

**Attention and the specificity of emotion processing
impairments in adolescents with a diagnosis of
Attention-Deficit/Hyperactivity Disorder with or
without comorbid Conduct Disorder**

by

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Thesis submitted for the degree of Doctor of Philosophy

2017



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Summary of Thesis

Impairments in emotion processing have been separately associated with Attention-Deficit/Hyperactivity Disorder (ADHD) and Conduct Disorder (CD). However, due to their common co-occurrence it is unclear if these impairments are general to ADHD or are specific to ADHD with comorbid CD. In addition, the extent to which they are associated with ADHD or CD symptom severity or callous-unemotional (CU) traits is uncertain and the role atypical attentional allocation to facial features plays in these impairments also remains poorly understood.

Participants with a diagnosis of ADHD either with or without comorbid CD performed four emotion processing tasks. Participants with ADHD and comorbid CD showed impairments in all emotion processing tasks compared to ADHD alone. They showed specific impairments in both the conscious recognition and automatic processing of fear faces, general impairments across emotion categories in affective (but not cognitive) empathy, and both reduced differential fear conditioning and a faster rate of extinction learning than ADHD alone. Groups did not differ in processing of the eye region of faces during emotion recognition or empathy tasks, suggesting other mechanisms may account for differences in emotion processing. As opposed to CD severity or CU traits, ADHD severity was the only predictor of reduced attention to the eye region. Further, while CD severity was most strongly negatively associated with both the conscious recognition and automatic processing of fear faces, and to affective empathy for happiness, CU traits were strongly associated with the ability to affectively empathise with negative emotions. The findings of the current thesis highlight the importance of assessing emotion processing and clinical heterogeneity in those with ADHD in order to offer appropriate and tailored interventions.

Acknowledgements

First and foremost I would like to thank my supervisor, Professor Stephanie van Goozen for your support, encouragement, and patience. Thank you for having the faith to take me on and allowing me the flexibility to develop skills both in research and related fields during the course of my PhD. Thanks also go to Professor Anita Thapar and Dr Kate Langley for their expertise and valuable feedback during all stages of the PhD.

A huge thanks must go to Zoe, Laura, Amy, Olga, Sharifah, Ellie, Sophie and Tom for your help with data collection and thank you to Val for your optimism and perseverance in recruitment. Thanks to everyone I've had the pleasure of sharing an office with over the three years. Thank you for the conversation and for providing a listening ear, allowing my sometimes incoherent thoughts to transition into something resembling understandable prose.

Thank you to Emily for your love, understanding, encouragement and continued tolerance of the student lifestyle. Thanks for always being a reminder to continue living life outside my PhD. A special thanks to Mum and Dad for always being there, and for instilling in me a curiosity for the world. Thanks to the rest of my family and friends for their support and for all being different, and therefore posing questions that require answers.

Thank you to Cardiff University School of Psychology and the Economic and Social Research Council for funding my studentship, without such financial support I would have been unable to carry out this research.

Thank you to all the schools, teachers, and inclusion officers that helped me in Newport, Wales for room provisions and your assistance in recruiting participants, you really made my control testing run smoothly. Last but not least, I am hugely grateful to all the adolescents and their families for taking part. Without your willingness to help this research would not have been possible, thank you.

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List of Abbreviations

ACC	Anterior cingulate cortex
ACQ	Acquisition
ADHD	Attention-Deficit/Hyperactivity Disorder
ANOVA	Analysis of Variance
AOI	Area of Interest
APA	American Psychiatric Association
ASD	Autism Spectrum Disorders
CD	Conduct Disorder
CEPS	Cardiff Emotional Pictorial Stroop
CESS	Cardiff Empathy Scoring System
CS	Conditional stimulus
CU	Callous and unemotional
dACC	Dorsolateral anterior cingulate cortex
DAWBA	Development and Well Being Assessment
DBD	Disruptive Behaviour Disorders
DLPFC	Dorsolateral prefrontal cortex
DSM	Diagnostic and Statistical Manual of Mental Disorders
EF	Executive function
ER	Emotion regulation
EXT	Extinction
FER	Facial Emotion Recognition
HC	Healthy controls
HR	Heart rate
Hz	Hertz
ICD	International Classification of Diseases
IES	Integrated Emotion Systems
IQ	Intelligence Quotient
NICE	The National Institute for Health and Care Excellence
NIMH	National Institute of Mental Health
OCD	Obsessive Compulsive Disorder
ODD	Oppositional Defiant Disorder
OFC	Orbitofrontal cortex
PFC	Prefrontal cortex
PTSD	Post-Traumatic Stress Disorder
SCR	Skin conductance response
SDQ	Strengths and Difficulties Questionnaire
SES	Socioeconomic status
TFF	Time to first fixation
UG	Ultimatum game

UK	United Kingdom
UR	Unconditional response
US	Unconditional stimulus
VIM	Violence Inhibition Mechanism
vmPFC	Ventromedial prefrontal cortex
WASI	Wechsler Abbreviated Scale of Intelligence
YO	Young offender
YOS	Young Offending Services

1. Chapter 1: Introduction

1.1. Introduction

Children and adolescents with a diagnosis of Conduct Disorder (CD) display a persistent pattern of antisocial behaviour that results in significant costs to the individual, their family members and society at large (Scott, Knapp, Henderson, & Maughan, 2001). This is apparent both during childhood and adolescence but also when these individuals reach adulthood. While for many, the antisocial behaviour central to those with conduct disorder persists through much of their adult lives, the fact that a large number of individuals do not show such a persistence both through adolescence and subsequently adulthood raises the possibility of developing effective interventions to help these individuals. While parenting interventions have shown promise (Kazdin, 2016), they are limited by their cost and reliance of co-operative parents. As a result, focus in the literature has shifted to developing interventions based on specific underlying aetiological factors and mechanisms responsible for the observed impairments apparent in those with the disorder (Hubble, Bowen, Moore, & van Goozen, 2015).

However, CD is rarely an isolated disorder and comorbidities are frequently observed. One of particular prevalence, and relevance to the current thesis, is that of Attention-Deficit Hyperactivity Disorder (ADHD) which is found in nearly half of those with CD (National Resource Centre on ADHD, 2004). Due to this overlap, the identification of such aetiological factors and mechanisms to target with interventions is made more complicated due to uncertainty as to which are specifically associated with the additional CD of those with ADHD and which are more generally related to ADHD itself. A specific set of impairments which have been identified in both those with ADHD and CD separately are emotion processing impairments; both in terms of the processing of the emotions of others and the

appropriate evocation and regulation of emotion in the individual themselves.

However, studies have rarely accounted for the above co-occurrence. Therefore in those studies in which impairments have been found in those with CD, it is not clear if these are in fact driven by comorbid ADHD, while in studies which have found impairments in ADHD, it is not clear if these are in fact driven by comorbid CD. Further, associated issues found in those with CD is the presence or not of callous and unemotional (CU; Hawes & Dadds, 2005) traits, as well as the severity of the CD symptoms themselves, which too have been implicated in emotion processing impairments. Therefore further research is warranted to identify the impairments which are specifically associated with each aspect of these clinical presentations.

In order to provide a more comprehensive account of the background and underlying issues above, the current chapter will first define and describe CD and its associated problems, then give an overview of current interventions and their limitations. Following this, it will define and describe ADHD, and subsequently outline the aetiological factors found to be important in predicting both CD and ADHD. It will then highlight the extent to which their aetiologies differ as well as describing the shared and specific impairments found in CD and ADHD. Next, an overview will be provided of the key theories that have attempted to make sense of the antisocial behaviour which characterises those with a diagnosis of CD.

