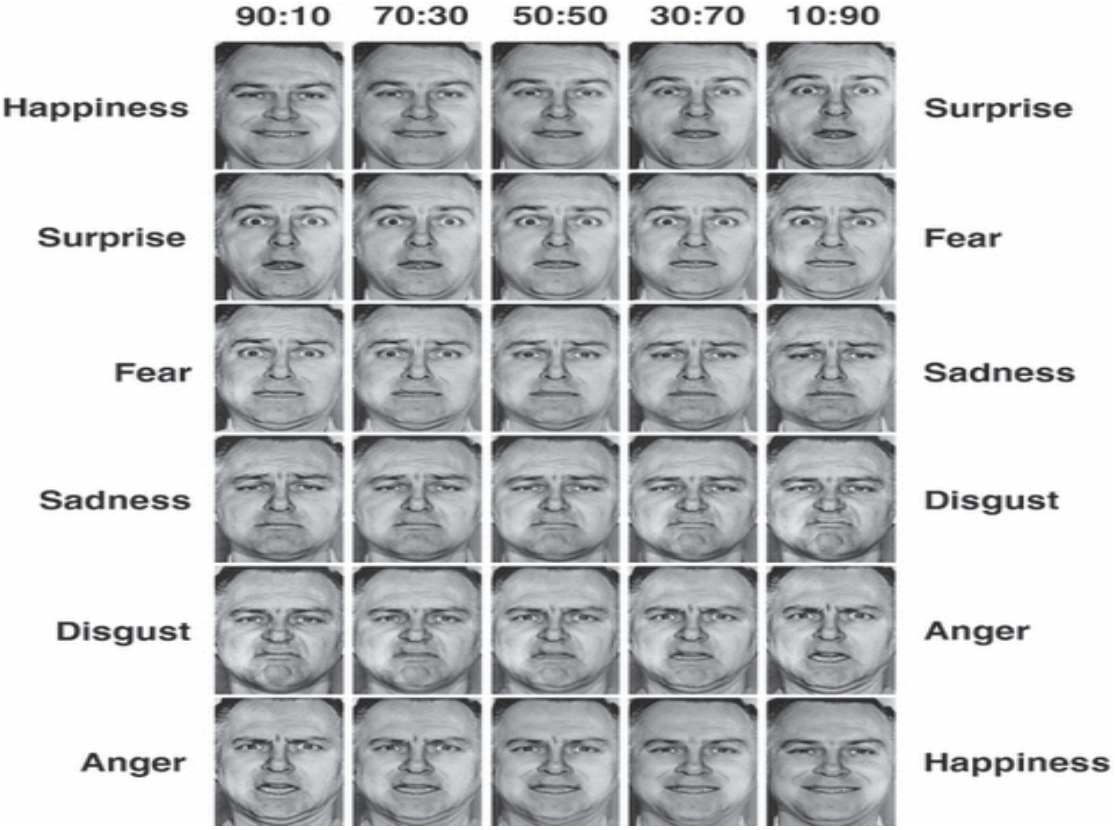

Figure 6.1. Facial expression continua used in the Emotion Hexagon Task.

The photographs of the faces were in black and white. The morphed faces were presented individually in the centre of the computer monitor in a pseudo-random order. Participants were asked to label the morphed facial expression by clicking on the emotion label they felt was the correct one using the mouse. Labels for each of the six different emotion options were displayed along the bottom of the screen. Each facial expression was presented for 3sec and participants were asked to name the correct expression by selecting the corresponding emotion label, i.e. happiness, surprise, fear, sadness, disgust, or anger. Emotion labels were presented until a response was made. The order of the labels was pseudo-randomised across six blocks to reduce response biases. Each block contained thirty faces; 24 faces where the emotion was presented at 90% or 70% (four for each emotion) and six faces which were 50-50% morphs. There was a 2sec inter-trial interval, where participants viewed a blank screen. In total, the task contained 165 trials, including an initial practice block of 15 trials. Only trials where the emotion was presented at 90% or70% were analysed i.e.120 trials in total, 20 for each of the six emotions. The 50%-50% morphed faces were not analysed as there is no "objective" correct answer. The task was administered using the E Prime software (version 2.0).

6.2.2.3 Callous-Unemotional traits

Please see chapter 3 for details about the Inventory of Callous-Unemotional traits (ICU; (Frick, 2004b). Only 73 out of 75 participants that completed this task completed the self-reported inventory.

6.2.3 Procedure

Please see Chapter 3. Participants attended a testing session at the University of Southampton, during which the present task and the remainder of assessments presented in Chapter 3 were administered.

6.2.4 Data Analysis

Statistical analyses were performed using the Statistical Package for the Social Sciences, Version 21 (SPSS Inc., Chicago, IL). For the analysis we followed the same two-stage process as in the previous chapters. During the first stage we compared task performance between the controls (n=27) and all the participants with a CD diagnosis (n=48). In the second stage, we divided the CD participants into two groups: CD-ADHD (n=22) and CD+ADHD (n=26). The Emotion Hexagon data were not normally distributed; therefore non-parametric analysis was performed. Kruskal-Wallis H tests were used to examine group differences for each emotion separately. Mann-Whitney U tests were used as post-hoc tests for group comparisons; p value thresholding for multiple comparisons with Bonferroni correction was also conducted (.05/6, p =.008). Effect sizes were calculated manually and reported as 'r' (small ≥ 0.1, medium ≥ 0.3, large ≥ 0.5; (Cohen, 1988c). Finally, in order to investigate the relationship between CU traits, ADHD and levels of emotion recognition accuracy, multiple linear regression analysis within the CD sample only, was employed. We performed three hierarchical linear regression analyses for the emotions that had been significantly associated with group effects, within the combined CD groups. In the first step, we added CU traits and ADHD status (yes+, no-) as predictors. In the second step, we added a CU*ADHD status interaction term. IQ did not correlate significantly with recognition scores within the combined CD groups; therefore we did not include it as a predictor in the regression models.

6.3 Results

6.3.1 TDC versus CD

Please see Table 6.1 for the characteristics of the sub-sample that was included in the analysis of this task. The CD+ADHD group had significantly higher levels of CU traits than the other two groups, whereas both the CD sub-groups had lower IQ than the TDC group.

Table 6.1. Sub-sample characteristics in the Emotion Hexagon task

	TDC[a] (N=27)	**CD[b] (N=48)**	**Comparison**
	Mean (SD)	**Mean (SD)**	
Age	16.32 (1.24)	16.60 (1.34)	ns, p=.374
IQ Estimate	105.11 (9.17)	94.33 (6.77)	a>b, p<.001
BFRT	46.74 (2.80)	46.35 (3.27)	ns, p=.607
CU traits	22.37 (7.62)	30.22 (9.80)[+]	b>a, p=.001

+ N=46. TDC=Typically Developing Controls, CD=Conduct Disorder, IQ=Intelligence Quotient, BFRT=Benton Facial Recognition Test, CU= Callous-Unemotional, ns=not significant.

Relative to TDC, CD participants were less accurate in identifying anger (U=388.50, p=.004, r=-0.51), disgust (U=438.50, p=.02, r=-0.41), fear (U=358.00, p=.035, r=-0.38), sadness (U=460.00, p=.030, r=-0.39), and surprise (U=356.50, p=.001, r=-0.58). No group differences were observed in the identification of happiness (U=542.00, p=.166, r=-0.25) (figure 6.2).

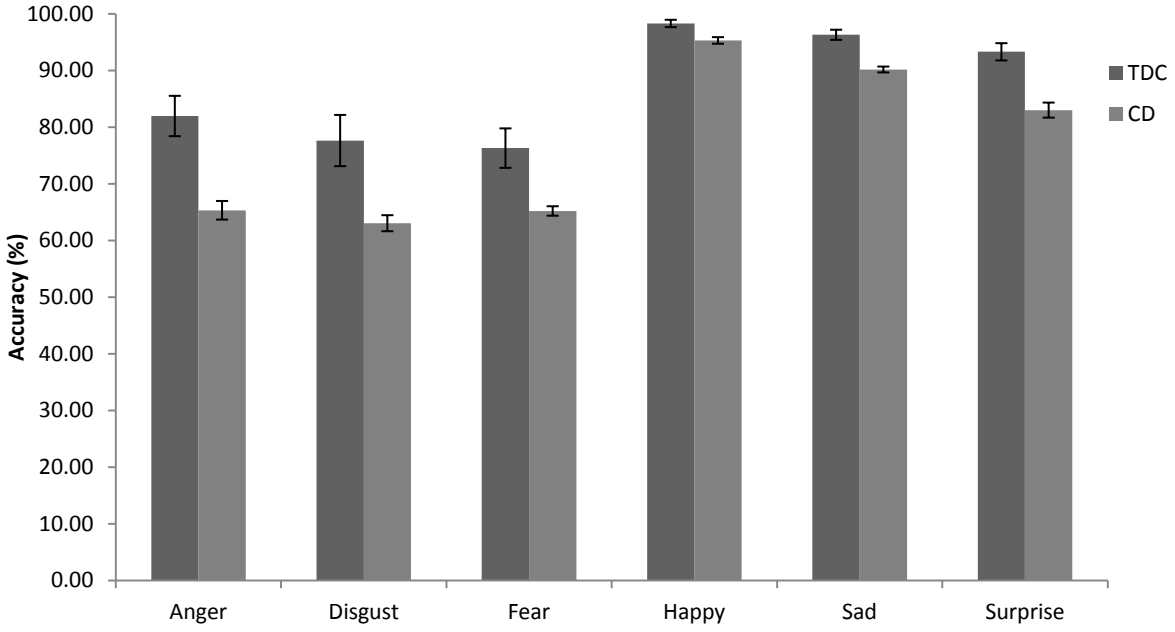

Figure 6.2. Facial emotion recognition accuracy by experimental group. Note. Error bars show standard error of the mean. TDC=Typically Developing Controls, CD = Conduct Disorder.

6.3.2 TDC versus CD-/+ADHD

Please see Table 6.2 for the characteristics of the sub-sample that participated in this task.

Table 6.2. Characteristics of the sub-sample in the Emotion Hexagon task

	TDC[a] (N=27)	**CD-ADHD[b] (N=22)**	**CD+ADHD[c] (N=26)**	**Comparison**
	Mean (SD)	**Mean (SD)**	**Mean (SD)**	
Age	16.32 (1.24)	16.98 (1.15)	16.28 (1.42)	ns, p=.119
IQ estimate	105.11 (9.17)	94.59 (6.35)	94.12 (7.23)	a>b,c, p<.001
BFRT	46.74 (2.80)	46.50 (3.28)	46.23 (3.33)	ns, p=.839
CU traits	22.37 (7.62)	26.18 (8.31)	33.92 (9.74)$^{+}$	c>a,b, p<.001

TDC=Typically Developing Controls, CD=Conduct Disorder, ADHD= Attention Deficit/ Hyperactivity Disorder, CD-ADHD=Conduct Disorder without Attention Deficit/Hyperactivity Disorder, CD+ADHD=Conduct Disorder with Attention Deficit Hyperactivity Disorder, IQ=Intelligence Quotient, BFRT=Benton Facial Recognition Test, CU= Callous-Unemotional, ns=not significant, +N=24.

There were significant group effects for anger [H (2) = 12.735, p=.002], disgust [H (2) = 6.725, p=.035] and surprise [H (2) = 17.981, p<.001]. There were strong trends towards significance for fear [H (2) =5.800, p=.055] and happiness [H (2) =5.818, p=.055]. No group effect was observed for sadness [H (2) = 5.102, p=.078] (figure 6.3).

Relative to TDC, CD+ADHD participants showed impaired recognition of anger (U=159.50, p=.001, r=-0.61), disgust (U=220.50, p=.020, r=-0.41) and surprise (U=124.00, p=<.001, r=-0.73). All of these effects survived correction for multiple comparisons with medium to large effect sizes. The trend for statistical significance in the main analysis for fear and happiness, resulted in significant group differences in post-hoc analysis between the TDC and the CD+ADHD groups with small effect sizes [fear: U=220.50, p=.020, r=-0.41; happiness: U=249.00, p=.036, r=-0.37]; CD+ADHD participants were less accurate than TDC.

There were no significant differences between TDC and CD-ADHD participants in any of the six emotions (p values ranged from 0.11 to 0.92). Relative to CD-ADHD, CD+ADHD participants showed impaired recognition of anger (U=179.00, p=.026, r=-0.39) and surprise (U=155.50, p=.006, r=-0.48). However, only the significant result in surprise survived correction for multiple comparisons. No differences were observed for disgust (p=.149), fear (p=.219) or sadness (p=.489). There was a non-significant trend for happiness (U=205.00, p=.056, r=-0.34) with CD+ADHD performing worse than CD-ADHD.

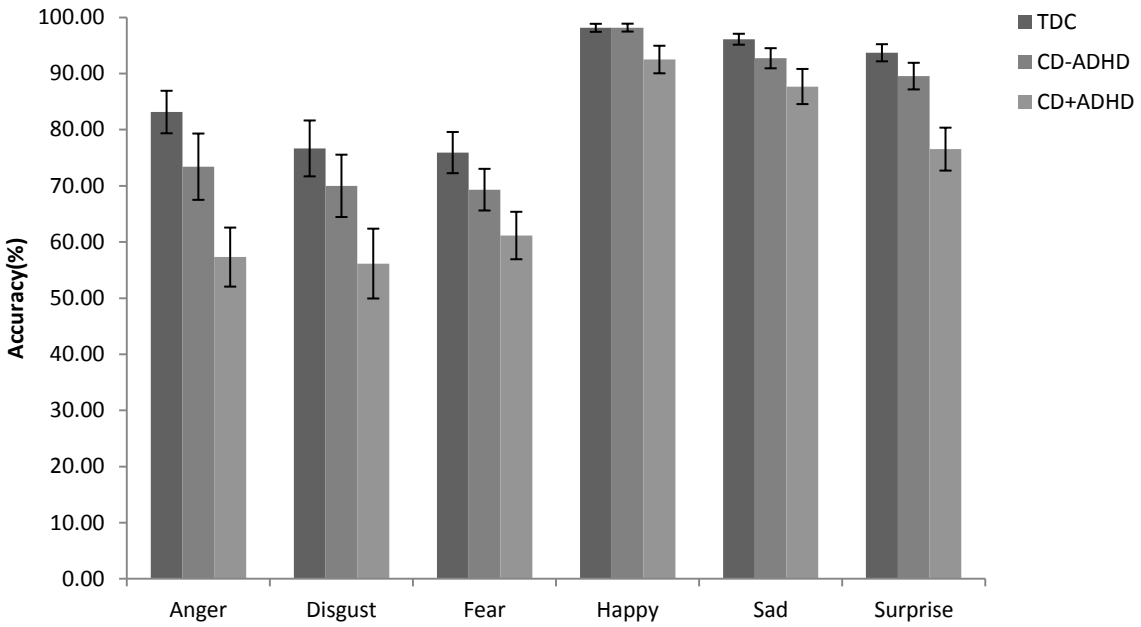

Figure 6.3. Facial emotion recognition accuracy by experimental group. Note. Error bars show standard error of the mean. TDC=Typically Developing Controls, CD = Conduct Disorder, ADHD=Attention Deficit Hyperactivity Disorder.