Penultimately, the areas of emotion processing in which to date the literature is unclear as to whether impairments are specific to additional CD or more generally a problem for ADHD, will be outlined. Specifically, emotion recognition, empathy, emotion regulation and fear conditioning as well as the role attention plays in such impairments. These areas form the focus of the current thesis.

The chapter will conclude by outlining the aims and hypotheses of the thesis.

1.1.1. Conduct Disorder

A diagnosis of Conduct Disorder (CD) is given when an individual displays a repetitive and persistent pattern of behaviour in which the basic rights of others, or major age appropriate social norms are violated. The diagnostic criteria include behaviours such as aggression to people or animals, the destruction of property, dishonesty and theft or the serious violation of rules. Importantly, for a diagnosis to be given, symptoms need to have been present in the preceding 12 months and result in a clinically significant impairment in either social, academic or occupational functioning (American Psychiatric Association (APA), 2013).

In the most recent edition of the diagnostic and statistical manual (DSM-V; APA, 2013) there are two specifiers that further define individuals characterised by such problems. The first relates to the age on onset. Individuals who show at least one of the symptoms of CD before the age of 10 are specified as a childhood-onset type; while those who do not display such symptoms until after the age of 10 are specified as an adolescent-onset type. Further, when diagnosing those with CD clinicians now also specify whether the individual displays a lack of prosocial emotions (also synonymously labelled Callous and Unemotional (CU) traits), including a lack of remorse or guilt, a lack of empathy, being unconcerned about their performance in work or other important activities, or displays shallow or deficient affect.

Given the severity of behaviours displayed in those with CD, it is perhaps not surprising that these come with significant costs to the individual, their immediate family and social networks, and to society at large. The prognosis of those with CD is typically poor. The list of negative outcomes for these individuals is long and includes antisocial and criminal behaviour (Carey, 2000; Dretzke et al., 2009),

psychiatric disorders, drug and alcohol abuse, higher rates of hospitalisation and mortality, higher rates of school dropout, lower levels of educational attainment, greater unemployment, family breakdown and intergenerational transmissions of conduct problems (Broidy, Nagin, & Tremblay, 2003; Burke, Loeber, & Birmaher, 2002; Farrington & Welsh, 2007; Loeber, Burke, & Lahey, 2000; Moffitt, 1993). The economic cost of those with CD is also significant. Adolescents with CD are more likely to require medical help at primary and secondary school, and will make significantly more use of primary care services (Edwards, C  illeachair, Bywater, & Hughes, 2007; McGroder & Hyra, 2009). It has been estimated that by the age of 28 years, the cost to health, social, educational and legal services is 10 times higher in individuals with a clinical diagnosis of CD (£70,000) than in those without such problems (£7423), while even those who demonstrate conduct problems at a sub-clinical threshold cost 3.5 times more (Scott et al., 2001). A more recent study estimated the annual cost of those with CD to be £6000 (Romeo, Knapp, & Scott, 2006) with a large percentage of this falling on the immediate family of the individual. Such costs are not specific to the United Kingdom, as studies from both the USA (Foster & Jones, 2005) and elsewhere in Europe (Bachmann, 2011) highlight the increased provisions required. Unsurprisingly, these costs are not limited to the individual in childhood or adolescence. Research has shown that 80% of all crimes in England and Wales are committed by those who at some point would have met criteria for a diagnosis of CD, while Loeber, Burke, and Lahey (2002) found that 82 – 90% of adults with Antisocial Personality Disorder met criteria for CD during their adolescent years.

When the prevalence of CD is taken into account, the societal impact is further underlined. It has been estimated that in the UK, approximately 1.5% of

children and adolescents meet criteria for CD (Goodman, Force, & Meltzer, 2002), while rates between 2 and 3.3% are observed in studies in the US (Roberts, Roberts, & Xing, 2007). In attempting to determine the global burden of CD, Erskine et al., (2014) estimated that CD was responsible for 5.75 million years lived with a disability globally, which accounted for 0.80% of all total years lived with disability. Given its prevalence, again it is perhaps unsurprising that CD is the most common reason for referral to child and adolescent mental health services (National Institute of Health and Care Excellence (NICE), 2013; Reid, 1993).

Despite the above prognoses, research has demonstrated that not all individuals who are diagnosed with CD in childhood or adolescence go on to behave antisocially in their adult life (Odgers et al., 2007), raising the possibility of developing effective interventions. The British government is now recognising the need to deal with antisocial behaviour by tackling the causes by funding the delivery of evidence-based programmes (Bywater et al., 2009).

1.1.2. Current Interventions

A large proportion of current interventions for those with CD target aspects of parenting (Lundahl, Risser, & Lovejoy, 2006; Reyno & McGrath, 2006) such as parent management training (PMT; Reid, Patterson, & Snyder, 2002) and the Incredible Years BASIC parenting programme (Webster-Stratton, 1981). Such interventions work on the premise that behaviours associated with CD are developed and sustained in the child's home by maladaptive parent-child interactions. Parents may unwittingly increase the likelihood of such behaviour through direct reinforcement, using ineffective commands and harsh punishment and failing to attend to appropriate behaviour (Kazdin, 2016). The treatment employs learning based procedures such as modelling, prompting, shaping, positive reinforcement,

extinction and mild punishment in order to develop more desirable behaviours. There is evidence to show that these treatments are effective, resulting in reductions in oppositional behaviour, aggression, and antisocial behaviour, while increases in prosocial behaviour have also been found (Kazdin, 2016). However, they are also costly, with PMT consisting of five to ten weekly sessions of up to 60 minutes each. In addition, these interventions are not effective for everyone. Despite a significant reduction overall, when examining changes at the individual level, Bywater et al., (2009) found that a parenting intervention only produced minimum significant change in 63% of children, while there is also evidence that parenting interventions are not always successful, especially when exploring long-term outcomes (Serketich & Dumas, 1996). Further, as is clear from the content of the interventions, their success is very much dependent on the commitment of the parent, and parental engagement is not always steadfast. For example, Kazdin (1996) suggests that in general 40-60% of parents drop out of treatment early. While even for those who engage in treatment, there are factors that moderate the degree of therapeutic change observed in the children, such as stressors that compete with participating in treatment, perceived treatment demands and perceived relevance of treatment (Kazdin, 2016). Further, the parent-therapist alliance has been found to predict the effectiveness of treatment (Kazdin & Durbin, 2012; Kazdin, Marciano, & Whitley, 2005; Kazdin, Whitley, & Marciano, 2006).

Because of the lack of universal effectiveness of blanket, parent-based treatments, researchers and clinicians have advocated tailoring interventions to address specific symptoms of the child with conduct problems as well as the specific aetiological factors which have resulted in their current presentation (Ng & Weisz, 2016; van Goozen, Fairchild, Snoek, & Harold, 2007). However, the challenge of

developing tailored interventions is made more complicated given the fact that CD is rarely an isolated disorder. Other disorders that are common amongst individuals with CD include substance use disorder, and anxiety disorders (Drabick, Gadow, & Sprafkin, 2006; Milin, Halikas, Meller, & Morse, 1991; Nock, Kazdin, Hiripi, & Kessler, 2006), and perhaps most commonly (and importantly for the current thesis), Attention-Deficit/Hyperactivity Disorder (ADHD; Biederman, 2005) .

1.1.3. Attention-Deficit/Hyperactivity Disorder (ADHD)

Attention-Deficit/Hyperactivity Disorder (ADHD), like CD, is one of the most common developmental disorders found in childhood, with prevalence estimates ranging between 3 and 9% (NICE, 2008). To receive a diagnosis of ADHD one must demonstrate a persistent pattern of inattentive and/or hyperactive-impulsive behaviour that gets in the way of daily functioning. ADHD is also further divided into three subtypes: an inattentive type which includes symptoms such as difficulty concentrating, disorganisation and being easily distracted, a hyperactive-impulsive presentation characterised by excessive movement/noise and problems controlling impulses, and a combined subtype characterised by symptoms of both (DSM-V; APA, 2013). Such symptoms of ADHD will typically present themselves across multiple settings resulting in behavioural, academic, and social impairments. ADHD is also not only confined to childhood and adolescence, with studies finding that approximately 60% of children with ADHD will continue to experience significant symptoms into adulthood (Kessler et al., 2005).

1.1.4. Overlap between CD and ADHD

ADHD and CD often co-occur. It has been estimated that CD is comorbid in 25% of children and 45% of adolescents with ADHD (National Resource Centre on ADHD, 2004), while children are 12 times more likely to have ADHD or CD if they have the

other disorder (Romano, Tremblay, Farhat, & Cote, 2006). In addition, until recently ADHD was classified as a Disruptive Behaviour Disorder (DBD) alongside CD and Oppositional Defiant Disorder (ODD). Such is the high rate of comorbidity between the disorders, many psychologists questioned if they were in fact distinct syndromes and instead represented subtypes of the same disorder (Hinshaw, 1987). However, this outdated perception of ADHD has now been replaced and it is currently viewed as a neurodevelopmental disorder alongside autistic spectrum disorders (ASD) and Dyspraxia (DSM 5; APA, 2013). Neurodevelopmental disorders result from structural differences in the brain as a result of developmental alternations which lead to a number of developmental delays (Frick & Nigg, 2012; van Herwegen & Riby, 2014). On the other hand, CD is classified as a behavioural disorder which can result from a number of potential causes, such as biological factors and environmental and social exposures (Murrihy, Kidman, & Ollendick, 2010). The onset of CD is typically later than ADHD (Frick & Nigg, 2012; Loeber & Keenan, 1994) and while both disorders share certain temperamental characteristics, the later onset of CD perhaps suggests that certain correlates of ADHD may predispose children for developing CD (Steinberg & Drabick, 2015). There is evidence that those with ADHD and accompanying childhood conduct problems are clinically distinctive (Silberg, Moore, & Rutter, 2015). This group shows greater symptom severity than those with either of the disorders alone. Their prognosis is also considerably poorer with significantly higher rates of criminality and antisocial behaviour in adulthood (Lynam, 1996). Interestingly, for this group who receive a diagnosis of both disorders, inherited factors have been found to be more important in explaining their behaviour than they are in explaining the behaviour of those who have either disorder in isolation (Faraone, 2000; Thapar, Harrington, & McGuffin,

2001).

Because of the overlap between CD and ADHD. It is important to consider the aetiological factors that lead to ADHD and the expression of ADHD symptoms and those that separately lead some of those with ADHD to further develop CD. The next section will consider causal factors presented in the literature which have attempted to explain ADHD and the antisocial behaviour that characterises CD symptomology.

1.1.5. Deficits Leading to ADHD

ADHD is primarily thought to be a disorder characterised by problems in executive functioning (EF; Barkley, 1997). Barkley (1997) argued such problems result from deficits in behavioural inhibition. Specifically, it is argued that behavioural disinhibition reduces the effectiveness of working memory, self-regulation, affective motivation arousal and internalisation of speech, and reconstitution (also known as self-play, which assists in private planning and problem solving). Barkley also later added self-awareness to the model (Barkley, 2012). Deficits in these functions in those with ADHD are thought to lead to problems in motor control, such as inhibiting task-irrelevant responses, and the execution of goal-directed responses, novel complex tasks, motor sequences and goal-directed persistence, while they also lead to a lack of sensitivity to response feedback, difficulty re-engaging in a task following a disruption, and control of behaviour by internally represented information (Antshel, Hier, & Barkley, 2014). Indeed, there is evidence of behavioural disinhibition in those with ADHD (Nigg, 2001), while research has also supported that those with ADHD have problems with non-verbal working memory, sustained attention, timing, and forethought, and deficits in privatisation of speech (Berk & Potts, 1991; Frazier, Demaree, & Youngstrom, 2004; Rapport et al., 2008;

Rubia, Smith, Taylor, & Brammer, 2007; Winsler, Diaz, Atencio, Mccarthy, & Chabay, 2000). The above deficits also lead to problems in emotional and motivational self-regulation. Those with ADHD display greater impulsive emotional expressions in their reactions to events, less objectivity in the selection of a response to an event, and reduced ability to adopt the perspective of another due to their lack of ability to delay their emotional reaction long enough to consider the other's point of view (Barkley, 1997). In addition, they display greater problems in self-soothing, self-distracting and ability to modify their attention to the emotionally provocative event to attenuate its impact (Barkley, 1997). They also have a reduced ability to construct a more socially appropriate and moderate emotion that would be supportive of their long-term welfare (Antshel et al., 2014). Together, and in general, such deficits mean that an individual with ADHD is more greatly influenced by external events rather than their own mental representations which result in them pursuing immediate gratification over deferred rewards. Nevertheless it should also be noted that not all those with a diagnosis of ADHD experience executive function deficits (Boonstra, Oosterlaan, Sergeant, & Buitelaar, 2005; Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005).

The deficits above are thought to be the result of structural and functional deficits in the key areas of the prefrontal cortex (PFC; Arnsten & Pliszka, 2011), caudate (Semrud-Clikeman et al., 2000), cerebellum (Berquin et al., 1998; Semrud-Clikeman et al., 2000) and cerebellar vermis (Berquin et al., 1998; Mostofsky, Reiss, Lockhart, & Denckla, 1998). The neurotransmitters dopamine and norepinephrine are responsible for communication within this network of brain regions, and the appropriate balance of these neurotransmitters is crucial for optimal functioning of the PFC (Pliszka, 2005). It is thought that a disruption in this balance leads to ADHD

symptomology and the medication which alleviates ADHD symptoms works by restoring this balance (Sharma & Couture, 2014).

1.1.6. Deficits Leading to CD

The aspect of functioning that most research is now starting to highlight as responsible for the antisocial behaviours displayed by those with CD is impairments in emotion processing. The central emotion that is most often found to be associated with these deficits is the processing of fear (e.g. Blair, Budhani, Colledge, & Scott, 2005).

The ability to perceive and express emotions is vital for social functioning as it allows one to be receptive to the intentions and needs of others and respond appropriately, helping maintain important social relationships (Fischer & Manstead, 2008). As a result, deficits in emotional processing can have negative consequences both for the individual with said deficits, and for those with whom they interact. One important consequence of such deficits is antisocial behaviour (Baumeister & Lobbestael, 2011). A number of academics have theorised which specific deficits are present in those displaying antisocial behaviour and how these deficits come to result in said behaviour. These will be reviewed below along with evidence that they are present in those with CD. Before that however, it is important to highlight that this thesis will predominantly be focusing on potential neurobiological and physiological mechanisms that explain antisocial behaviour in those with ADHD and CD. Nevertheless, as is clear from the success, in certain cases, of parental based treatments described above, environmental factors are also hugely important to explanations of antisocial behaviour (see box 1). These will not be the focus of the current thesis, but will be discussed where appropriate.