6.3.3 Callous-Unemotional traits

CU traits and ADHD status independently explained a significant amount of the variance in anger recognition scores ($F (2, 45) = 7.99$, $p<. 01$, adjusted $R^2 = 0.24$). Adding the CU traits*ADHD status interaction term to the model resulted in a poorer model fit ($F (3, 45) = 5.34$, $p = .003$, adjusted $R^2=0.23$). CU traits were significant predictors of anger recognition in both models. CU traits and ADHD status both explained a significant amount of the variance in disgust recognition scores ($F (2, 45) = 3.81$, $p=.03$, adjusted $R^2 = 0.11$). Adding the CU traits*ADHD status interaction term to the model resulted in a poorer model fit ($F (3, 45) = 2.49$, $p=.07$, adjusted $R^2 = 0.09$). CU traits were significant predictors of disgust recognition in both models. CU traits and ADHD status explained a significant amount of the variance in surprise recognition scores ($F (2, 45) = 5.66$, $p = .007$, adjusted $R^2 = 0.17$). Adding the CU traits*ADHD status interaction term to the model resulted in a poorer model fit ($F (3, 45) = 3.86$, $p =.02$, adjusted $R^2 = 0.16$). ADHD status was a significant predictor of surprise recognition in both models: the presence of ADHD resulted in poorer recognition of surprise. CU traits did not significantly predict surprise recognition in either model (Table 6.3).

Table 6.3 Results of the multiple linear regression analyses

	Anger Recognition Accuracy		Disgust Recognition Accuracy		Surprise Recognition Accuracy	
	Beta$^{\beta}$	**R^2**	**Beta$^{\beta}$**	**R^2**	**Beta$^{\beta}$**	**R^2**
Step 1		.271**		.151*		.208**
CU traits	-.475**		-.327*		-.229	
ADHD	-.096		-.116		-.314*	
Step 2		.276**		.151		.160*
CU traits	-.465**		-.328*		-.241	
ADHD	-.104		-.115		-.305*	
CU traits*ADHD	-.074		.009		.089	

ADHD=Attention Deficit Hyperactivity Disorder, CU= Callous-Unemotional. *p<.05, **p<.01, β=standardised coefficient

6.4 Discussion

We used a facial emotion recognition task to investigate emotion processing deficits in adolescents with CD compared to typically developing controls (TDC) and then the impact of comorbid ADHD. Lastly, given the extensive overlap between CD and CU traits, and between CD and ADHD we also tried to examine the contribution of CU traits to emotion recognition deficits. We report three main findings.

First, adolescents with CD were less accurate in recognising five out of the six emotional facial expressions that were included in the task - anger, disgust, fear, sadness and surprise. This result confirmed our hypothesis and is in line with previous studies that have used the same task in a CD population (Fairchild et al., 2010; Sully et al., 2015a). Contrary to the previous two studies, our study did not show any group differences in happiness recognition between CD and TDC. One possibility is that recognition deficits are less evident for expressions that possess identifiable markers such as wide, upturned corners of the mouth. In addition, our findings support the idea that children with CD display a global deficit in facial emotion recognition rather than a deficit in specific emotions only. However, in our study not all emotions survived corrections for multiple comparisons (please see chapter 8 for a detailed discussion on this).

Second, when the CD group was divided to those with and those without a diagnosis of comorbid ADHD, it became apparent that ADHD was driving most of the differences in emotion recognition deficits in the CD group. Adolescents with CD+ADHD showed significantly impaired emotion recognition of anger, disgust and surprise in relation to TDC, and anger and surprise compared to adolescents with CD but no ADHD. Again there were no differences between the three groups in the recognition of happiness. Against expectation, adolescents with CD but no ADHD did not show elevated levels of emotion recognition deficits compared to TDC. This was an important, unexpected finding and one that highlights the impact of comorbid ADHD in emotion processing in CD.

Emotion dysregulation in ADHD has been suggested to arise from deficits in orienting toward, recognizing, and/or allocating attention to emotional stimuli (Shaw, Stringaris, Nigg, & Leibenluft, 2014). Boys with ADHD have been found to exhibit significant difficulties in interpreting disgust and fear and at some extent surprise when compared to controls (Boakes et al., 2008). The same study also found that boys with ADHD and controls had the same ability recognising happiness, anger and sadness. The authors connected the emotion recognition deficits to comorbid conduct problems and basal ganglia irregularities in form or function. In addition, disgust difficulties have also been found in CD populations (Bowen et al., 2014; Fairchild et al., 2009, 2010). This contradicts our finding where CD-ADHD had the same level of disgust recognition as controls. In the case of the facial expression of surprise, a possible explanation for the low accuracy score in CD+ADHD may be that adolescents in that group perceived it as a different emotion. Surprise can have either positive or negative valence and it can be classed as a “transitory” response that often precedes the onset of fear (Boakes et al., 2008).

Overall, our results indicated that ADHD significantly exacerbates the non-significant levels of recognition deficits in anger, disgust and surprise found associated with CD. In studies where the impact of comorbid anxiety was investigated results showed that adolescents with anxiety alone and anxiety+CD performed the same or better than controls in emotion recognition. The presence of anxiety played a protective role by counteracting the effects of CD (Short et al., 2016). In our study, comorbid ADHD appeared to have the opposite effect in adolescents with CD. Future research should aim to distinguish the contribution of externalising and internalising comorbid disorders in the overall behavioural expression of CD.

Third, our regression analyses revealed the unique contribution of CU traits over and above the effects of ADHD to the observed emotion recognition deficits within the CD group. CU traits were significant independent predictors of anger and disgust recognition. Interestingly, ADHD status was not a significant predictor of either anger or disgust recognition. This suggests that the poorer anger and disgust recognition seen in the CD+ADHD group might have been driven by increased CU traits. CU traits did not significantly predict surprise recognition. This suggests that the poorer surprise recognition seen in the CD+ADHD group was driven by the presence of ADHD, and this was an independent predictor of surprise recognition. The impact of CU traits in predicting impairments in anger and disgust recognition is in line with empirical evidence (Dawel, O'Kearney, McKone, & Palermo, 2012), even though there has also been evidence for no significant differences in emotion recognition between adolescents with CD with low vs high CU traits (Sully et al., 2015a). Inconsistent findings in this area are not uncommon (Dadds et al., 2006; Lui, Barry, & Sacco, 2016) and this has been attributed, at least in part, to differences in the operationalisation of psychopathic/CU traits.

The current results when combined with those from the previous chapters highlights the role of comorbid ADHD as a potentially important driver of both cognitive and social-cognition deficits. While that was expected with regard to the "cool" EF this was not predicted with regard to emotional deficits – which have been previously thought to be a possible causal factor in the development of CD.

Limitations

The findings of this study should be interpreted in light of several limitations. First, our sample size was not sufficiently large to detect small differences in emotion recognition that may have existed between our CD-ADHD and CD+ADHD groups. Second, the facial expressions in the task were only presented at high intensities i.e. 70% or 90% intensity. This may have resulted in ceiling effects reducing the sensitivity of the task (Bowen et al., 2014). However, one should note that with the exception of happiness, group differences were detected – a finding that is not consistent with such a view. Third, our study used static facial expressions which may be classified as less ecologically valid than dynamic facial expressions. However, the use of the Emotion Hexagon task has been used in previous studies with children and adolescents with CD and has been classified as a valid emotion recognition task. Fourth, this study used only one measure of emotion recognition. Future

studies should employ additional tasks perhaps including tasks of vocal emotion processing (Chronaki et al., 2015) in order for a more comprehensive understanding regarding emotion recognition deficits in CD to be formed.

6.5 Summary

This is the first study to explicitly investigate the role of comorbid ADHD in emotion recognition deficits in CD. Against expectation and in contradiction to the prior literature we found that a broad pattern of facial emotion recognition deficits were limited to those individuals with CD and ADHD. On top of that CU traits made independent contributions to certain emotions.

Chapter 7 Higher level Theory of Mind (ToM) in adolescents with conduct disorder with and without comorbid attention-deficit/hyperactivity disorder: A study using the Director's Task.

7.1 Introduction

In Chapters 1 and 2 we highlighted the fact that the vast majority of studies of social cognition in CD focus on facial emotion recognition (Short et al., 2016; Sully, Sonuga-Barke, & Fairchild, 2015b) and affective empathy deficits (Happé & Frith, 1996; Lui et al., 2016; Sebastian et al., 2012)Difficulties in recognising facial affect are considered a cognitive marker of CD (Marsh & Blair, 2008). In Chapter 6 we showed that CD was related to facial emotion recognition deficits, although these effects were largely explained by the presence of comorbid ADHD and/or Callous-Unemotional (CU) traits. In this chapter we focus on a different aspect of social cognition, equally important for effective social functioning – Theory of Mind (ToM). ToM has been defined as the ability to understand and attribute beliefs, thoughts, desires, intentions and feelings to both ourselves and other people (Baron-Cohen, Leslie, & Frith, 1985). It is a skill that is essential for interpreting social situations and making inter-personal decisions because it allows one to take the perspective of another person – to see things from their point of view (Cavell, 1990).

Overall there is little evidence that CD is associated with ToM deficits (Dunno, Parker, Gilmour, & Skuse, 2010; Ha, Sharp, & Goodyer, 2011). This is perhaps somewhat unexpected. At first sight ToM deficits seem to be closely related to a lack of affective empathy which is a hallmark feature of CU traits – so distinguishing it during considerations of ToM in CD, which so often overlaps with CU traits, is essential. Studies in children and adolescents with conduct problems and high levels of CU traits have reported reduced affective empathy, but no deficits in ToM (Blair, 2005; Jones, Happé, Gilbert, Burnett, & Viding, 2010; Schwenck et al., 2012)This highlights the dissociation between these two social cognitive processes in CD.

In contrast, there has been some suggestion that children with ADHD have deficits in ToM. Some studies have found no differences between children with ADHD and typically developing children (TDC) (Charman, Carroll, & Sturge, 2001; Dyck, Ferguson, & Shochet, 2001; Perner, Kain, & Barchfeld, 2002); but others have reported deficits (Sodian, Hülsken, & Thoermer, 2003). Children with elevated ADHD traits have been reported to have low

levels of social perspective-taking compared to children with low levels of ADHD traits (Marton, Wiener, Rogers, Moore, & Tannock, 2008). However, the most comprehensive review of social dysfunction on ADHD (by Nijmeijer et al., 2008) interestingly, notwithstanding the lack of evidence for ToM deficits in CD and ODD, concluded that the deficits in social cognition in ADHD can be accounted for by these comorbid disorders.

However, these studies have two limitations: a) their samples included only children up to 12 years old and b) the ToM tasks used are limited to basic false-belief tasks. More generally, studies of ToM in CD and ADHD have been criticised for using a very "narrow" definition of ToM, potentially resulting in misinterpretation of the null findings (Sharp & Vanwoerden, 2014). Furthermore, no studies have used ecologically-valid paradigms to assess social perspective-taking in CD and no studies have compared adolescents with CD alone and those with comorbid ADHD. To address this limitation, in this study we employed the Director's Task (DT; Dumontheil, Apperly, & Blakemore, 2010),which measures a combination of ToM and executive functions (EFs) to explore whether CD is associated with higher level ToM skills deficits and whether these deficits are exacerbated by the presence of comorbid ADHD – because of their known EF deficits.

The DT is designed to test adolescents' ability to understand another person's perspective (the director) and inhibit their own perspective. Successful completion of the task requires both adequate mentalising and high levels of response inhibition. More specifically the DT examines social perspective-taking by presenting participants with an array of object pairs, all of which are visible to the participant but only some of which are visible to the Director. They then have to choose the one of a pair visible to both themselves and the Director and to ignore the one that is visible to only themselves. Performance in this condition is contrasted to various control conditions where either no choice between objects is required (only the object observable by both parties is present) or where the Director's perspective is not required (when the participants have to select objects based on physical features of the objects setting (e.g., a grey background). This allowed an index of the ability to take another person's perspective into account in decision making independent of other more general executive abilities.

Given the high level of social perspective-taking required, the DT is more age appropriate than standard false-belief ToM tasks - which typically-developing children can successfully pass by the age of 4 years (Wimmer & Perner, 1983). It is also more ecologically valid than

some ToM tasks given its realistic communication situation (Keysar, Barr, Balin, & Brauner, 2000; Keysar, Lin, & Barr, 2003; Symeonidou, Dumontheil, Chow, & Breheny, 2016).

We hypothesised that participants with CD will show deficits in this higher level perspective-taking when compared with a typically-developing control (TDC) group. These effects would be specific to situations where the Director's perspective needed to be taken into account to choose which object to move i.e. critical condition. In such condition individuals with CD would be less accurate and slower than TDC. This would be the case independently of ADHD. However, individuals with ADHD would show more general deficits in performance across conditions due to the EF deficits (as illustrated in chapter 4). A key dependent variable is the speed with which individuals make their choices of which objects to move – rather than whether they made the correct move. The task therefore has the potential to identify subtle differences in perspective- taking that may have remained hidden if only correct/incorrect answers were analysed. In this way a slower response to when the Director's perspective needs to be taken into account (compared to other conditions when it doesn't) would indicate a ToM deficit.

7.2 Method

7.2.1 Sample

Please see Chapter 3.

7.2.2 Measures

7.2.2.1 Assessment of Autistic Traits

We used the Autism-Spectrum Quotient (AQ) (Baron-Cohen et al., 2001) to assess the presence of autistic-spectrum traits in our sample (please see chapter 3 for a full description). All of the adolescents in our sample scored below the cut-off for the presence of clinically significant levels of autistic traits.

7.2.2.2 Experimental Task

The task was based on the one used in a previous study by Dumontheil, Apperly and Blakemore (2010) (for the specific design and task parameters, please see Apperly et al., 2010; Symeonidou et al., 2016). Participants were seated in front of a computer screen and presented with a 4x4 grid in the form of a set of shelves that contained eight different objects (see figure 7.1).