Box 1. The importance of environmental factors and their interaction with neurobiological mechanisms

There are a number of environmental factors that increase risk of the antisocial behaviour observed in those with conduct disorder. Such factors include economic problems in the household (Moffitt, Caspi, Rutter, & Silver, 2001), living in a low socioeconomic status and living in a high crime neighbourhood (Farrington, 1998), parents being criminally active (Farrington, 2000), family conflict (Wells & Rankin, 1991), poor parenting methods (such as punitive discipline; Frick, Lahey, & Loeber, 1992; Simons, Wu, Conger, & Lorenz, 1994; Stormshak, Bierman, McMahon, & Lengua, 2000), child abuse (Fergusson, Horwood, & Lynskey, 1996; Stouthamer-Loeber, Loeber, Homish, & Wei, 2001), associating with deviant peers (Ary, Duncan, Duncan, & Hops, 1999), academic underachievement (Maguin & Loeber, 1996), and experience of daily stressors (Mathijssen, 1999).

The interaction between environmental exposure and neurobiological and child specific factors are also hugely important (van Goozen et al., 2007). It is clear from all studies on risk of antisocial behaviour that interaction effects are crucial given that there are rarely studies that find risk factors lead to antisocial behaviour in 100% of cases. It is likely that neuropsychological risk is a two stage process. For example, it may be that an individual is marked by deficits in empathic responding, and yet due to very effective parenting, socialisation may still be able to be achieved. Had another parenting strategy been used, antisocial behaviour may have developed (Raine, 2002a; van Goozen et al., 2007). A striking example of environmental and neuropsychological risk interacting comes from a studies exploring the association between poor fear conditioning and aggression. Raine & Venables (1981) found poor fear conditioning among antisocial children

from a higher social class, but not in antisocial children from a relatively lower social class. It is thought that in the high social class group, a number of other risk factors were absent which leads to poor fear conditioning being a more prominent factor explaining antisocial behaviour, while in the low social class group other risk factors more strongly explained antisocial behaviour so the impact of poor conditioning was less important. This is not an isolated finding, with a review (Raine, 2002b) describing 39 empirical studies noting interactions between environment and neurobiology. In this review, two main themes emerged. First when neurobiological and environment factors are grouping variables and antisocial behaviour is the outcome, then the presence of both risk factors exponentially increases rates of antisocial and violent behaviour (Raine, Brennan, & Mednick, 1994). On the other hand, when the neurobiological measure is the dependent variable, environmental factors are found to moderate the relationship between neurobiological factors and antisocial behaviour, such that the relationships are strongest for those from benign home backgrounds. Such a pattern of results has been labelled the “social push perspective” (Raine, 2002, p. 314). According to this idea, the relation between antisocial behaviour and biological risk factors is stronger when adverse social circumstances are absent, as in those cases the influence of environmental factors is minimised and therefore neurobiological predispositions can better explain why someone will engage in antisocial behaviour. On the other hand, when adverse environmental conditions are present, this is more likely to emerge as a significant relationship with antisocial behaviour.

The mechanisms underlying antisocial behaviour are important to outline due to the centrality of antisocial behaviour in the diagnosis of CD. As described earlier, a diagnosis of CD is given when a child or adolescent displays behaviours such as aggression to people or animals, the destruction of property, theft and the violation of rules. These are all behaviours which can be considered antisocial. Indeed, definitions of antisocial behaviour in relation to the legal realm are behaviours that would be defined as criminal and result in incarceration. Many of the diagnostic criteria of CD would be considered criminal in specific contexts. In addition, in the literature, antisocial behaviour in general has been operationalised in terms of clinical disorders, of which CD is one (antisocial personality disorder (ASPD) and psychopathy are others; Morgan & Lilienfeld, 2000). Further, criminality and delinquency are significantly correlated with clinical disorders related to antisocial behaviour (Moffitt, 1988). Therefore, theories of antisocial behaviour in general are important to consider as they offer clues as to potential mechanisms which could be targeted with interventions for those with CD.

1.1.7. Low Arousal Theories of Antisocial Behaviour

Individuals who engage in antisocial behaviour are supposed to have chronically low levels of physiological arousal (Raine, 1996; van Goozen et al., 2007). Evidence for this comes from numerous measures of autonomic and central nervous system functioning including skin conductance activity (Fowles, 1993; Raine, Venables, & Williams, 1990a), heart rate (Ortiz & Raine, 2004), resting EEG (Raine, Venables, & Williams, 1990b), and hypothalamic-pituitary-adrenal axis (HPA) activity (McBurnett et al., 1991; van Goozen et al., 1998). There are three differing perspectives on how such low arousal leads to antisocial behaviour; sensation-

seeking theory (Zuckerman, 1979), the low fear hypothesis (Raine, 2002), and the somatic marker hypothesis (Bechara & Damasio, 2005).

1.1.7.1. Sensation seeking.

The sensation seeking theory (Zuckerman, 1979) proposes that there is an optimal level of arousal which an individual can experience and being below this optimal level is an aversive state in which individuals are motivated to eliminate. As a result, those with tonically low levels seek out stimulation to raise their arousal levels. One such source of stimulation is antisocial behaviour. Support for this theory comes from El-Sheikh, Ballard, and Cummings (1994), who found that preschool boys who choose to watch videos of people showing intense anger, had lower resting heart rates in comparison to those that chose to watch videos of a more mild depiction of anger. In addition, these boys who were more sensation seeking also demonstrated increased levels of externalising behaviour. Furthermore, longitudinal data also support this theory. Raine, Venables, & Mednick (1997) found that the resting heart rate of children at age 3 years predicted sensation seeking and aggression when they were 11 years old. Further, Gower and Crick (2011) found that both lower systolic and diastolic blood pressure and lower heart rate predicted increased engagement in classroom physical and relational aggression. A recent systemic review and meta-analysis of 114 studies (Portnoy & Farrington, 2015) also suggests that the relationship between low resting heart rate and antisocial behaviour is highly replicable. Many of the studies in the review explored this relationship in those with CD, 11 of which found a negative relationship between low resting heart rate and antisocial behaviour, while 5 found a positive relationship (low resting heart rate was associated with reduced antisocial behaviour), therefore the finding is not always present. Further, sensation seeking is not a parsimonious explanation for antisocial

behaviour, as there are many activities that one can engage in that are stimulating and yet not antisocial. Therefore, a sensation seeking disposition must interact with other factors.