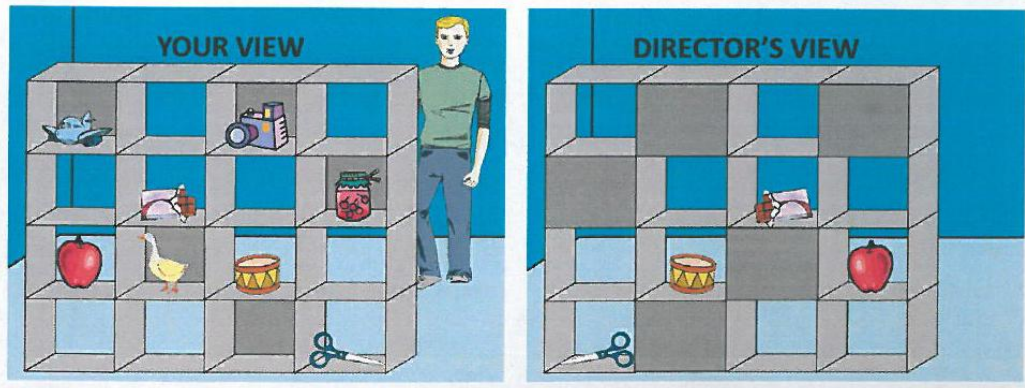

Figure 7.1. Screen panels with the instructions used to explain the task to the participants. The left grid is shown from the participant's point of view, while the right grid is shown from the Director's point of view.

The task had two conditions (Director, No-Director) and two trial types (Control, Experimental) (see Figure 7.2). The task also contained "filler" trials. For these trials,

participants were instructed to move objects in clear slots only. The objects were present in the grid only once i.e. there was only one ball in contrast with the control and experimental trials where there were 2 or 3 balls to choose from respectively.

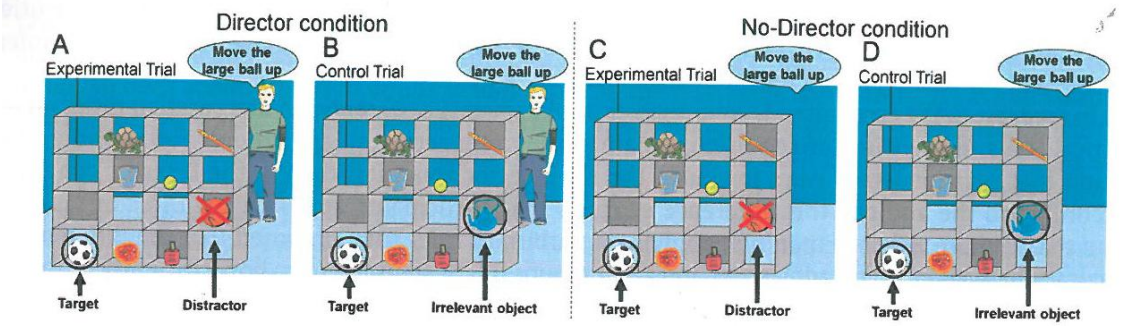

Figure 7.2. Panels A, B, C, and D show examples of the task stimuli. In A and B the Director is asking the participant to "move the large ball up". In the Experimental trial, the participant had to take the Director's perspective in order to move the right object. In the control trial, the distractor was replaced by an irrelevant object. In C and D, which depict the No-Director condition, the participant was told that the instructions about moving the objects do not refer to items located in slots with grey background. That applied for both the Experimental and Control trials. Adapted from "Development of online use of theory of mind during adolescence: and eye-tracking study" by I. Symeonidou, I. Dumontheil, W-Y. Chow, and R. Breheny, 2016, Journal of Experimental Child Psychology, 149,p.81-97. Copyright 2016 Elsevier Inc.

Director condition (Figure 7.2; A&B): a male figure (the Director) was standing behind the shelves, viewing the set of shelves from behind (figure 7.1a). Participants were given headphones and were asked to listen to the Director's instructions and move one of the objects in the grid, e.g. "move the scissors left". Using a mouse, participants had to click on the object they thought the director was referring to and drag it to the instructed position. In each of the trials, five objects were **not** visible to the Director (those in the slots with dark grey backgrounds).

No-Director condition (Figure 7.2; C&D): participants were instructed that the Director had gone but they would still need to move objects in the grid. However, the instructions referred only to the objects in the clear slots, and the objects in the slots with the dark grey

background had to be ignored. Consequently, the No-Director trials were identical to the Director trials, except that instead of taking the Director's perspective into account when moving objects, the participants had to ignore all of the objects in the occluded slots. Therefore, the correct responses were the same as in the Director condition.

The difference between the Director and No-Director conditions was that in the former, participants had to take into account that the director was not able to see certain objects, whereas in the latter participants were given an explicit rule to facilitate performance. Both conditions required the use of a variety of executive functions. The only difference was that in the Director condition participants also required perspective-taking ability, which is the ability to mentally represent what another person can actually see (Flavell, Everett, Croft, & Flavell, 1981).

The order of the experimental, control and filler trials was counterbalanced between the participants in both conditions. The Director condition was always presented before the No-Director condition. This way the participants were prevented from applying the rules that were given in the No-Director condition to the Director condition (as per Dumontheil et al., 2010).

Two sets of eight different grid patterns were used. Each set was presented once with an occluded object-distractor (Experimental trial) and once with an irrelevant object (Control trial). The stimuli used in the different sets were not repeated for the same participant, as one set was used in the Director condition and the other in the No-Director condition.

Participants were shown the grid for 2s before the instructions were presented. Three auditory instructions, each lasting 2.2s, were given per stimulus and participants had an additional 3.6s to respond. Each grid display was presented with instructions for two filler trials and one Control or Experimental trial. Thus each condition (Director and No-Director) contained forty-eight filler trials, eight Control and eight Experimental trials (sixty-four in total). Each condition lasted approximately 5.5 minutes.

7.2.3 Procedure

Please see Chapter 3.

7.2.4 Data preparation

In order to test whether the participants had understood the task, we checked the response ranges in the critical Condition (Director, Experimental). We confirmed that there was a wide range of responses across participants, instead of a bimodal distribution of participants either doing the task well or failing completely. This is in line with previous research using the same task (Dumontheil et al., 2010). Mean accuracy (MACC) and response times (MRT) were calculated for every participant in each Condition and Trial type. The mean response times were calculated from the correct responses only for each participant. Participants with no correct response in one of the two conditions, i.e. Director, No-director, were omitted from the analysis. This resulted in the removal of 2 TDC participants, 3 CD-ADHD and 3 CD+ADHD participants.

7.2.5 Data Analysis

A mixed design repeated measures Analysis of Variance (ANOVA) was used with two within-subjects factors (Condition: Director, No-Director; Trial type: Control, Experimental) and Group membership as the between-subjects factor. Filler trials were not included in the analysis (Dumontheil et al., 2010). The dependent variables (DVs) were the mean accuracy (MACC) level and the mean response time (MRT; calculated for the correct responses only). For the statistically significant differences, the analysis was rerun with IQ included as a covariate. Significant main effects and interactions were investigated with simple main effects analyses. Statistical analyses were performed using the Statistical Package for the Social Sciences, Version 21 (SPSS Inc., Chicago, IL). Effect sizes are reported as partial eta-squared (η_p^2; small≥.01, medium≥ .06, large ≥.14) (Cohen, 1988). To examine the strength and direction of the relationships between the different task outcome variables and participants' cognitive ability, Pearson correlations were conducted. For the analyses we followed the same two-stage process as in the previous chapters. During the first stage, we compared task performance between TDC subjects (n=30) and all of the participants with a CD diagnosis (n=51). In the second stage, we divided the CD participants into two groups: CD-ADHD (n=23) and CD+ADHD (n=28).

7.3 Results

7.3.1 Accuracy data

7.3.1.1 TDC versus CD

There were significant main effects of Condition [F (1, 79) = 50.15, p<.001, η_p^2 =0.388] and Trial type [F (1, 79) = 93.57, p<.01, η_p^2 =0.542] on accuracy levels across the two groups Both TDC and CD participants made more errors in the Director condition and in the Experimental trials – as expected, the condition where the Director's perspective needed to be taken into account to choose which of the two similar objects needed to be moved was the most difficult. There was a significant Condition x Trial type interaction [F (1, 79) =56.45, p<.001, η_p^2 =0 .417]. Simple main effect analysis indicated that the Director condition was less accurate than the No-Director condition across trials [F (1, 79) = 63.19, p<.001]. The three-way interaction of Condition x Trial type x Group [F (1, 79) =3.67, p=.059, η_p^2 =0 .044] approached significance. Simple effects analysis indicated that the TDC group was significantly more accurate than the CD group, but only for the Control trials in the No-Director condition (F = 5.41, p=.02; please see Figure 7.3).There were no significant main effects of Group on task performance [F (1, 79) =2.13, p=.149, η_p^2 = .026], no significant Condition x Group interaction [F (1, 79) =0.709, p=.402, η_p^2 = 0.009] and no significant Trial type x Group interaction [F (1, 79) =0.04, p=.836, η_p^2 =0 .001].

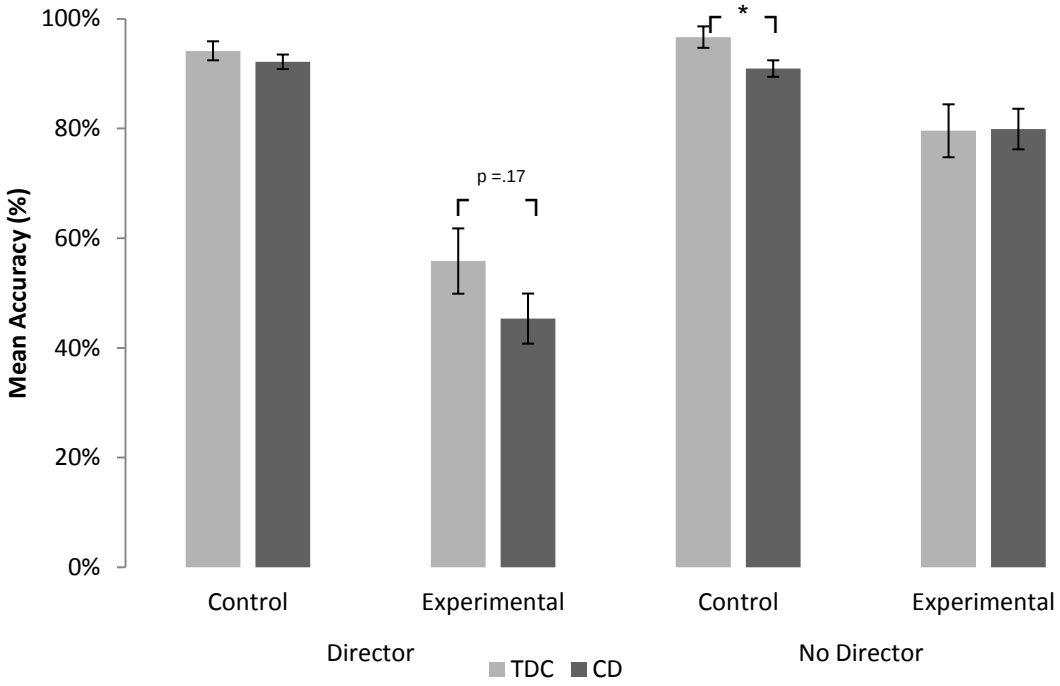

Figure 7.3. Graph illustrating the accuracy interactions between the TDC and CD groups. Note. TDC=Typically Developing Controls, CD=Conduct Disorder.*p<.05

7.3.1.2 TDC versus CD-/+ADHD

There was a main effect of Condition [F (1, 78) = 56.129, p<.001, η_p^2 =0.418] with the Director condition to be less accurate than the No-Director condition [F (1, 78) = 58.52, p<.001] and a main effect of Trial type [F (1, 78) = 98.96, p<.001, η_p^2 =0.559] with the Experimental trial types to be less accurate than the control trials [F (1, 78) = 96.51, p<.001]. There was a significant Condition x Trial type interaction [F (1, 78) =69.134, p<.001, η_p^2 =0 .470] where the Experimental trials were less accurate in the Director than in the No-Director condition. There was a marginally non-significant three-way Condition x Trial type x Group interaction [F (2, 78) =2.650, p=.077, η_p^2 =0 .064] with the TDC group to be more accurate than CD+ADHD in the No-Director Control trials (F=6.05, p =.02) (see Figure 7.4).There were no main effects of Group on task performance [F (2, 78) =1.303, p=.278, η_p^2 = .032], no significant Condition x Group interaction [F (2, 78) =0.756, p=.473, η_p^2 = 0.019] and no significant Trial type x Group interaction [F (2, 78) =0.035, p=.965, η_p^2 =0.001].

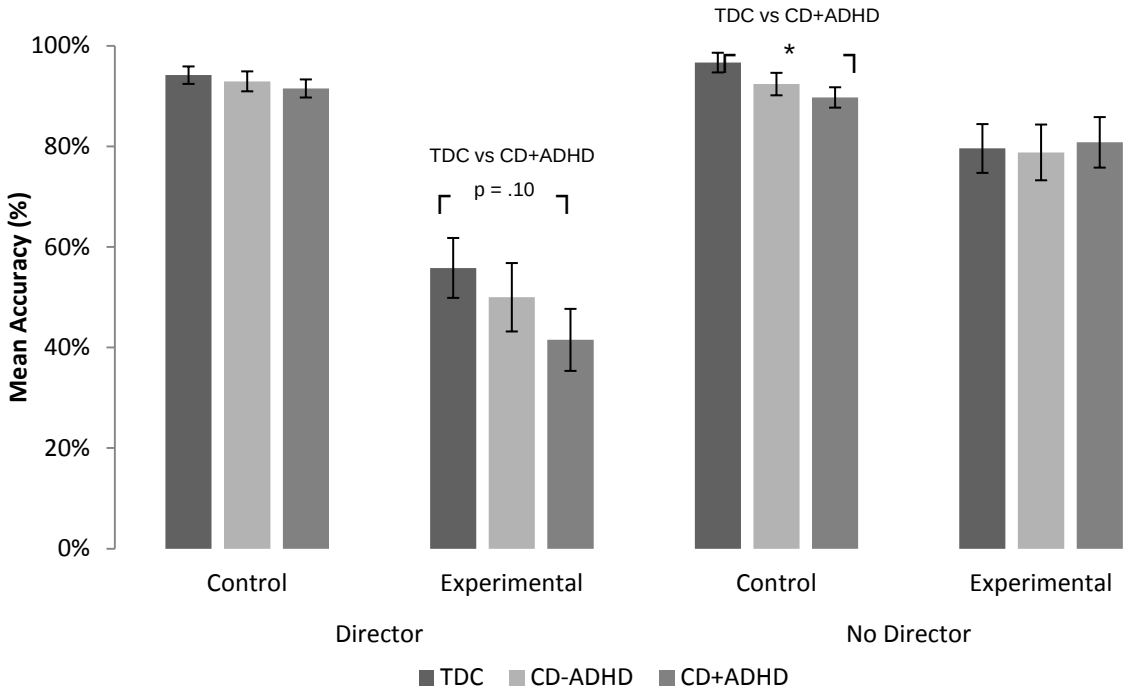

Figure 7.4. Graph illustrating the Accuracy interactions between the TDC and CD-/+ADHD groups. Note. TDC=Typically Developing Controls, CD=Conduct Disorder, ADHD=Attention Deficit/Hyperactivity Disorder. *p<.05

7.3.2 Response time data

7.3.2.1 TDC versus CD

There were no significant main effects of Group [F (1, 71) = 1.116, p=.294, η_p^2 =0.015], Condition [F (1, 71) = 1.271, p=.263, η_p^2 =0.018] or Trial type [F (1, 71) = 0.208, p=.650, η_p^2 =0.003] on response time for the correct trials. The Trial type x Group interaction (figure 7.5) was significant [F (1, 71) = 4.703, p=.033, η_p^2 =0.062]. Simple main effect analysis revealed that Control trials were quicker than Experimental Trials but only in the CD group [F (1, 71) = 7.08, p=.010]. When we repeated the main analysis including IQ as a covariate, the Trial type x Group interaction did not reach significance [F (1, 70) = 2.625, p=.110, η_p^2 =0.036] which points to an IQ effect on task performance. The three-way Condition x Trial Type x Group interaction [F (1, 71) = 3.094, p=.08, η_p^2 =0.042] was marginally non-significant. Simple main effects analysis quicker showed that the TDC group was quicker than the CD group in the No-Director condition for the Control trials only (F=6.05, p=0.02). In addition, the Director Experimental trials were marginally quicker than the No-Director

Experimental trials but only for the TDC group (F = 2.91, p=0.09). The remaining interactions Condition x Group [F (1, 71) = 0.180, p=.294, η_p^2 =0.015], Condition x Trial type [F (1, 71) = 0.796, p=.375, η_p^2 =0.011] were not significant.