1.1.7.2. Low fear hypothesis.

The low fear hypothesis was originally put forward by Lykken (1957, 1995) to explain the development of psychopathy (although it has also been used to explain antisocial behaviour in general; Raine, 2002b). Lykken coined the term ‘fear quotient’ to refer to an individual’s temperamental fearfulness. In his model, fear is described as “an aversive state that elicits arousal and escape behaviour (Lykken, 1995, p. 135). Therefore an individual’s fear quotient represents the extent of arousal that is experienced, and this is variable across individuals. A fear reaction to some stimuli is innate, such as an unexpected loud noise, while fear of less explicitly aversive stimuli is learned via the conditioning process. When a behaviour leads to an aversive outcome, an association is made between the act and the outcome. As a result, when an individual has an impulse to commit this act, fear will be elicited due to its previous pairing with the aversive outcome (Lykken, 1995) and therefore will be likely to avoid the act in the future. If the individual avoids the act they will also receive negative reinforcement due to the avoidance of the aversive state, which will increase the likelihood of further avoidance in the future. Psychopaths (who are characterised by a deficient experience of emotion, an arrogant and deceitful interpersonal style, and behaviours characterised by impulsivity and irresponsibility, and importantly elevated level of antisocial behaviour; Cooke & Michie, 2001) are argued to show a low fear quotient, and therefore a reduced experience of fear. This low fear quotient means psychopaths will be less likely to avoid the punished act. Such a deficit means that punishment strategies following antisocial behaviour are

ineffective which also creates difficulties in the socialisation processes that rely on learning through punishment (Lykken, 1995) and a lack of socialisation may lead to poor conscience development (Raine, 1993). Importantly, this deficit is argued to be specific to fear, rather than an attenuated experience of emotion in general (Lykken, 1995). The low fear theory's relevance to antisocial behaviour in childhood is supported by research findings that low autonomic nervous system (ANS) and hypothalamic-pituitary-adrenal (HPA) axis arousal provides the groundwork for a disinhibited, fearless temperament in infancy and childhood (Baker, Shelton, Baibazarova, Hay, & van Goozen, 2013; Kagan, Reznick, & Snidman, 1987; Scarpa, Raine, Venables, & Mednick, 1997). Such low levels of arousal have also been argued to reflect a more general risk factor for antisocial behaviour because those with low arousal will have problems being attentive to and responding to environmental cues that help with decision making and guiding behaviour. Such a relationship has been conceptualised in the somatic marker hypothesis (Bechara & Damasio, 2005).

1.1.7.3. The somatic marker hypothesis.

The somatic marker hypothesis (Bechara & Damasio, 2005) is based on the assumption that there is a cortical-subcortical system (which includes the frontal lobes and the limbic system) involved in decision making. Such decision making is influenced to a large extent by affect processes. The "marker" described by the somatic marker hypothesis is an emotional-visceral marker that guides experience and as a result influences decision making. The production of the somatic marker is thought to be generated by the ventromedial prefrontal cortex (vmPFC) and/or the amygdala due to research showing that damage to these areas results in an inability to experience somatic states associated with both positive and negative affect (Dinn

& Harris, 2000). These states are expressed as changes in bodily arousal (e.g. changes in heart rate, skin conductance).

The prefrontal cortex is argued to be the primary region that integrates all sensory and affective information into a cognitive amalgam (Damasio, 1996; Damasio, Tranel, & Damasio, 1990). When people are exposed to situations which involve elements that have previously been organised and classified affectively, these past markers are activated to guide subsequent cognition and decision making. For example, an experience already marked as 'bad' produces a somatic state (e.g. increased heart rate) that acts as a signal in guiding subsequent experience. In the case of this negative marker the result may be to avoid the situation. Applying this to antisocial acts, in a healthy individual, when faced with a decision to commit an antisocial act, there will be activation of the association between the antisocial act and a previous associated punishment and therefore result in emotional aversion.

Evidence for the somatic marker hypothesis comes from studies that have explored patients' with vmPFC and amygdala lesions (areas known to be involved in emotion processing; Sobhani & Bechara, 2011) who performed the IOWA gambling task (Bechara, Tranel, & Damasio, 2000). This task requires participants to choose between decks of cards which either result in large immediate rewards and high future losses (disadvantageous decks) or decks which result in lower immediate gain but smaller future loss (advantageous decks). The typical finding on this task is that healthy control participants (without vmPFC or amygdala lesions) have a preference for the advantageous decks, while participants with the above lesions prefer the disadvantageous decks (Bechara, Tranel, & Damasio, 2000). Skin conductance activity was also measured while participants completed this task. Interestingly, control participants began to generate anticipatory skin conductance responses prior

to selecting a card, which suggests that they were beginning to react to imagined scenarios (i.e., they were anticipating the potential contingency before they were explicitly aware of the result). Participants with vmPFC lesions, on the other hand, although generating SCRs to the immediate rewards or punishment, did not generate anticipatory SCRs while choosing a deck. Furthermore, when examining the SCRs of participants with amygdala lesions, it was found that not only did they fail to generate anticipatory SCRs, but they also failed to generate them in response to the rewards or losses (Bechara, Damasio, Damasio, & Lee, 1999). Along with providing evidence for the somatic marker hypothesis, this study gave some insight into the different functions of the vmPFC and amygdala. Both groups were impaired in their ability to make decisions, but the amygdala seems to be crucial in attaching an affective or emotional label to the *stimuli*, while the vmPFC seems to be involved in integrating or attaching the emotional marker to the *associated event*.

The above theories centre around deficits in arousal in explaining antisocial behaviours, next theories will be introduced that suggest that impairments in the processing of the emotions of others lead to antisocial behaviour.

1.1.8. The Processing of the Emotions of Others

1.1.8.1. The violence inhibition mechanism.

The Violence Inhibition Mechanism (VIM), proposed by Blair (1995), is a cognitive mechanism, which is activated by non-verbal communications of distress in others and results in increased autonomic activity, increased attention, and activation of the threat response system (Blair, 1995). The VIM was developed from the idea that various other social animals have been shown to inhibit an aggressive attack when a conspecific displays cues for submission (Eibl-Eibesfeldt, 1970; Lorenz, 1966). For example, it is known that when a dog rolls on its back and exposes its throat, another

dog with which it is engaged in a fight will desist from an attack. In humans, the distress cues of others (e.g. a fearful face) are assumed to activate a predisposition to withdraw from the situation in the observer who processes these cues. For example, an individual engaged in a violent attack may stop when they witness the fearful face of another. The strength of the withdrawal response is variable and a function of the degree of activation of the VIM. For example, an isolated sad facial expression may stimulate limited withdrawal, while a screaming, wide eyed, and erratic individual may excite a stronger withdrawal response (Blair, 1995). Importantly, the VIM is activated automatically and not dependent upon the context in which a distress faces is presented (Blair, 1999). Nevertheless, there exist other cognitive mechanisms which may override the VIM. For example, despite the VIM being activated in an individual, there may still exist motivational reasons for continuing with an attack, which override the VIM and therefore do not result in a withdrawal response.

As well as a distress cue activating the VIM in isolated incidents, distress cues can also form associations with the behaviours that caused them, via classical conditioning. The processing of the distress cue and subsequent activation of the VIM can be considered the unconditional response, which will then form associations with the behaviours which caused their activation. As a result, the mere consideration of a behaviour (e.g. aggression) that would result in a distress cue and therefore activation of the VIM, will stimulate a withdrawal response (Blair, 1995). It is thought that such a process is involved in the development of moral socialisation and empathy (Blair, 1995). Evidence in support of the existence of the VIM comes from studies that show that distress cues result in the termination of both violent altercations (e.g. Perry & Perry, 1974) and non-violent disputes over property ownership (Camras, 1977).

The first evidence that such a mechanism may be dysfunctional in individuals who display antisocial behaviour came from research on psychopaths. In two studies, participants observed confederates receiving, what they were led to believe as real electric shocks, while their skin conductance response was recorded. It was found that psychopaths were less responsive to the displays of distress by the shocked individuals than controls (Aniskiewicz, 1979; House & Milligan, 1976; however, see Sutker, 1970). In addition, Blair, Jones, Clark, & Smith (1997) examined skin conductance responses of psychopaths and their matched controls in response to neutral stimuli, threatening stimuli and distress cues. While psychopaths showed comparable autonomic responses to neutral and threatening stimuli, they showed reduced responses to the stimuli depicting a distress cue. Importantly, there is also evidence of such a deficit in children with CD. Eisenberg et al., (1996) found that children with greater behavioural problems were less likely to avert their gaze from a film depicting the distress of others, while Blair (1999), also found that children high in psychopathic traits showed reduced electrodermal responses to distress cues and threatening stimuli than control children. Furthermore, a study by van Goozen, Snoek, Matthys, van Rossum, & van Engeland (2004) found a negative relationship between CD severity and startle response to unpleasant pictures including images of distress, such that the more severe the conduct problems the smaller the startle response children had to such images.

1.1.8.2. The integrated emotions systems (IES) model.

The Integrated Emotions Systems (IES) model (Blair, 2005) is an attempt to merge both the low-fear and VIM theories into one etiological account of antisocial behaviour. According to this theory, genetic susceptibility to amygdala dysfunction is central to both the low fearfulness and reduced responsiveness to the distress cues

of others found in those with antisocial behaviour problems (Blair, Mitchell, & Blair, 2005; Blair, Peschardt, Budhani, Mitchell, & Pine, 2006; Blair, 2006).

The amygdala is central to limbic system functioning and plays a key role in the processing of social and emotional information (Adolphs et al., 1999). It is functionally connected to other brain areas, such as the frontal cortices, the basal ganglia, the hippocampus and hypothalamus, and together these areas are involved in the recognition and regulation of emotions (e.g. Adolphs, Tranel, Damasio, & Damasio, 1994; Baird et al., 1999; Bechara et al., 1995), as well as memory for emotional information. The amygdala has also more recently been considered as important in enhancing attention to emotionally salient cues in the environment (e.g. Adolphs, 2010). Importantly, it is also involved in conditioning processes that require emotions (e.g. learning from punishment; (Davidson, Fox, & Kalin, 2007; DeLisi, Umphress, & Vaughn, 2009; LeDoux, 2000). As was described in relation to the VIM above, it is a breakdown of this conditioning process, as a result of amygdala dysfunction, that is thought to lead to the development of antisocial behaviour (Blair, 2005). In regards to the distress cues of others, emotional expressions of fear, sadness and happiness are thought to initiate stimulus-reinforcement learning, which allows the individual to learn the value of the object or actions to which they are displayed. According to the IES, antisocial behaviour (such as aggression) comes to be regarded as 'bad' because of the association of representations of these behaviours with the aversive feedback in the form of the distress shown by the victims of these behaviours (Blair, 2013). Amygdala dysfunction, and consequent impaired stimulus-reinforcement learning and responsiveness to the distress of others, results in a deficient response following antisocial behaviour. And as argued by the VIM above, over time, these deficits lead

to impaired socialisation and impaired moral conscience development (Blair, 1995).

Evidence for this model in explaining antisocial behaviour comes from studies revealing psychopaths have reduced amygdala volume relative to comparison individuals (Tiihonen et al., 2000) and reduced activation of the amygdala during emotional memory tasks (Kiehl et al., 2001). In addition, a number of fMRI studies have found amygdala responses to sad and fearful expressions but not angry or disgusted expressions (Blair, Morris, Frith, Perrett, & Dolan, 1999; Morris et al., 1996; Phillips, Young, Senior, & Brammer, 1997), consistent with the suggestion that the amygdala is sensitive to signals of distress. Evidence has also shown that the amygdala is involved in aversive conditioning and the potentiation of the startle reflex by visual primes (Angrilli et al., 1996; LaBar, LeDoux, Spencer, & Phelps, 1995). Furthermore, as has been demonstrated with patients with amygdala lesions, psychopaths too have impairments in aversive conditioning and potentiation of the startle reflex (Lykken, 1957; Patrick, 1994). As will be more fully reviewed below, children and adolescents with CD have also been shown to have deficits in the recognition of distress cues (Fairchild, Stobbe, van Goozen, Calder, & Goodyer, 2010; Fairchild, van Goozen, Calder, Stollery, & Goodyer, 2009), aversive (fear) conditioning (Fairchild et al., 2010), and have reduced gray matter volumes in the amygdala (Fairchild et al., 2011).

Taken together, the above theories suggest that individuals with high levels of antisocial behaviour have both reduced physiological arousal and deficits in their ability to process emotional stimuli. However, given the high overlap, and the fact that ADHD often precedes CD, it is unclear if such mechanisms are present in ADHD, and therefore present an increased risk factor for those with ADHD to go on to develop CD, or whether the deficits are specific to CD, and not associated with

ADHD. As emphasised earlier, it is important to understand the specificity of such deficits to enable the appropriate application of interventions.

1.1.9. Are Emotional Processing Impairments Specific to CD or Additionally Present in ADHD?

In comparison to EF, few studies have explored emotional functioning in ADHD. Some research evidence has pointed to emotional abnormalities in those with ADHD, such as reduced emotion recognition, inhibition, and empathy (Cadesky, Mota, & Schachar, 2000; Kats-Gold, Besser, & Priel, 2007; Maedgen & Carlson, 2000; Sinzig, Morsch, & Lehmkuhl, 2008); however, there is often a lack of control for comorbid CD. Barkley (1997) suggests that emotion dysregulation is a key component in the aetiology of ADHD because it results from another of its symptoms, lack of inhibition. A lack of an ability to effectively inhibit responses also causes problems in inhibiting emotional responses. On the other hand, other studies have found that emotional processes and inhibitory control are independent of one another (Blaskey, Harris, & Nigg, 2008) and instead it may be that those with ADHD only present with emotional impairments in the presence of comorbid disorders, such as DBDs and internalising mood disorders (Factor, Reyes, & Rosen, 2014).

The most convincing evidence that emotional impairments do not characterise those with ADHD comes from studies comparing hot and cold EF. Research has suggested that EF can be distinguished into hot affective processes and cold cognitive aspects (e.g. Rubia, 2011). As described above, ADHD has consistently been associated with cold cognitive impairments, such as difficulties with cognitive and motor response inhibition, sustained attention, and timing functions (Rubia et al., 2001, 2007; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). Children with CD alone too, have been found to have impairments on tasks of

cognitive and motor inhibition (Herba, Tranah, & Rubia, 2006), sustained attention (Dougherty, Bjork, Marsh, & Moeller, 2000), and set shifting (Lueger & Gill, 1990). However, these studies failed to control for comorbid ADHD. Further, the studies in which ADHD has been controlled for, have found no cold cognitive impairments in those with CD (Avila, Cuenca, Félix, Parcet, & Miranda, 2004; Klorman et al., 1999; Scheres, Oosterlaan, & Sergeant, 2001; van Goozen, Cohen-Kettenis, Snoek, Matthys, Swaab-Barneveld, van Engeland, 2004).

The pattern of results for studies that have examined hot executive functioning, on the other hand, are much more consistent. On such tasks, children with CD, either alone, or with comorbid ADHD are more impaired than children with ADHD alone (Rubia, 2011). Children with CD are found to be consistently impaired on response preservation tasks. In these tasks a certain way of responding (a response-set) is initially rewarded, but over time it is then punished. Adaptive responding on this task involves changing ones way of responding in order to be flexible to changes to the rewards and punishments. However, in those with CD, they show a tendency to continue to respond using the response set that was initially rewarded (Matthys, van Goozen, Snoek, & Van Engeland, 2004). In addition, adolescents with CD also show impairments in their performance on delayed gratification tasks, in which they have to choose between smaller immediate rewards and delayed larger ones (Dolan & Lennox, 2013; van Goozen, Cohen-Kettenis, et al., 2004). Together, these behavioural studies suggest that ADHD and CD have separable deficits. CD is associated with deficits in emotional EF, while ADHD is characterised by cognitive EF impairments. These distinct deficits may also explain why children who have both ADHD and comorbid CD have more problems and a worse prognosis than either of the disorders alone because they will have both

cognitive and emotional EF deficits.