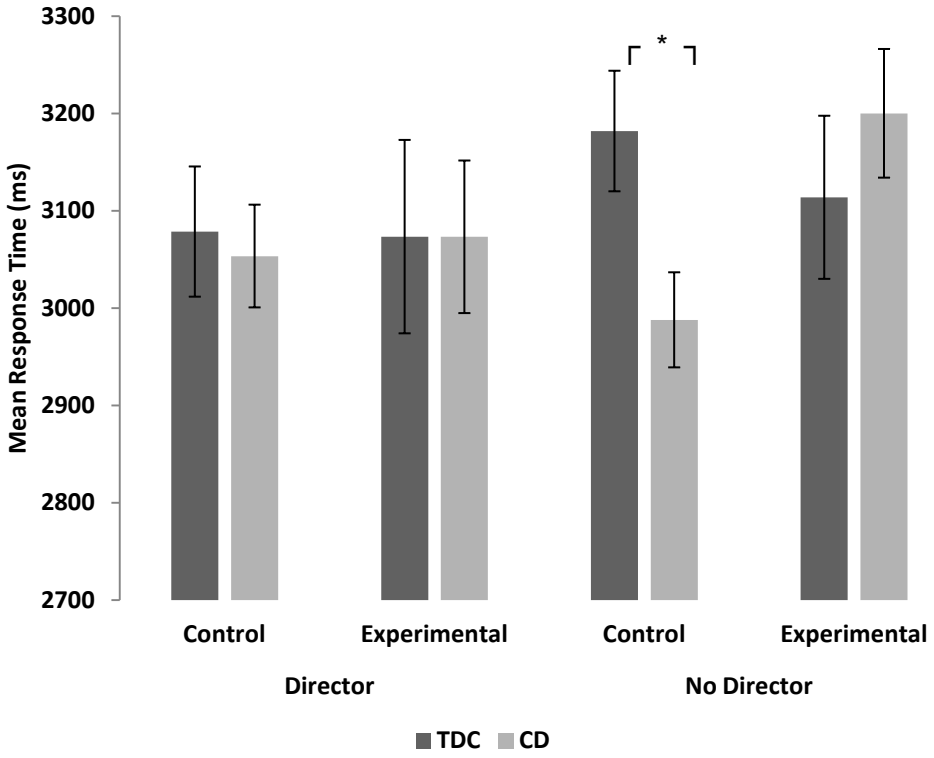

Figure 7.5. Graph illustrating the Condition x Trial type x Group interaction between the TDC and CD-/+ADHD groups. TDC=Typically Developing Controls, CD=Conduct Disorder. *p<.05

7.3.2.2 TDC versus CD-/+ADHD

There were no main effects of Group [F (2, 70) = 0.123, p=.884, η_p^2 =0.004], Condition [F (1, 70) = 1.188, p=.279, η_p^2 =0.017] or Trial type [F (1, 70) = 3.123, p=.082, η_p^2 =0.043] on task performance (figure 7.6). The Trial type x Group interaction was significant [F (2, 70) = 3.310, p=.042, η_p^2 =0.086] (figure 7.5). Simple main effect analysis indicated that the TDC group was marginally faster than CD+ADHD in the Experimental trials (F=3.021, p=.08). Also, Control trials were faster than Experimental trials in the CD+ADHD group only (F=8.43, p=.005). When we replicated the main analysis with IQ as a covariate, the Trial type x Group interaction did not reach significance [F (2, 69) = 2.289, p=.109, η_p^2 =0.062] which points to an IQ effect on task performance. The remaining interactions between Condition x Group [F (2, 70) = 1.340, p=.268, η_p^2 =0.037], Condition x Trial type [F (1, 70) = 2.552, p=.115, η_p^2 =0.035] and Condition x Trial type x Group [F (2, 70) = 2.552, p=.139, η_p^2 =0.055], did not reach statistical significance.

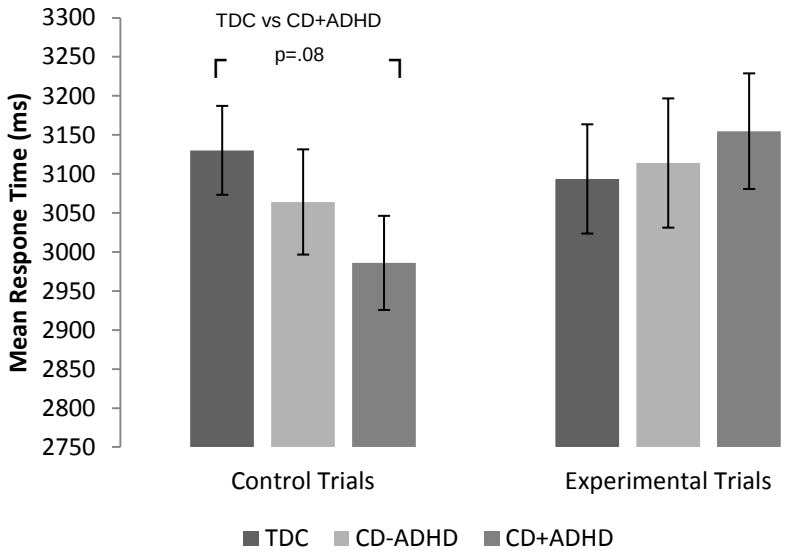

Figure 7.6. A trial type x group interaction between TDC, CD-ADHD and CD+ADHD.
TDC=Typically Developing Controls, CD=Conduct Disorder, ADHD= Attention Deficit/ Hyperactivity Disorder, CD-ADHD=Conduct Disorder without Attention Deficit/Hyperactivity Disorder, CD+ADHD=Conduct Disorder with Attention Deficit Hyperactivity Disorder, Con=control, Exp=experimental.

7.3.3. Relationship between task performance and cognitive ability

Table 7.1 shows that cognitive ability was significantly related to the accuracy scores for the Director condition in both types of trials. In contrast, cognitive ability was not related to the accuracy scores for the No-Director condition in either trial type and the response time for both conditions and both trial types.

Table 7.1: Correlations between cognitive ability and performance variables from the Director's Task

	1	2	3	4	5	6	7	8
1. IQ								
2. Director Control_Acc	.266*							
3. Director Exp_Acc	.316**	-.012						
4. No-Director Con_Acc	.175	.065	.066					
5. No-Director Exp Acc	.170	-.035	.426**	.245*				
6. Director Control RT	.080	-.044	.208	-.003	.024			
7. Director Exp RT	-.124	-.452**	.082	.089	.452**			
8. No-Director Con RT	.075	-.052	-.121	-.072	-.240*	.559**	.170	
9. No-Director Exp RT	-.004	-.089	-.208	-.139	-.238*	.403**	.147	.592**

IQ=Intelligence Quotient, Acc=Accuracy, Con=Control, Exp=Experimental, RT=Response Time. * $p < 0.05$, ** $p < 0.01$.

7.4 Discussion

We used a perspective-taking task to investigate whether adolescents with CD show ToM deficits compared to typically developing controls (TDC). This study extends previous literature in the area as we used a broader definition of ToM and we tested the hypothesis using an ecologically valid, computerised task designed specifically for adolescents. We also divided the CD group into those with and without comorbid ADHD to investigate whether ADHD alters the neuropsychological profile associated with ToM deficits in CD. There were four main findings of interest which we discuss below.

First, there were no group differences in accuracy between the TDC and CD groups in the critical condition (Director, Experimental). This runs counter to our initial hypothesis as we were expecting ToM deficits in the CD group independent of ADHD. Both groups made more errors in the Director than in the No-Director condition. This is in line with previous research using the Director's task (Humphrey & Dumontheil, 2016) where participants made more errors when the cognitive load increased i.e. deciding which of three versus two objects needed to be moved. The three objects correspond to the Director condition, where the participants had to take another person's perspective as opposed to following a pre-determined rule (No-Director condition). Our finding indicates there is a relationship between ToM and EF, as a heavier cognitive load resulted in more egocentric (i.e. self-oriented) errors (Bull, Phillips, & Conway, 2008). When we divided the CD group into CD-ADHD and CD+ADHD, we found that the TDC group was marginally more accurate than the CD+ADHD group but only in the No-Director Control trials. This means that there were no group differences in the critical condition. All three groups were equally less accurate in the perspective-taking condition and found the critical condition equally difficult. Again this runs counter to our hypothesis. It seems that neither CD nor ADHD had an additional impact on task performance when compared to controls.

Second in terms of response time, no differences were found between the TDC and CD groups in the critical condition. The two groups differed only in the Control trials, with the TDC group to be quicker than the CD group in the No-Director condition. However, as perspective-taking was not required for this condition, the group difference in response time cannot be accounted to a ToM deficit. Furthermore, analysis indicated a rather unexpected finding. Once we divided the groups to those with and without comorbid ADHD, the

CD+ADHD group was marginally quicker than the TDC group in the Experimental trials regardless of condition.

Third, as there were IQ differences between the groups we included this variable as a covariate in the main analyses. Once this was done, all the differences between the groups became non-significant. However this was proven to be weak (please see fourth point below).

Fourth, the results from the bivariate correlations between the outcome variables of the task and IQ indicated that the cognitive ability of the participants was not significantly related to response time performance. This means that IQ did not have a strong effect on task performance. Furthermore, IQ was positively correlated with the accuracy scores for the Director condition for both trial types. This means that the higher the cognitive ability the better the perspective-taking ability.

Strengths and limitations

The findings of this study should be interpreted in light of several limitations. First, our sample size may not have been sufficiently large to detect subtle differences in perspective-taking deficits between our CD-ADHD and CD+ADHD groups. However, this is the first study that directly contrasts groups with CD and/without ADHD defined using formal diagnostic criteria and it provides the basis for future work on ToM in these populations. Second, the differences in interactions for response time should be interpreted with caution as results indicated an influence of IQ on these findings. Third, only one ToM task was used in this study. Future studies should combine both cognitive and affective ToM tasks in adolescents with CD in order to better understand the different aspects of social cognition in externalising disorders. Furthermore, the task that we used required intact ToM and EF i.e. inhibition control, in order to perform well. Therefore group differences could be caused by difficulties in either domain or complex interactions between those two. However, the Director's task is a novel paradigm which combines cognitive processes, i.e. visual perspective taking, with their online use in a realistic, communicative game (Dumontheil, Küster, Apperly, & Blakemore, 2010). Our study therefore provides a basis for further research on ToM in CD.

7.5 Summary

This is the first study to use a perspective-taking task in adolescents with CD to investigate ToM deficits. In addition, this is the first study to explicitly investigate the impact of

comorbid ADHD on ToM in CD. We found that adolescents with CD performed similarly to typically-developing controls (TDC). When we divided the CD group into those with and without ADHD, we found no group differences in the critical condition between the three groups. Furthermore, there were no significant differences in the response time. Our study does not provide evidence for a deficit in perspective-taking in adolescents with CD – regardless of ADHD comorbidity.

Chapter 8 General Discussion

The primary aim of this thesis was to investigate the effect of comorbid Attention-Deficit/Hyperactivity Disorder (ADHD) on the neuropsychological profile of adolescents with Conduct Disorder (CD). The sample consisted of three groups: typically-developing controls (TDC), adolescents with CD only (CD-ADHD) and adolescents with CD and comorbid ADHD (CD+ADHD). This chapter will first summarise and synthesise the key empirical findings from the studies described earlier in the thesis. Following that, theoretical and clinical implications of the findings will be discussed. Finally, the strengths and limitations of the studies included in this thesis will be highlighted, along with considerations for future research.

8.1 Summary of key findings

Overall, individuals with CD displayed a range of different neuropsychological deficits across cold and hot aspects of executive functioning and emotion processing. The effects on Theory of Mind/perspective taking were less clear. In some domains, neuropsychological deficits linked to CD were driven (or exacerbated) by comorbid ADHD (impaired cool EF and emotion processing) whereas in other domains (hot EF, deficits appeared to be associated with CD itself.

8.1.1 Clinical and temperamental characteristics

One of the aims of this thesis was to characterise individuals with CD with and without ADHD in terms of their clinical and personality characteristics. This would allow us to form a more complete neuropsychological profile for the two groups. As mentioned in the introductory chapters, past research has yielded mixed findings with some studies showing exacerbating or no effects of comorbid ADHD on CD.

First, we examined the clinical and temperamental characteristics associated with CD and tested whether these differed as a function of the presence of comorbid ADHD. Some of the findings were interesting and unexpected. In terms of the clinical characteristics we found that adolescents in both the CD-ADHD and CD+ADHD groups reported significantly higher levels of autistic traits than the adolescents in the TDC group. This was unexpected as there is little evidence that CD in and of itself is associated with autistic traits – although the evidence relating to ADHD strongly supports an overlap between these conditions (Simonoff et al., 2008). In this case it did not look like ADHD increased the risk of elevated autistic symptoms

and CD was the main driver. In contrast, we found that it seemed that it was ADHD that was specifically associated with CU traits – whereas all of the literature would suggest that CU traits are specifically associated with CD. In fact, we found that the pure CD group did not differ from the TDC group. This striking finding suggests that the presence of ADHD is the driver of psychopathic and CU traits in CD – which goes against conventional wisdom. The final surprising clinical finding is that CD was not associated with elevated state or trait anxiety. This goes against an extensive literature linking CD and ADHD to overlapping internalising disorders (Beauchaine, Gatzke-Kopp, & Mead, 2007).