The separable deficits reported above are also supported by studies utilising structural and functional brain imaging methods. Rubia (2011) reviewed the evidence of brain dysfunction in those with ADHD and CD and found that ADHD was characterised predominantly by abnormalities in the inferior frontal, striatal, parietotemporal, and cerebellar regions which mediate cold cognitive EF such as inhibition, attention and timing functions, while CD was consistently associated with abnormalities in the hot paralimbic system that regulates affect and motivation, and consists of lateral orbital and ventromedial PFC, superior temporal lobes, and underlying limbic structures, most prominently, the amygdala.

The above tasks have so far separated ADHD and CD related to executive functioning tasks that involve sensitivity to rewards and punishments. However, as is clear from the models/theories above, a central aspect explaining antisocial behaviour is a deficit in the processing of distress cues. Currently there is a scarcity of research examining the specificity of processing of distress cues in those with CD and ADHD and it is still unclear in certain areas if ADHD shares the same deficits as CD and therefore whether ADHD itself is a risk factor for later antisocial behaviour. In addition, there is still a lack of consensus as to the mechanism underlying the link between amygdala dysfunction and impairments in the processing of distress cues. Gaps in the literature in relation to four areas of emotional processing in regards to its specificity to CD or ADHD in general will be briefly reviewed below. However, the introduction to each experimental chapter will discuss in more detail the preceding research that has motivated the research questions.

1.1.10. Emotion Recognition, Empathy and Attention

In line with the IES and VIM models, emotion recognition impairments have been reported in a variety of antisocial populations (Blair et al., 2004; Blair, Colledge, Murray, & Mitchell, 2001; Bowen, Morgan, Moore, & van Goozen, 2014; Dadds et al., 2006; Glass & Newman, 2006), including those with CD (Fairchild et al., 2009). From the above studies, and also in line with the IES, it would appear that the above deficits in recognition are found most often in fearful and sad expressions. Further, in a meta-analysis of 20 studies, Marsh & Blair (2008) found a robust link between antisocial behaviour and a specific deficit in the recognition of fearful faces. Further evidence that it is specifically fear that individuals with CD have problems with comes from findings that those with amygdala lesions have specific impairments in recognising fearful expressions (e.g. Adolphs et al., 2005), and such impairments have also been reported in those with CD (Fairchild et al., 2011).

However, a more recent meta-analysis of emotion recognition in psychopaths across both facial and vocal modalities, found they had problems with a range of emotions (not only fear and sadness; Dawel, O’Kearney, McKone, & Palermo, 2012). Such findings are at odds with theories that suggest the amygdala specifically detects distress related stimuli. However, more recent theories have suggested that the amygdala is important for the detection of salient and socially relevant information (Adolphs, 2010). For example, damage to the amygdala has been associated with abnormal processing of the eye-region of faces (Adolphs et al., 2005; Spezio et al., 2007). Together these findings suggest that there may be a more general dysfunction in the attentional mechanisms of those displaying antisocial behaviour (Dadds et al., 2006) and since the eye region is particularly important for the recognition of fear (Bassili, 1979), this may explain why fear recognition is the most common deficit observed (Adolphs et al., 2005). Indeed, a recent study in

young offenders, motivated by the reasoning that emotion recognition impairments may be due to a lack of attention to the eye region of the face, trained individuals to look towards the eye region when recognising emotions (Hubble et al., 2015). Following this training, not only was there an increase in emotion recognition performance, but also a reduction in the severity of crimes committed in the training group compared to the control group (another group of young offenders who did not receive the training). However, a confound of this study was that along with redirecting participants attention, explanations were also given which helped participants interpret the facial configuration of the face; therefore it is possible that not attentional deficits but misinterpretation of the features of emotional faces explain poor recognition performance. Indeed, there is a literature, which explains aggressive behaviour as resulting from hostile attributional biases (Dodge & Newman, 1981; Dodge, Price, Bachorowski, & Newman, 1990), whereby aggressive individuals misinterpret neutral expressions as hostile or threatening (e.g. anger). As it is theorised that only distress related cues lead to amygdala activation and therefore withdrawal from aggressive responses, it is possible that those who commit antisocial acts interpret such distress cues (e.g. fear) as anger and therefore this does not result in amygdala activation and subsequent withdrawal.

A further related problem in the Hubble et al. intervention study (2015) is that they did not assess the prevalence of ADHD in the young offender sample. The prevalence of ADHD in young offenders has been found to be high (Young et al., 2010), while those with ADHD have also been found to have deficits in emotion recognition (Pelc, Kornreich, Foisy, & Dan, 2006; Singh et al., 1998). However, due to the prevalence of comorbid CD it is not clear if these deficits are a reflection of ADHD or CD. Indeed, one study found that emotion recognition deficits in ADHD

were explained by co-occurring CD symptoms (Schwenck et al., 2013). Not only is it still unclear whether adolescents with ADHD have deficits in emotion recognition, it may also be that if they do have deficits, these are due to different underlying mechanisms than those of CD. It is possible that in the described intervention study (Hubble et al., 2015) addressing the attentional problem was only effective because it addressed a core deficit of those with ADHD (namely, attention) which may have been present in the sample.

Another area of emotion processing whereby impairments have been associated with both ADHD and CD in separate studies is empathy. There have been many attempts to define empathy over the years (see Preston & de Waal, 2002), but broadly it can be understood as the understanding and sharing of another's emotional state (Eisenberg & Strayer, 1990). Empathy is not a unitary construct and evidence exists for sub-components of empathy, namely, affective (or emotional), cognitive and motor empathy (Blair, 2005). Affective empathy involves vicariously experiencing the emotion that is more consistent with another person (Bons et al., 2013). Cognitive empathy is the ability to understand what another person is thinking or feeling (Blair, 2005), while motor empathy is the ability to unconsciously and automatically mirror the facial expression of another person (also known as facial mimicry; Blair, 2005). Empathy is crucial for appropriate social behaviour (Decety, Bartal, Uzefovsky, & Knafo-Noam, 2016) and has consistently been argued to be important for the inhibition of antisocial behaviour (Hoffman, 1987; Miller & Eisenberg, 1988). Although studies have found that those with CD have a deficit in empathic abilities (Cohen & Strayer, 1996), studies vary as to whether such deficits are general to both cognitive and affective empathy (Anastassiou-Hadjicharalambous & Warden, 2008; D. Cohen & Strayer, 1996), or whether the deficits are specific to

affective empathy (van Goozen et al., 2016; van Zonneveld, Platje, De Sonnevile, van Goozen, & Swaab, 2017). It is also still unclear if empathy deficits as observed in those with CD are present for all emotions, or specific to emotions representing distress cues (particularly fear). The IES would predict that empathy deficits would only be present for fear and sadness (Blair, 2005).

As above in relation to emotion recognition, the issue is complicated further by evidence that those with ADHD have also been found to have less empathy when compared to typically developing controls (Braaten & Rosen, 2000; Dyck, Ferguson, & Shochet, 2001), but again it is unclear if such differences were driven by ADHD symptomology or instead were a result of comorbid CD (e.g. Marton, Wiener, Rogers, Moore, & Tannock, 2009). Very recent research has begun to try to isolate empathy deficits in these two groups of children by comparing affective and cognitive empathy in those with ADHD alone and those with ADHD and comorbid CD (van Goozen et al., 2016). Interestingly, participants did not differ in cognitive empathy, however, in relation to affective empathy, participants with comorbid CD were found to have significantly reduced affective empathy. Further, despite what would be predicted by the IES, affective deficits were not only restricted to distress emotions (fear and sad) but also found for happiness. To date, however, this is the only study that has explored separable empathy deficits in those with ADHD and CD, and further research in this area is therefore warranted.