In terms of their personality characteristics, the findings were more consistent with predictions. It seemed to be ADHD specifically that was associated with impulsivity levels, which is to be expected given that impulsivity is a key aspect of the ADHD phenotype. The CD-ADHD group reported similar impulsivity levels to the TDC group. This finding shows that the presence of comorbid ADHD worsens the impulsivity levels within CD. ADHD also specifically contributed to elevated scores in the behavioural activation system – reward responsiveness subscale - as the CD+ADHD group scored significantly higher than the other two groups. Finally, ADHD did not contribute to elevated scores in the drive subscale of the behavioural activation system as both CD-ADHD and CD+ADHD had significantly higher scores than the TDC group. Overall, differences in temperament were largely related to ADHD. This is not surprising given that the impulsivity is part of the ADHD behavioural profile.

8.1.2 "Cool" Executive Functions

Previous research on the effects of comorbid ADHD on the neuropsychological profile of CD in Interference Control (IC) and Working Memory (WM) has been inconsistent (Morgan & Lilienfeld, 2000; Rubia, 2011). We employed two "cool" executive function (EF) tasks to explicitly investigate the impact of ADHD on these cognitive processes. We found that the CD group as a whole performed poorer than controls in an Eriksen-Flanker task (IC) and an N-Back task (VWM). When the groups were divided, it became apparent that ADHD was driving the differences in group performance. The CD+ADHD group performed worse than the CD-ADHD and the TDC groups. The CD-ADHD group performed in a similar way to the TDC group. Furthermore, we tested the relationship between the outcome measures of the two tasks and we found that IC ability and WM capacity were positively correlated. This was consistent with previous research (Stins, Polderman, Boomsma, & de Geus, 2005b).

Our study extended research in this area by showing that adolescents with CD+ADHD had a weaker capacity to process and hold information which in turn affected their ability to control sources of distraction and vice-versa. This was not the case for adolescents with CD-ADHD. Deficits in "cool" EFs seem to be restricted to children and adolescents with CD and comorbid ADHD rather than those with "pure" CD. This finding is consistent with those models that conceptualise ADHD as in part a disorder of cool EF, underpinned by alterations within dorsal fronto-striatal brain networks (Rubia, 2011; Zelazo & Carlson, 2012)

8.1.3 "Hot" Executive Functions

Previous research has highlighted deficits in motivational processes in CD (Fanti et al., 2016; Sully, Sonuga-Barke, Savage, & Fairchild, 2016). These are demonstrated by group differences of tasks tapping "hot" EFs, such as tasks that require participants to choose between different rewards at different delays and to cope with the frustration of imposed delay. Yet, studies that have investigated delay-related decision-making in CD are rare, and they have typically not controlled for the impact of comorbid ADHD (Dolan & Lennox, 2013; Dolan & Park, 2002). This is a problem because theories of ADHD have highlighted a motivational route to the disorder implicating an exaggerated response to delay (ref).

We employed two "hot" EF tasks; the MIDA which measures impulsive choice of small immediate over large delayed rewards and the DeFT which measures delay-related frustration. We found that the CD group as a whole showed a stronger preference for immediate rewards compared to the TDC group. When we divided the groups to examine the impact of comorbid ADHD, perhaps against expectation, results indicated that this impulsive choice in the CD group was not driven by ADHD – the CD+ADHD group choose the small immediate reward no more than the CD-ADHD group. This was a striking finding considering the direct relationship between impulsive choice and ADHD found previously. However, it should be noted that some of that evidence comes from studies that have not controlled for comorbid CD. Delay frustration, at least as measured by the DeFT, was not associated with CD either in the CD-ADHD or the CD+ADHD group. We also tested the relationship between the outcome measures of the two tasks. Not surprisingly, given our results, we found no association between the two tasks. Adolescents with CD-ADHD seem to prefer instant gratification without showing high levels of frustration when delay is imposed. Deficits in "hot" EFs seem to characterise children and adolescents with CD independent of comorbid ADHD. This offers support for models of CD that highlight problems with delay of

gratification and the altered neural circuitry in ventral fronto-striatal networks (Rubia, 2011) but not equivalent models of ADHD (ref). More generally the results of the first two empirical chapters offer support to Rubia's (2011) view that CD is a disorder of hot EF and ADHD one of cool EF.

8.1.4 Emotion Processing

Previous research has indicated a general pattern of deficits in recognising facial emotional expressions including surprise, anger, fear and disgust in CD (Short et al., 2016; Sully et al., 2015a). Some of this has highlighted the distinctive of contribution of callous-unemotional (CU) traits to this pattern. Little work on CD has examined the effect of ADHD comorbidity. This is problematic as previous research seems to suggest that ADHD is also associated with deficits in facial emotion recognition (Bora & Pantelis, 2016; Schönenberg, Schneidt, Wiedemann, & Jusyte, 2015).

Using a facial emotion recognition task – the Emotion-Hexagon - we investigated whether previous findings could be replicated once the contribution of ADHD was taken into account. In line with previous research, when we considered the CD group as a whole we found that adolescents with CD performed worse than the TDC group in the recognition of five out of six facial expressions. When our CD group was divided to CD-ADHD and CD+ADHD, we saw a graded pattern of deficits: TDC > CD-ADHD > CD+ADHD. Adolescents with CD+ADHD were significantly less accurate in recognising anger, disgust and surprise compared to the TDC group, and surprise compared to the CD-ADHD group. However, again, against expectation, there were few significant differences between the TDC and CD-ADHD groups. Furthermore, we also tested the contribution of ADHD symptoms and CU traits to the observed emotion recognition deficits within the CD sample. We found that while CU traits were a stronger predictor for anger and disgust, ADHD was an independent predictor of surprise recognition.

Taken together, these results indicate that it is comorbid ADHD and its association with CU traits that has a negative impact on emotion recognition in CD – rather than CD itself. This surprising finding undermines models of CD that place deficits of the processing of emotions at the heart of the disorder (Mullin & Hinshaw, 2007), and also encourages us to explore further the reasons why individuals with ADHD have problems with emotion processing. One possible reason is that the deficits reflect more general attentional abilities rather than specific socio-cognitive misrepresentations (Villemonteix et al., 2017).

8.1.5 Social Perspective Taking-Theory of Mind

The majority of studies of social cognition in CD have focused on facial emotion recognition and interpersonal empathy. In our final empirical chapter, we included a task which measures another facet of social cognition – Theory of Mind (ToM). Previous research on ToM in CD has been rare. In addition, research findings on ToM deficits in ADHD have been mixed (Nijmeijer et al., 2008). We used a perspective-taking task – the Director's Task (DT) – and examined whether adolescents with CD exhibit ToM deficits compared to TDC. We were also interested in the contribution of comorbid ADHD. Our focus was on the impairments observed in the critical condition of the task, which indicated the presence of a ToM deficit. Counter to our hypothesis, neither group seemed to show impairments in ToM although there was a tendency for the comorbid group to perform less well on the condition that required the participants to take on board the perspective of the Director. This suggests that if deficits are present they are subtle, and associated with ADHD rather than CD. Future research should try and explore these effects in larger samples.

8.2 Implications

Our results provide new insights on the impact of the comorbidity of ADHD in the neuropsychology of CD. The first important theoretical contribution is that our findings give support to the notion that CD is a "hot" EF disorder whereas ADHD is associated with "cool" EF deficits. This distinction is not new. Zelazo and Müller (2002) suggested that "cool" EFs involve abstract, decontextualized problems whereas "hot" EFs involve problems that require the regulation of affect and motivation. Our findings support those of Dolan and Lennox (2013) and Rubia (2011) and suggest that this distinction between aspects of EF is valid within the context of CD research. However, it seems to contradict the work of a number of researchers such as Sonuga-Barke (2001) and Sagvolden et al. (2005) who argue that delay and reward related problems are at the core of the ADHD phenomenon.

The second theoretical implication deriving from our findings is that our results did not support the model of behavioural inhibition/activation system (BIS/BAS) activity (Quay, 1993). In this system, CD is viewed as a disorder characterised by an overactive BAS (excessive reward seeking behaviour) and an underactive BIS (reduced sensitivity to punishment/ termination of reward). Even though the BAS scores were elevated for two subscales, the BIS scores were equivalent across the three groups suggesting no decrease in avoidance behaviour or inhibition of response.

The third theoretical implication is the contribution of ADHD to emotion recognition deficits in CD. Theoretical models should start taking into consideration this possible contribution. Furthermore, educational training programmes targeting the improvement of emotion recognition performance should take into account the difficulties that individuals with ADHD encounter in terms of learning, i.e. inattention to social cues.

The fourth theoretical implication is that impulsive choice and delay-related frustration seem to be two independent constructs. Our data from the delay-related task do not support the theory that children and adolescents with Disruptive Behaviour Disorders (DBD) are characterised by poor affect regulation (Nigg, Goldsmith, & Sachek, 2004). Although it should be noted that this task is tapping one aspect of affect regulation i.e. delay-related frustration.

In addition to the theoretical contributions, our findings have a number of clinical implications that could be of aid to the design and implementation of future school-based interventions and parenting programmes. First, we showed that our groups have different behavioural profiles. Adolescents with CD had lower IQ than the adolescents in the TDC group. This potentially sheds light to aspects of cognitive ability that can be strengthened and improved through personalised programmes, within an educational system that accommodates the needs of every student. Furthermore, within our sample we identified that the adolescents with CD+ADHD had significantly higher CU traits and impulsivity scores. The latter, combined with the second clinical implication where we showed that ADHD significantly exacerbates the non-significant levels of recognition deficits in emotions like anger, disgust and surprise, suggests that adolescents with CD are possibly at a disadvantage when making decisions based on reading facial expressions and perhaps body language – both important components of successful social interaction. Third, adolescents with CD+ADHD demonstrated a weaker visuo-spatial working memory capacity. Parenting programmes should include modules that teach parents how to recognise that deficit and at the same time training them into how to maximise their child's memory capacity. Fourth, we showed that delay-related frustration levels in adolescents with CD do not differ to those without. This is an important finding as it demonstrates that adolescents with behavioural difficulties may have a good frustration threshold, perhaps making it easier for parents to connect with their children via communication exercises that promote understanding. Fifth, we demonstrated that adolescents with CD, regardless of ADHD comorbidity, had no perspective-taking deficit. Again, this is something that parenting programmes should take

into consideration and incorporate role plays promoting dialogue techniques into their curriculum.

8.3 Strengths

Our study had a number of advantages over previous research in this area. Firstly, to our knowledge, this is the first study to explicitly examine the impact of comorbid ADHD on a sample of adolescents with "pure" CD. This is extremely important given the very high rates of comorbidity between the two disorders (Angold et al., 1999; Caron & Rutter, 1991). As discussed previously, the majority of studies looking at the neuropsychological deficits of CD have rarely controlled for ADHD comorbidity and its impact on the neuropsychology of CD (Blair, 2015; Morgan & Lilienfeld, 2000; Oosterlaan et al., 1998; Rubia, 2011).

Second, the majority of studies investigating the neuropsychology of CD have included a mixed Disruptive Behaviour Disorders (DBD) group. This has been found to be quite problematic given the evidence that ODD has distinct neuropsychological features (Copeland, Shanahan, Erkanli, Costello, & Angold, 2013). Our sample was well-characterised from a clinical perspective. We used interview-based diagnostic assessments which ensured that participants in our clinical groups had clinically significant behavioural problems and all had a diagnosis of CD. Furthermore, we also ensured that our clinical groups did not have clinically significant levels of autistic traits, given that Autism Spectrum Disorders (ASD) and CD have often been examined together in terms of their social cognition deficits (O'Nions et al., 2014).

Third, to our knowledge this is the first study that directly compares adolescents with "pure" CD on a broad range of tasks in order to examine the impact of ADHD on the neuropsychology of CD. This thesis included "hot" and "cool" EF tasks, an emotional recognition task and a ToM task. This variety of cognitive domains helped us to have a clearer picture of the pattern of deficits in CD and ADHD when compared to patterns of typical development.

Fourth, we directly tested the hypothesis that CD is a "hot" EF disorder and we extended this line of research by showing that impulsive choice is a unique characteristic of CD, independent of ADHD. This on its own is quite a striking finding and needs to be explored further.

Fifth, against expectation ADHD and not CD per se was associated with CU traits. Previous research has shown elevated levels of CU traits in ADHD but their impact as mediators of neuropsychological deficits has not been investigated before. Here we found that these ADHD-related CU traits contributed to the deficits seen in emotion recognition.

Sixth, CD was independently associated with ASD. This link has not been highlighted before. However, it could be vital in understanding the social deficits that CD individuals display especially with regard to the interpretation of other people's intentions. Interestingly, CD was not associated with ToM impairment – a core deficit in ASD.

8.4 Limitations

Despite the substantial strengths of the present study, the results should be interpreted in the light of a number of limitations. First, the present studies did not include a group of adolescents with "pure" ADHD. This would have helped interpret the effects of ADHD per se, rather than only looking at the effects of ADHD within CD. It is possible that pure ADHD and comorbid CD and ADHD are completely different constructs and so one cannot draw any inferences about the nature of the former from observations about the latter. However, it should be noted that we are still not sure whether a "pure" form of ADHD exists, especially in adolescence. In addition, even though the overwhelming majority of studies use a control group with typically developing adolescents, we should pay attention to the methodological constraints about the generalisability of the findings; using a clinical sample versus a sample of super-controls within a restricted laboratory set of tasks may cause an implicit bias against the adolescents with CD.

Second, the groups included in this thesis were not matched on cognitive ability. The CD group had lower mean estimated IQ than the TDC group. However, this is a common finding. Lower IQ within CD populations as opposed to typically developing children and adolescents has been a consistent finding in studies with community and clinical CD samples (Nock et al., 2006; R. M. Short et al., 2016; Sully et al., 2016). In addition, researchers should be apprehensive when attempting to match clinical and control groups in terms of cognitive ability; "cherry-picking" CD individuals with higher IQs could have detrimental effects to the construct validity of the disorder. We controlled for IQ within the statistical analyses – and in some cases, this did reduce the magnitude of the group differences.

Third, even though we examined multiple cognitive domains, using a variety of behavioural tasks, we did not include a ToM task with an empathy element mainly due to testing time restrictions; this inclusion would have enabled us to explicitly compare and contrast the differences between perspective-taking and empathy deficits in adolescents with CD.

Fourth, our sample size may not have been sufficiently large to detect small differences between the comorbid CD+ADHD group and CD-ADHD group. Therefore, it is possible that we would have been able to demonstrate additional differences between the two groups, had we been able to recruit a larger sample.

Fifth, due to the nature of the study and the difficulties associated with recruiting these particular clinical groups, it was necessary to test multiple hypotheses on data from single experiments (i.e. the effects of the various task conditions, as well as the differences among our three groups). This simultaneous testing of multiple hypotheses (e.g. by using pairwise contrasts) inflates the risk of making false-positive statistical inferences: in the worst case, if n independent hypotheses are tested, the risk of a type I error increases n times (Bender & Lange, 2001; Noble, 2009). In order to control for the type I error rate, we employed Bonferroni corrections to our post-hoc tests. This meant that the statistical significance of our findings was reduced in some cases (e.g. difference in anger recognition between the CD-ADHD and the CD+ADHD groups in chapter 6). However, we note that a. the Bonferroni correction is conservative (Noble, 2009), and b. this study is the largest of its kind and it provides a useful platform for future work in the neuropsychological phenotype of CD and comorbid ADHD.

8.5 Future research directions

The main goal of this thesis was to test in what way the presence of comorbid ADHD influences the neuropsychological profile associated with CD. Our cross-sectional data allowed us to investigate neuropsychological patterns of CD in terms of task performance. We were able to show patterns, correlations, similarities and/or differences between our three groups. The question that remains unanswered (and was not in the scope of this thesis) is: what are the mechanisms that underlie comorbidity in CD and ADHD and what is the direction of the causal links between the two disorders? Future longitudinal research is needed, with the endeavour of identifying "pure" cases of ADHD. This would enable researchers to make direct comparisons between CD, CD+ADHD and ADHD and establish common and distinct neuropsychological patterns.

There is also the need for a universal definition of comorbidity and how that can be tested both in cross-sectional and longitudinal studies. As mentioned in chapters 1 and 2, the term comorbidity is problematic because of neuropsychological heterogeneity and lack of causal specificity (Coghill, 2014). In addition, taking into account that CD and ADHD symptoms are not stable but change over time, it would be interesting to see which symptoms persist or weaken in the "pure" CD and ADHD cases and which symptoms persist or weaken in the comorbid CD+ADHD cases.

Further to the research on the deficits and externalising symptoms of CD, an approach focused more on social factors like childhood adversity, family and peer social pressures and societal expectations and how these contribute to the emergence of comorbidity and psychopathology, would help clinicians to design more effective clinical interventions; the ultimate goal should be personalised programs with preventative elements (Cerdá, Sagdeo, Johnson, & Galea, 2010). Community-based research and implementation of educating programs about the nature of CD and ADHD deficits can also be a way forward in making societies more tolerant and understanding of the behaviours that children and adolescents with these disorders present. Another very promising strand of future research is targeting interparental relationships and parenting paradigms and improve those through incorporation of knowledge from quantitative genetic research (Harold, Leve, & Sellers, 2017). In Figure 8.1 we present a sketch of future CD research questions and themes for further investigation.

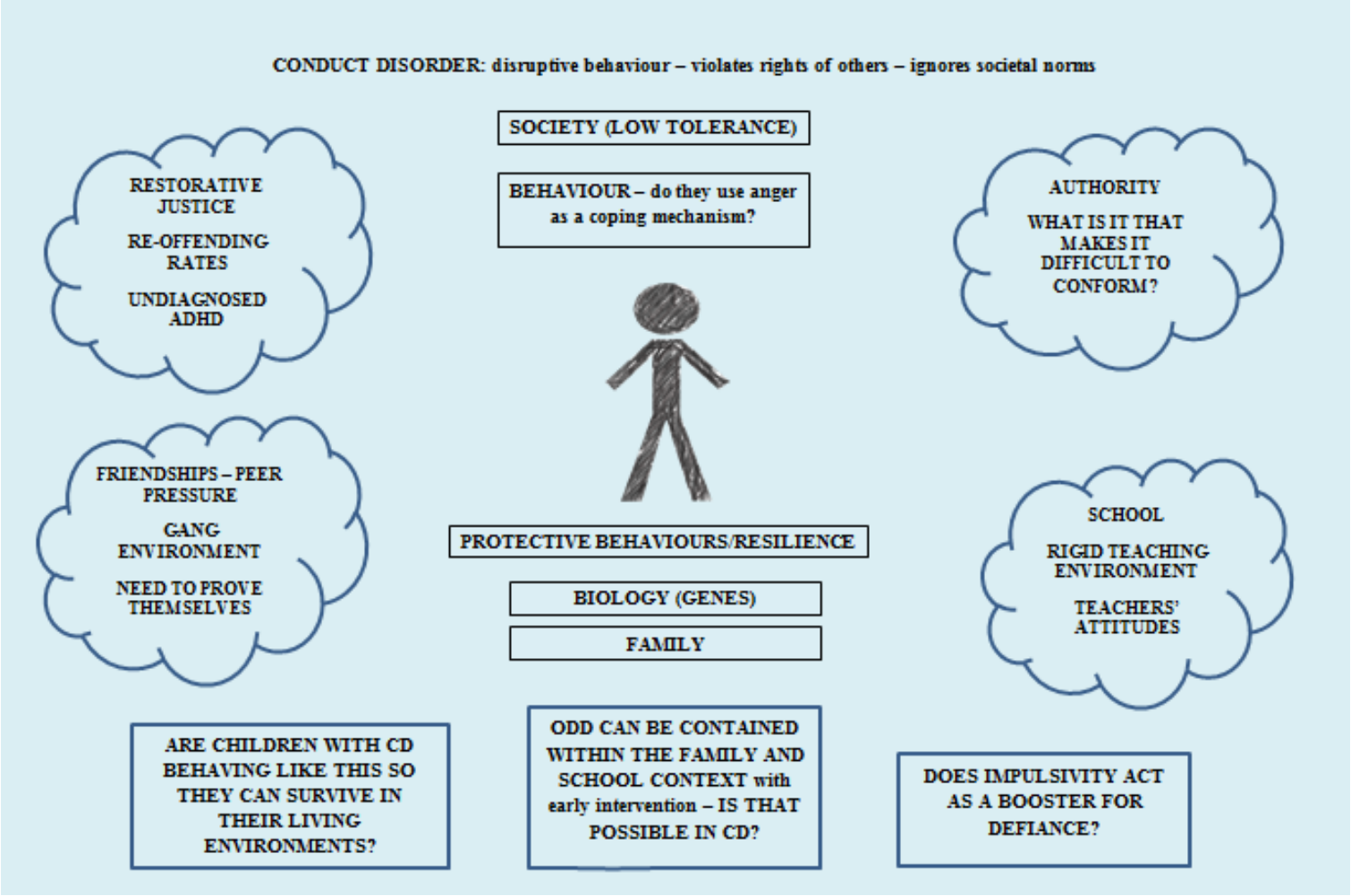

Figure 8.1. Research themes in conduct disorder for further future investigation. CD=conduct disorder, ADHD=attention deficit/hyperactivity disorder, ODD= oppositional defiant disorder.

General Conclusion

Previous research has acknowledged that CD is often accompanied by ADHD. However, the majority of studies of neuropsychological deficits in CD have had two main problems: their sample consisted primarily of a mixed disruptive behaviour disorders (DBD) group and the presence of ADHD was not accounted for. Therefore the impact of ADHD has rarely been investigated in a systematic way. We addressed this gap in the literature by using a well-characterised clinical sample of adolescents with CD-ADHD and CD+ADHD to examine the effects of comorbid ADHD on a range of different neuropsychological domains. We found different effects of ADHD in different neuropsychological domains – some of them quite surprising. The presence of ADHD accounted for deficits in "cool" executive functions seen in the CD group as a whole. This was not the case for "hot" executive function tasks where adolescents with CD were found to have a deficit independent of ADHD. The results on a facial emotion recognition task showed that even adolescents with CD had a wide range of emotion recognition deficits these were largely driven by ADHD in conjunction with CU traits. There was little evidence that CD or ADHD were related to deficits in Theory of Mind (ToM) although this may have been due to a small sample size. Considered together, these results indicate the need for careful sample characterisation and inclusion in clinical groups, as the impact of comorbid ADHD does appear to have a negative effect on the neuropsychology of CD. Clinicians should also take these results into consideration when designing future clinical interventions. Individuals with CD-ADHD may require a different therapeutic approach from individuals with CD+ADHD.

Closing remark

How different would our society's approach to CD be if we associated its "deficits" with resilience, adaptation or even survival? Starting modular and becoming modular are two different things (Karmiloff-Smith, 1998). The focus should now be more on the genetic risk factors and environmental/familial effects rather on just how to design more effective restorative justice programmes. How did children with CD learn to associate specific behaviours with specific outcomes and vice-versa? Time and research will tell whether, when taken from an evolutionary perspective, CD behaviour can be described as a protective factor, associated with resilience.

"Toward a developmental perspective on conduct disorder: this title amounts to an oxymoron. Not only is there nothing developmental about the diagnosis of conduct disorder, but also the resistance of its diagnostic criteria to contextual information about a child's developmental history, capacities and circumstances might even be viewed as anti-developmental." (Richters & Cicchetti, 1993, p.3).

APPENDIX A. Materials used in the study

A1. Participant information sheet (Youth Offending Teams)

Participant Information Sheet (Version 1, 30/05/2012)

Study Title: The impact of attention on behavioural difficulties during adolescence.
Researcher: Nadia Peppa
ERGO Study ID number: 1990
RGO reference number: 8668

Please read this information carefully before deciding to take part in this research. If you are happy to participate you will be asked to sign a consent form.

Hello. My name is Nadia Peppa and I am a PhD student at the University of Southampton. We are currently working on a new and interesting project looking at how attention problems may have an impact on behavioural difficulties. We would like to invite you to take part in this project. Please could you read this information sheet carefully as it gives you more information about the study? You can ask us any questions you like and tell us if there is anything you don't understand.

What is the research about?

The aim of this study is to investigate why people with attention problems often show behavioural difficulties, by studying teenagers with attention problems alone, behavioural difficulties alone, or both attention and behavioural difficulties together. We are also interested in recruiting a control group of young people with neither attention problems nor behavioural difficulties. In order to study these issues, we will be using a method called Event Related Potential (ERP) testing, which measures the brain's activity, and a set of computerised neuropsychological tasks.

The study involves a home visit, in which we will introduce the study and its aims, and give you an opportunity to ask questions. We will ask you and your parent/carer questions about your typical moods, thoughts, and behaviours. We will also ask you and your parent/carer to nominate one of your teachers/tutors at school to complete a very short questionnaire about

whether you have difficulties concentrating or following through instructions. We are not interested in your grades or exam performance and the information your teacher/tutor gives us will remain confidential.

If you decide to take part, and on a day that is convenient to you, you will be invited to the University of Southampton to complete three tasks that look at attention and self-control, responses to reward and delay and emotion recognition. For these tasks you will be asked to wear a soft cap with sensors to measure the electrical signals coming from your brain. On a different day, we will invite you to come back to the University, this time to complete a set of computer-based tasks which will look at attention and self-control, memory, responses to reward and delay, face recognition, emotion recognition and perspective taking. You will also be asked to fill in questionnaires regarding your general well-being and personality. By doing this study, we hope to understand better the relationship between behavioural difficulties and attention problems.

Why have I been chosen?

We are recruiting teenagers aged between 11-18 years, who may have had behavioural difficulties or may have behavioural difficulties at the moment. We are approaching youth offending teams for this purpose. Our sample is what is called an 'opportunity' sample – anyone who chooses to take part can do so, providing they are not affected by the study's exclusion criteria. It is up to you to decide whether or not to take part. If you do, you will be given this information sheet to keep and be asked to sign a consent form. If you are aged below 16 your parent/carer will also have to give consent. You are still free to withdraw at any time and without giving a reason. You can also choose to opt out of any part of the study.

What is Event Related Potential testing? Will it hurt?

The Event Related Potential method allows us to measure the electrical activity of the brain. Our brains are constantly producing small electrical signals, so we will just be measuring signals that your brain produces all the time. The sensors that are attached to the soft cap detect tiny electrical signals, so when this cap is placed on the head they record the electrical activity of the brain. This is completely painless and harmless. You can see someone wearing one of these caps below. All the sensors will be filled with a gel to improve the quality of the signal. There is no risk involved; the sensors do not pass electrical activity back to the brain. Should you decide that you don't want to continue with the testing, the cap can be removed in less than a minute.

What will happen to me if I take part?

If you agree to take part in this study, we would like to come to your house to explain to you and your parent/carer the different aspects of the study. During this first visit we would like to ask you some questions regarding your behaviours and lifestyle to see if you fit the criteria for the study. We would like to ask your parent/carer the same questions in a different room. This part should take about 1 hour and you and your parent/carer will be paid for your time (£10 each).

If you fit the inclusion criteria you will be invited to the University of Southampton to complete a set of tasks. The study is divided in two parts each of which takes place on different days. We would like you to take part in both aspects of the study, and we really hope you do, however if you feel uncomfortable you can opt out of either the ERP testing or the computer tasks.

We will invite you to the School of Psychology, University of Southampton for the ERP testing. For this you will be asked to wear a soft cap with sensors attached on it. It will feel a bit like you are wearing a swimming cap. When the cap is on your head you will be asked to relax and rest for 5 minutes while we record your brain activity while you are doing nothing other than resting. Next you will be asked to complete three tasks that involve paying attention and self-control, making choices based on delay and reward and recognising faces and voices. You will also have the opportunity to ask questions at the beginning and while we are fitting you the cap, and you will be given lots of breaks between the tasks. This part of the study will last around 3 hours and you will be paid £25 for your time.

On a different day, you will be invited to come to the University to complete the second part of the study. This part involves taking part in computer-based tasks which will be fully

explained to you before the testing begins. The first set of tasks looks at how we can suppress a response and how good our memory is when asked to position objects in a specific sequence. The second set requires you to play a game of where you have to shoot down enemy battle ships to protect your space ship and win points. The third set requires you to recognise faces and emotions, and the last set requires you to understand situations from another person's perspective. You will also be asked to fill in questionnaires regarding your general well-being and personality traits. There will be small breaks between the different tasks. You are free to stop taking part in the study at any time. This part of the study will last approximately 3 hours and you will be given £20 for taking part.

Are there any benefits in my taking part?

You will be reimbursed for your time and travel (£10 for the interview, £25 for the ERP testing, and £20 for completing the computer tasks). In addition, by taking part in a study like this you will help us to understand more about the relationship between attention problems and behavioural difficulties during adolescence. There is no intended clinical benefit to you from taking part in this research.

Are there any risks involved?

There are no risks involved in this study. The only discomfort you may experience is that you may need to wash your hair after the ERP testing because of the gel. However, the gel washes off easily and you can wash your hair at the University before you leave. We will provide you with a towel, shampoo and hair dryer.

Will my participation be confidential?

Yes. All the information we collect about you, including your name and address, will be kept private. Only people in the research team will know your name and address. Your identity will be protected by changing your name to a subject code during analysis. Records of your personal details will only be kept if you agree. We assure you that any information given will be strictly confidential, not shown to your parent or carer, and you can leave out questions you are not comfortable with. All questionnaires will be number coded and if the study is written up for publication, the paper(s) will not include names or any other identifying characteristics.

The only time when we might have to talk to people outside of the research team is: When you tell us information that makes us concerned that your safety or the safety of another person in your family is at risk. In this instance, we may be duty bound to refer you on to

someone who may be able to help, e.g. your GP. We will not pass on this information to anyone else without letting you know first.

What happens if I change my mind?

You are able to withdraw from the experiments at any time, without explaining why. We may use any data collected up to the point of withdrawal.

What happens if something goes wrong?

If you have a concern or complaint regarding any aspect of this study, you can contact the Head of Research Governance, Dr Martina Prude (02380 595058, mad4@soton.ac.uk) who will be happy to help.

Where can I get more information?

Thank you for taking the time to read this letter. If you would like more information or if you have any questions please contact us by e-mail or telephone (Nadia Peppa: nadia.peppa@soton.ac.uk; 02380 594594, Graeme Fairchild: g.f.fairchild@soton.ac.uk).

If you would like to talk to an independent person regarding involvement in this research we recommend you speak with your: family, friends or a teacher at school.

Yours sincerely,

Nadia Peppa, Graeme Fairchild and Edmund Sonuga-Barke

If you have questions about your rights as a participant in this research, or if you feel that you have been placed at risk, you may contact the Head of Research Governance, Dr Martina Prude (02380 595058, mad4@soton.ac.uk) who will be happy to help.

UNIVERSITY OF Southampton

A.2 Consent form

CONSENT FORM (Version 1, 30/05/2012)

Study title: The impact of attention on behavioural difficulties during adolescence.

Researcher name: Nadia Peppa
ERGO Study ID number: 1990
RGO reference number:

The aim of this study is to investigate why people with attention problems often show behavioural difficulties, by studying teenagers with attention problems alone, behavioural difficulties alone, or both attention and behavioural difficulties together. We will also be recruiting a control group of young people with neither attention problems nor behavioural difficulties. The study involves a home visit, in which we will introduce the study and its aims, and give you an opportunity to ask questions. We will also ask you and your parent/carer questions about your typical moods, thoughts, and behaviours. If you decide to take part, and on a day that is convenient to you, you will be invited to the University of Southampton to complete three tasks that look at attention and self-control, responses to reward and delay and emotion recognition. For these tasks you will be asked to wear a soft cap with sensors to measure the electrical signals coming from your brain. The testing session will last approximately 3 hours. On a different day we will invite you to come again at the University, this time to complete a set of computer-based tasks which will look at attention and self-control, memory, responses to reward and delay, face recognition, emotion recognition and perspective taking. You will also be asked to fill in questionnaires regarding your general well-being and personality. This last part of the study will last approximately 3 hours. There will be small breaks between the different tasks.

To give your consent to participate in the study, please initial the boxes below to show that you agree with each of the statements, and sign and date this form.

I have read and understood the information sheet (30/05/2012 /version 1) and have had the opportunity to ask questions about the study.	☐
I agree to take part in this research project and agree for my data to be used for the purpose of this study	☐
I understand my participation is voluntary and I may withdraw at any time without my legal rights being affected	☐
I am happy to be contacted regarding other research projects in the Developmental Brain-Behaviour Laboratory. I therefore consent to the research team retaining my personal details on a database, kept separately from the research data detailed above. The 'validity' of my	☐

Data Protection

I understand that information collected about me during my participation in this study will be stored on a password protected computer and that this information will only be used for the purpose of this study. All files containing any personal data will be encrypted or stored in a locked filing cabinet.

- If participant is age 16 or over, please could they sign below to give consent:

Name of participant (print name)……

Signature of participant……..

Date……

- If participant is under the age of 16, consent is required from their parent/guardian, please could they sign below:

Name of Parent (print name)……

Signature of parent……..

Date……

- The teenage participant should sign below to indicate their willingness to take part:

Name of Participant (print name)……

Signature of participant……..

Date……

A.3 K-SADS screen – Preliminary interview - YOUTH

We would like to ask you a few questions about how you've been feeling ***over the last 12 months****. It isn't a test of any kind. There are no right or wrong answers – all I'd like you to do is tell me as honestly as you can how you've been feeling. The information you give us today is* ***confidential*** *and will go no further. However, if we think that you are having problems at the moment which could benefit from help, then we will discuss the possible options with you, e.g. referring you to a doctor. If you provide information that makes me concerned about your safety or the safety of another person in your family, I may be duty bound to refer you or them on to someone who can help, e.g., your GP. We will not pass on this information to anyone outside the research team without telling you first.*

The first thing I'd like to ask is whether you have ever seen a healthcare professional (e.g. a GP, an Educational Psychologist, a Psychiatrist or a counsellor) for any other reason apart from routine illness? This could include emotional difficulties or mood changes.

Have you ever been prescribed medication for anything apart from routine illness (e.g. colds, coughs or flu)?

1) MAJOR DEPRESSIVE EPISODE

At least 1 from the following 3 symptoms present for more than half the time for a period of at least 2 weeks:

DEPRESSION	Current	Yes/No	Onset:
Everyone has good days and bad days, but in the past 6 months has there been a time when you've felt down, miserable or depressed for days on end? How long did this feeling last? Do you feel like this at the moment? Have you ever gone through a time in your life when you felt like this?	Past 12 months	Yes/No	Onset/dur:
	Lifetime	Yes/No	Dates/dur:

IRRITABILITY	Current	Yes/No	Onset:
Has there been a time when you've felt irritable or angry for most, or all of the time, for days on end? How long did this last? What about recently? Is there a reason why you felt angry?	Past 12 months	Yes/No	Onset/dur:
	Lifetime	Yes/No	Dates/dur:

LOSS OF INTEREST/PLEASURE	Current	Yes/No	Onset:
What about a time when you completely lost interest in doing things or stopped going out? Or felt you couldn't have fun or enjoy the things you used to? How long? What about now?	Past 12 months	Yes/No	Onset/dur:
	Lifetime	Yes/No	Dates/dur:

2) <u>GENERALISED ANXIETY DISORDER</u>	Current	Yes/No	Onset:
Would you describe yourself as a 'worrier'? Have you been worrying a lot about things that have happened to you or might happen? What sort of things? Does the worrying affect your everyday life? Is it difficult to control? How long have you felt like this?	Past 12 months	Yes/No	Onset/dur:
	Lifetime	Yes/No	Dates/dur:

3) <u>PANIC DISORDER</u>	Current	Yes/No	Onset:
Have you ever had a panic attack (that's when you suddenly feel very afraid, or even feel you might die, when there's no reason to feel like that)? Did you ever feel like you couldn't breathe, or that you were having a heart attack? When did this happen? How many times has it happened to you in the last 6 months? ***If endorsed for lifetime, frequency of lifetime panic attacks?***	Past 12 months	Yes/No	Onset/dur:
	Lifetime	Yes/No	Dates/dur:

4) <u>SOCIAL PHOBIA</u>	Current	Yes/No	Onset:
Some people feel very shy when they are in social situations. Have you ever found it very hard to talk to people you don't know? Even if it was someone your own age? Would you ever avoid social situations (e.g. parties) because you felt so uncomfortable around strangers or worried what people would think about you? Would it really scare you to have to speak in front of people or answer questions in class?	Past 12 months	Yes/No	Onset/dur:
	Lifetime	Yes/No	Dates/dur:

5) <u>SPECIFIC PHOBIAS</u>	Current	Yes/No	Onset:
Has there ever been a time when you were really scared of spiders, snakes, dogs, insects, heights, the dark or something like that? What would happen if you saw ___? Would your heart beat faster? Or would you find yourself unable to move? Would you feel so afraid by _____ that you wouldn't do things or go out? Is it like that now or only when you were younger?	Past 12 months	Yes/No	Onset/dur:
	Lifetime	Yes/No	Dates/dur:
6) <u>OBSESSIONS/COMPULSIONS</u>	Current	Yes/No	Onset:
Have you ever been bothered by thoughts or images that make no sense to you, but keep coming into your head for no reason? What about habits that make no sense to you, like counting things several times? Or do you repeat things over and over, like washing your hands or checking whether your door is locked? Are you bothered by this at the moment?	Past 12 months	Yes/No	Onset/dur:
	Lifetime	Yes/No	Dates/dur:
7) <u>POST-TRAUMATIC STRESS DISORDER</u>	Current	Yes/No	Onset:
Has anything traumatic or tragic happened to you in the last year? I mean something serious like being attacked or in car accident, or in some other sort of serious danger or nearly dying? Have you witnessed something like that happening to someone else? When? If yes, get details of the event in question. *How scared were you at the time? How did you act? Did you find you couldn't think straight? Were you shaky or jittery or restless? How did you feel afterwards? Did you find it difficult to think about anything else? How long did this go on for (i.e. weeks, months or years)? How about now?*	Past 12 months	Yes/No	Onset/dur:
	Lifetime	Yes/No	Dates/dur:

8) <u>ALCOHOL USE/ABUSE</u>	Current	Yes/No	Onset:
How much alcohol do you drink in a typical week? What do you drink – beer, wine or spirits? How often? Do you get drunk? Has using alcohol ever caused you to have any health problems (physical/psychological)? Have you ever had to go to a doctor or hospital because of drinking too much? Have you ever missed school/work because you've been too hungover or drunk?	Past 12 months	Yes/No	Onset/dur:
	Lifetime	Yes/No	Dates/dur:

9) <u>SUBSTANCE USE/ABUSE</u>	Current	Yes/No	Onset:
Have you ever tried recreational drugs (e.g., cannabis)? Which drugs have you tried? If you regularly take drugs, how often do you do this? Have you ever had any health problems (physical/psychological) as a result of using drugs? Have you ever had to go to a doctor or hospital because of taking drugs? Have you ever missed school/work because you've been high or on a comedown?	Past 12 months	Yes/No	Onset/dur:
	Lifetime	Yes/No	Dates/dur:
N.B. Establish which drugs and pattern of drug-taking (i.e., at least five times for any one group of drugs).			

10) <u>OPPOSITIONAL DEFIANT DISORDER</u>	Current	Yes/No	Onset:
In the last 12 months have you been in trouble a lot at school, or at home? Have you ever been suspended or excluded from school? Do you lose your temper easily? Do you get into a lot of arguments, maybe with teachers or parents? What about at the moment?	Past 12 months	Yes/No	Onset/dur:
	Lifetime	Yes/No	Dates/dur:

A.4 State-trait anxiety inventory (Trait)

STAI – T

A number of statements which people have used to describe themselves are given below. Read each statement and then mark the appropriate number to the right of the statement to indicate **HOW YOU GENERALLY FEEL.** There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe how you generally feel.

		Not at all	**A little**	**Somewhat**	**Very much so**
1	I feel pleasant	1	2	3	4
2	I feel nervous and restless	1	2	3	4
3	I feel satisfied with myself	1	2	3	4
4	I wish I could be as happy as others seem to be	1	2	3	4
5	I feel like a failure	1	2	3	4
6	I feel rested	1	2	3	4
7	I am “calm, cool, and collected”	1	2	3	4
8	I feel that difficulties are piling up so that I cannot overcome them	1	2	3	4
9	I worry too much over something that really doesn’t matter	1	2	3	4
10	I am happy	1	2	3	4
11	I have disturbing thoughts	1	2	3	4
12	I lack self-confidence	1	2	3	4
13	I feel secure	1	2	3	4
14	I make decisions easily	1	2	3	4
15	I feel inadequate	1	2	3	4
16	I am content	1	2	3	4
17	Some unimportant thought runs through my mind and bothers me	1	2	3	4
18	I take disappointments so badly that I can’t put them out of my mind	1	2	3	4
19	I am a steady person	1	2	3	4
20	I get in a state of tension or turmoil as I think over my recent concerns and interest	1	2	3	4

A.5 State-trait anxiety inventory (State)

STAI - S

A number of statements which people have used to describe themselves are given below. Read each statement and then mark the appropriate number to the right of the statement to indicate how you **FEEL RIGHT NOW, that is AT THIS MOMENT.** There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best.

		Not at all	**A little**	**Somewhat**	**Very much so**
1	I feel calm	1	2	3	4
2	I feel secure	1	2	3	4
3	I feel tense	1	2	3	4
4	I feel strained	1	2	3	4
5	I feel at ease	1	2	3	4
6	I feel upset	1	2	3	4
7	I am presently worrying over possible misfortunes	1	2	3	4
8	I feel satisfied	1	2	3	4
9	I feel frightened	1	2	3	4
10	I feel uncomfortable	1	2	3	4
11	I feel self-confident	1	2	3	4
12	I feel nervous	1	2	3	4
13	I feel jittery	1	2	3	4
14	I feel indecisive	1	2	3	4
15	I am relaxed	1	2	3	4
16	I feel content	1	2	3	4
17	I am worried	1	2	3	4
18	I feel confused	1	2	3	4
19	I feel steady	1	2	3	4
20	I feel pleasant	1	2	3	4

A.6 Inventory of callous-unemotional traits

ICU (YV)

Please read each statement and decide how well it describes you. Mark your answer by circling the appropriate number (0-3) for each statement. Do not leave any statement unrated.

		Not at all true	Somewhat true	Very true	Definitely true
1	I express my feelings openly	0	1	2	3
2	What I think is “right” and “wrong” is different from what other people think	0	1	2	3
3	I care about how well I do at school or work	0	1	2	3
4	I do not care who I hurt to get what I want	0	1	2	3
5	I feel bad or guilty when I do something wrong	0	1	2	3
6	I do not show my emotions to others	0	1	2	3
7	I do not care about being on time	0	1	2	3
8	I am concerned about the feelings of others	0	1	2	3
9	I do not care if I get into trouble	0	1	2	3
10	I do not let my feelings control me	0	1	2	3
11	I do not care about doing things well	0	1	2	3
12	I seem very cold and uncaring to others	0	1	2	3
13	I easily admit to being wrong	0	1	2	3
14	It is easy for others to tell how I am feeling	0	1	2	3
15	I always try my best	0	1	2	3
16	I apologize (“say I am sorry”) to persons I hurt	0	1	2	3
17	I try not to hurt others’ feelings	0	1	2	3
18	I do not feel remorseful when I do something wrong	0	1	2	3
19	I am very expressive and emotional	0	1	2	3
20	I do not like to put the time into doing things well	0	1	2	3
21	The feelings of others are unimportant to me	0	1	2	3
22	I hide my feelings from others	0	1	2	3
23	I work hard on everything I do	0	1	2	3
24	I do things to make others feel good	0	1	2	3

A.7 Autism quotient

AQ

Read each of the following 50 statements very carefully and state how strongly you agree or disagree with it by ticking one of the boxes next to the statements.

		Definitely agree	**Slightly agree**	**Slightly disagree**	**Definitely disagree**
1.	I prefer to do things with others rather than on my own.	1	2	3	4
2.	I prefer to do things the same way over and over again.	1	2	3	4
3.	If I try to imagine something, I find it very easy to create a picture in my mind.	1	2	3	4
4.	I frequently get so strongly absorbed in one thing that I lose sight of other things.	1	2	3	4
5.	I often notice small sounds when others do not.	1	2	3	4
6.	I usually notice car number plates or similar strings of information.	1	2	3	4
7.	Other people frequently tell me that what I've said is impolite, even though I think it is polite.	1	2	3	4
8.	When I'm reading a story, I can easily imagine what the characters might look like.	1	2	3	4
9.	I am fascinated by dates.	1	2	3	4
10.	In a social group, I can easily keep track of several different people's conversations.	1	2	3	4
11.	I find social situations easy.	1	2	3	4
12.	I tend to notice details that others do not.	1	2	3	4
13.	I would rather go to a library than a party.	1	2	3	4
14.	I find making up stories easy.	1	2	3	4
15.	I find myself drawn more strongly to people than to things.	1	2	3	4
16.	I tend to have very strong interests, which I get upset about if I can't pursue.	1	2	3	4
17.	I enjoy social chit-chat.	1	2	3	4
18.	When I talk, it isn't always easy for others to get a word in edgeways.	1	2	3	4
19.	I am fascinated by numbers.	1	2	3	4
20.	When I'm reading a story, I find it difficult to work out the characters' intentions.	1	2	3	4
21.	I don't particularly enjoy reading fiction.	1	2	3	4
22.	I find it hard to make new friends.	1	2	3	4
23.	I notice patterns in things all the time.	1	2	3	4
24.	I would rather go to the theatre than a museum.	1	2	3	4
25.	It does not upset me if my daily routine is disturbed.	1	2	3	4
26.	I frequently find that I don't know how to keep a conversation going.	1	2	3	4

		Definitely agree	Slightly agree	Slightly disagree	Definitely disagree
27.	I find it easy to "read between the lines" when someone is talking to me.	1	2	3	4
28.	I usually concentrate more on the whole picture, rather than the small details.	1	2	3	4
29.	I am not very good at remembering phone numbers.	1	2	3	4
30.	I don't usually notice small changes in a situation, or a person's appearance.	1	2	3	4
31.	I know how to tell if someone listening to me is getting bored.	1	2	3	4
32.	I find it easy to do more than one thing at once.	1	2	3	4
33.	When I talk on the phone, I'm not sure when it's my turn to speak.	1	2	3	4
34.	I enjoy doing things spontaneously.	1	2	3	4
35.	I am often the last to understand the point of a joke.	1	2	3	4
36.	I find it easy to work out what someone is thinking or feeling just by looking at their face.	1	2	3	4
37.	If there is an interruption, I can switch back to what I was doing very quickly.	1	2	3	4
38.	I am good at social chit-chat.	1	2	3	4
39.	People often tell me that I keep going on and on about the same thing.	1	2	3	4
40.	When I was young, I used to enjoy playing games involving pretending with other children.	1	2	3	4
41.	I like to collect information about categories of things (e.g. types of car, types of bird, types of train, types of plant, etc.).	1	2	3	4
42.	I find it difficult to imagine what it would be like to be someone else.	1	2	3	4
43.	I like to plan any activities I participate in carefully.	1	2	3	4
44.	I enjoy social occasions.	1	2	3	4
45.	I find it difficult to work out people's intentions.	1	2	3	4
46.	New situations make me anxious.	1	2	3	4
47.	I enjoy meeting new people.	1	2	3	4
48.	I am a good diplomat.	1	2	3	4
49.	I am not very good at remembering people's date of birth.	1	2	3	4
50.	I find it very easy to play games with children that involve pretending.	1	2	3	4

A.8 Youth psychopathic traits inventory

YPI

Instructions: This sheet consists of a number of statements that deal with what you think and feel about different things. Read each statement carefully and decide how well the particular statement applies to you. You can choose between four different alternatives on each statement. **Answer each statement as you most often feel and think, not only how you feel right now.**

· **Answer ALL statements.**
· **Do not put a mark between the alternatives.**
· **Only one answer per statement.**

IMPORTANT!!! There are no answers that are "Right" or "Wrong". You cannot score worse or better than anyone else. We are interested in what you think and feel, not in what is "Right" or "Wrong".

		Does not apply at all	**Does not apply well**	**Applies fairly well**	**Applies very well**
1.	I like to be where exciting things happen.	1	2	3	4
2.	I usually feel calm when other people are scared.	1	2	3	4
3.	I prefer to spend my money right away rather than save it.	1	2	3	4
4.	I get bored quickly when there is too little change.	1	2	3	4
5.	I have probably skipped school or work more than most other people.	1	2	3	4
6.	It's easy for me to charm and seduce others to get what I want from them.	1	2	3	4
7.	It's fun to make up stories and try to get people to believe them.	1	2	3	4
8.	I have the ability not to feel guilt and regret about things that I think other people would feel guilty about.	1	2	3	4
9.	I consider myself as a pretty impulsive person.	1	2	3	4
10.	I'm better than everyone on almost everything.	1	2	3	4
11.	I can make people believe almost anything.	1	2	3	4
12.	I think that crying is a sign of weakness, even if no one sees you.	1	2	3	4
13.	If I won a lot of money in the lottery I would quit school or work and just do things that are fun.	1	2	3	4
14.	I have the ability to con people by using my charm and smile.	1	2	3	4
15.	I am good at getting people to believe in me when I make something up.	1	2	3	4
16.	I have often been late to work or classes in school.	1	2	3	4
17.	When other people have problems, it is often their own fault, therefore, one should not help them.	1	2	3	4
18.	It often happens that I talk first and think later.	1	2	3	4
19.	I have talents that go far beyond other people's.	1	2	3	4
20.	It's easy for me to manipulate people.	1	2	3	4
21.	I seldom regret things I do, even if other people feel that they are wrong.	1	2	3	4
22.	I like to do things just for the thrill of it.	1	2	3	4
23.	It's important to me not to hurt other people's feelings.	1	2	3	4

		Does not apply at all	Does not apply well	Applies fairly well	Applies very well
24.	Sometimes I lie for no reason, other than because it's fun.	1	2	3	4
25.	To be nervous and worried is a sign of weakness.	1	2	3	4
26.	If I get the chance to do something fun, I do it no matter what I had been doing before.	1	2	3	4
27.	When someone asks me something, I usually have a quick answer that sounds believable, even if I've just made it up.	1	2	3	4
28.	When someone finds out about something that I've done wrong, I feel more angry than guilty.	1	2	3	4
29.	I get bored quickly by doing the same thing over and over.	1	2	3	4
30.	The world would be a better place if I were in charge.	1	2	3	4
31.	To get people to do what I want, I often find it efficient to con them.	1	2	3	4
32.	It often happens that I do things without thinking ahead.	1	2	3	4
33.	Pretty often I act charming and nice, even with people I don't like, in order to get what I want.	1	2	3	4
34.	It has happened several times that I've borrowed something and then lost it.	1	2	3	4
35.	I often become sad or moved by watching sad things on TV or film.	1	2	3	4
36.	What scares others usually doesn't scare me.	1	2	3	4
37.	I'm more important and valuable than other people.	1	2	3	4
38.	When I need to, I use my smile and my charm to use others.	1	2	3	4
39.	I don't understand how people can be touched enough to cry by looking at things on TV or movie.	1	2	3	4
40.	I often don't/didn't have my school or work assignments done on time.	1	2	3	4
41.	I am destined to become a well-known, important and influential person.	1	2	3	4
42.	I like to do exciting and dangerous things, even if it is forbidden or illegal.	1	2	3	4
43.	Sometimes I find myself lying without any particular reason.	1	2	3	4
44.	To feel guilty and remorseful about things you have done that have hurt other people is a sign of weakness.	1	2	3	4
45.	I don't let my feelings affect me as much as other people's feelings seem to affect them.	1	2	3	4
46.	It has happened that I've taken advantage of (used) someone in order to get what I want.	1	2	3	4
47.	I like to spice up and exaggerate when I tell about something.	1	2	3	4
48.	To feel guilt and regret when you have done something wrong is a waste of time.	1	2	3	4
49.	I usually become sad when I see other people crying or being sad.	1	2	3	4
50.	I've often gotten into trouble because I've lied too much.	1	2	3	4

A.9 Behavioural Inhibition System

BIS-11

People differ in the ways they act and think in different situations. This is a test to measure some of the ways in which you act and think. Read each statement and circle the appropriate number on the right. Do not spend too much time on any statement. Answer quickly and honestly.

		Never	Sometimes	Often	Always
1.	I plan tasks carefully.	1	2	3	4
2.	I do things without thinking.	1	2	3	4
3.	I make-up my mind quickly.	1	2	3	4
4.	I am happy-go-lucky.	1	2	3	4
5.	I don't "pay attention."	1	2	3	4
6.	I have "racing" thoughts.	1	2	3	4
7.	I plan trips well ahead of time.	1	2	3	4
8.	I am self-controlled.	1	2	3	4
9.	I concentrate easily.	1	2	3	4
10.	I save regularly.	1	2	3	4
11.	I "squirm" at plays or lectures.	1	2	3	4
12.	I am a careful thinker.	1	2	3	4
13.	I plan for job security.	1	2	3	4
14.	I say things without thinking.	1	2	3	4
15.	I like to think about complex problems.	1	2	3	4
16.	I change jobs.	1	2	3	4
17.	I act "on impulse."	1	2	3	4
18.	I get easily bored when solving thought problems.	1	2	3	4
19.	I act on the spur of the moment.	1	2	3	4
20.	I am a steady thinker.	1	2	3	4
21.	I change residences.	1	2	3	4
22.	I buy things on impulse.	1	2	3	4
23.	I can only think about one thing at a time.	1	2	3	4
24.	I change hobbies.	1	2	3	4
25.	I spend or charge more than I earn.	1	2	3	4
26.	I often have extraneous thoughts when thinking.	1	2	3	4
27.	I am more interested in the present than the future.	1	2	3	4
28.	I am restless at the theatre or lectures.	1	2	3	4
29.	I like puzzles.	1	2	3	4
30.	I am future oriented.	1	2	3	4

A.10 Neighbourhood Environment Scale

NES

The following statements are about your neighbourhood (where you live). Please indicate whether they are true or false by circling one of the numbers on the right.

		True	False
1	Within walking distance of my house there is a park or playground where I like to walk and enjoy myself, playing sports or games.	1	0
2	There are plenty of safe places to walk or play outdoors in my neighbourhood.	1	0
3	Every few weeks, some kid in my neighbourhood gets beat-up or mugged.	1	0
4	Every few weeks, some adult gets beat-up or mugged in my neighbourhood.	1	0
5	In my neighbourhood, I see signs of racism and prejudice at least once a week.	1	0
6	In my neighbourhood, many yards and alleys have broken bottles and rubbish lying around.	1	0
7	I have seen people using or selling drugs in my neighbourhood.	1	0
8	In the morning or later in the day, I often see drunk people on the street in my neighbourhood.	1	0
9	Most adults in my neighbourhood respect the law.	1	0
10	There are abandoned or boarded-up buildings in my neighbourhood.	1	0
11	I feel safe when I walk around my neighbourhood by myself.	1	0
12	The people who live in my neighbourhood often damage or steal each other's property.	1	0
13	The people who live in my neighbourhood always take care of each other and protect each other from crime.	1	0
14	Almost every day I see homeless people walking or sitting around in my neighbourhood.	1	0
15	In my neighbourhood, the people with the most money are the drug dealers.	1	0
16	In my neighbourhood, there are a lot of poor people who don't have enough money for food and basic needs.	1	0
17	For many people in my neighbourhood, going to church on Sunday or religious days is a very important activity.	1	0
18	The people who live in my neighbourhood are the best people in the world.	1	0

A.11 Behavioural inhibition/activation scales

BIS/BAS

Each item of this questionnaire is a statement that a person may either agree with or disagree with. For each item, indicate how much you agree or disagree with what the item says. Please respond to all the items; do not leave any blank. Choose only one response to each statement. Please be as accurate and honest as you can be. Respond to each item as if it were the only item. That is, don't worry about being "consistent" in your responses. Choose from the following four response options:

1 = very true for me 2 = somewhat true for me 3 = somewhat false for me 4 = very false for me

1	A person's family is the most important thing in life.	1	2	3	4
2	Even if something bad is about to happen to me, I rarely experience fear or nervousness.	1	2	3	4
3	I go out of my way to get things I want.	1	2	3	4
4	When I'm doing well at something I love to keep at it.	1	2	3	4
5	I'm always willing to try something new if I think it will be fun.	1	2	3	4
6	How I dress is important to me.	1	2	3	4
7	When I get something I want, I feel excited and energized.	1	2	3	4
8	Criticism or scolding hurts me quite a bit.	1	2	3	4
9	When I want something I usually go all-out to get it.	1	2	3	4
10	I will often do things for no other reason than that they might be fun.	1	2	3	4
11	It's hard for me to find the time to do things such as get a haircut.	1	2	3	4
12	If I see a chance to get something I want I move on it right away.	1	2	3	4
13	I feel pretty worried or upset when I think or know somebody is angry at me.	1	2	3	4
14	When I see an opportunity for something I like I get excited right away.	1	2	3	4
15	I often act on the spur of the moment.	1	2	3	4
16	If I think something unpleasant is going to happen I usually get pretty “worked up”.	1	2	3	4
17	I often wonder why people act the way they do.	1	2	3	4
18	When good things happen to me, it affects me strongly.	1	2	3	4
19	I feel worried when I think I have done poorly at something important.	1	2	3	4
20	I crave excitement and new sensations.	1	2	3	4
21	When I go after something I use a "no holds barred" approach.	1	2	3	4
22	I have very few fears compared to my friends.	1	2	3	4
23	It would excite me to win a contest.	1	2	3	4
24	I worry about making mistakes.	1	2	3	4

A.12 Debriefing statement

Debriefing Statement *(written)* (Version 1, 30/05/2012)

Study Title: ***The impact of attention on behavioural difficulties during adolescence.***

We would like to take this opportunity to *thank you* for taking part in our study. We are very appreciative of the time you have given up to take part. The aim of this research is to investigate why people with attention problems often show behavioural difficulties during adolescence.

You were asked to attend the School of Psychology to take part in computerised tasks measuring your ability to stop your responses, memorise patterns of shapes, make decisions based on delay and reward, recognise facial expressions and understand another person's way of thinking. These tasks will help us to better understand the relationship between attention problems and behavioural difficulties.

You were also asked to fill in questionnaires regarding your ability to concentrate, lifestyle, personality.

Once again, let us remind you that the results of this study will not include any identifying details and that your data will be number coded and treated as confidential. If you have any further questions regarding the study, please contact me, Nadia Peppa, nadia.peppa@soton.ac.uk , 02380 594594.

Thank you for your participation in this study.

Name _____ Date _____

Signature _____

If you have questions about your rights as a participant in this research, or if you feel that you have been placed at risk, you may contact the Head of Research Governance, Dr Martina Prude (02380 595058, mad4@soton.ac.uk) who will be happy to help.

If you require further advice and support after the project you can contact 'No Limits', a charity that offers information, counselling and support for young people under 26 living in Southampton and Hampshire. Their contact details are: 023 8022 4224 and http://www.nolimitshelp.org.uk/home.

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