Again, as with emotion recognition, the mechanisms driving the empathy deficits in those with CD are far from clear. A lack of attention to the eye region of the face has been suggested as a potential mechanism (Bons et al., 2013). However, to date only one recent study has examined attention in children at risk of future antisocial behaviour (due to having a sibling displaying such behaviours), and found

that despite showing deficits in affective empathy, there was no difference in attention allocation between those at risk of antisocial behaviour and typical controls when processing the empathy inducing stimuli (van Zonneveld et al., 2017).

However, this study only explored children at risk of future antisocial behaviour, none had yet displayed such behaviours themselves, nor did any have a diagnosis of CD or ADHD; therefore it remains unclear whether attentional deficits underlie empathy impairments in these clinical groups.

1.1.11. Emotion Regulation

Emotion regulation refers to the processes in which “individuals influence which emotions they have, when they have them, and how they experience and express them” (Gross, 1998, p. 271). Such processes may either be automatic or controlled, and conscious or unconscious. Gross (1998) postulated that emotion regulation can be regulated at five stages along an emotion generative process: (1) the selection of the situation, (2) the modification of the situation, (3), the deployment of attention, (4) change of cognitions, and (5) the modulation of responses.

Healthy functioning relies upon effective emotion regulation. When emotion regulation is impaired, emotional dysregulation is the result and this can be characterised by rapid and poorly controlled shifts in emotion, emotional expressions or experiences which are out of proportion for the context, or are against social norms, and the irregular allocation of attention to emotional stimuli (Shaw, Stringaris, Nigg, & Leibenluft, 2014). Emotional dysregulation is thought to be central to initiating and maintaining the symptoms of psychopathology (Cole, Michel, & Teti, 1994). Indeed, emotion regulation problems are implicated in over half of DSM-IV Axis I, and all of Axis II disorders (Gross & Levenson, 1997).

As with the above aforementioned areas of emotion processing, deficits in

emotion regulation have been found in both ADHD and CD in separate studies. Indeed, emotion dysregulation is thought to be particularly important in ADHD. A recent meta-analysis (Shaw et al, 2014) found prevalence estimates of emotion dysregulation of between 24% and 50% in clinic based studies. It is thought that adolescents with ADHD show problems in emotion regulation due to their reduced capacity for inhibition resulting in difficulties withholding a response for long enough to gather the information necessary to understand emotionally charged situations (Barkley, 1997). In addition, emotion dysregulation has also been used to explain the antisocial behaviour displayed in those with CD (Cole, Michel, & Teti, 1994). Neurobiological circuits (including the orbitofrontal cortex (OFC), ventromedial prefrontal cortex (vmPFC), dorsolateral prefrontal cortex (DLPFC), amygdala, and anterior cingulate cortex (ACC)) are thought to be dysfunctional in those with CD, and it is these areas that are involved in both top-down regulation of (negative) emotions and the bottom-up processing of environmental cues that ordinarily produce emotional restraint (Davidson, Putnam, & Larson, 2000). In support of emotion regulation being deficient in CD, numerous studies have found impairments in emotion regulation in adolescents with CD compared to controls (Calkins & Dedmon, 2000; Deborde, Maury, & Aitel, 2015; Kostiuk & Fouts, 2002; McLaughlin, Hatzenbuehler, Mennin, & Nolen-Hoeksema, 2011). Therefore further research is required that considers both ADHD and CD together to explore the extent to which emotion regulation difficulties are driven by individual or both disorders.

A problem with research into emotion regulation is that it to a large extent (although see Northover, Thapar, Langley, & van Goozen, 2015a) relies on self- or informant report (Anastopoulos et al., 2012; Sobanski et al., 2010; Strine et al., 2006). Such methodology has long been suggested to lack reliability and validity

(Nisbett & Wilson, 1977), especially in judgements that are made retrospectively (Robinson & Clore, 2002). As mentioned above, some studies have begun to explore other measures of emotion regulation, specifically by making use of the ultimatum game (UG; Northover et al., 2015a; Schoorl, Van Rijn, De Wied, Van Gooen, & Swaab, 2016). In this task participants have to choose to accept or reject offers made to them. Some offers are clearly fair (e.g. an equal share of a reward), while others are clearly unfair (a very biased share of the reward in favour of the proposer of the offer), and still others are ambiguous (offers 70/30 and 60/40 splits). It is argued that it is rational to accept all offers made because accepting offers results in both the participant and the responder receiving the reward proposed in the offer, while rejecting offers result in the reward being withheld from both parties. As a result, rejected offers, particularly the rejection of ambiguous offers, is taken as evidence of a failure to regulate emotions. When utilising this paradigm, Northover et al., (2015a) found that participants with ADHD and comorbid CD rejected a significantly higher amount of ambiguous offers than ADHD alone. However, there are problems with this paradigm. Emotion regulation as determined by the UG is based on the assumption that the acceptance of unfair offers is a rational decision because from an economic perspective, the rejection of offers is irrational because it results in personal loss. However, from a social perspective, rejection of unfair offers can be seen as a rational, altruistic action to preserve social norms. Rather than maximising self-interest, the participant chooses to punish the socially inappropriate action, therefore a decision to reject an offer is not necessarily a result of a failure in regulation (Fehr & Fischbacher, 2003; Knoch, Pascual-Leone, Meyer, Treyer, & Fehr, 2006). Indeed, similar rejection rates are found in a modified version of the UG in which participants play on behalf of a third party, compared to one played by

themselves (Civai, Corradi-Dell'Acqua, Gamer, & Rumiati, 2010). In addition, it is assumed that the production of unfair offers results in an emotional response (anger in particular), but as physiological responses are usually not measured, it remains unclear whether an emotional response was evoked. Therefore, it is clear from the above, that the emotion regulation literature, especially in relation to ADHD and CD, would benefit from an objective measure of emotion regulation that clearly involves some aspect of emotional processing.

1.1.12. Fear Conditioning

As has been highlighted in a number of the theories of antisocial behaviour presented earlier in the chapter, emotion learning plays an important role in explanations of antisocial behaviour (Blair et al., 2005).

Fear conditioning is a basic form of learning in which a fearful response comes to be associated with a previously neutral stimuli. Experimentally, this is typically done by pairing a neutral stimuli, such a colour presented on a screen, with an aversive loud noise. With their continued combined pairing, the previously neutral stimulus will also come to evoke a fear response that can be recorded via physiological measurements such as skin conductance responses (Sterzer, 2010). A number of studies have shown that fear conditioning is impaired in both males (Fairchild, van Goozen, Stollery, & Goodyer, 2008) and females (Fairchild, Stobbe, van Goozen, Calder, & Goodyer, 2010) with CD. Further, there is strong longitudinal evidence that poor fear conditioning at a young age predicts later antisocial behaviour. Gao, Raine, Venables, Dawson, & Mednick (2010) and Gao, Raine, Venables, Dawson, & Mednick (2010) found that poor fear conditioning at age 3 predicted aggressive behaviour at age 8 and criminal behaviour by the age of 23, respectively.

While the previous studies presented above did not consider co-morbid ADHD, a recent study compared fear conditioning ability in those with ADHD and comorbid CD compared to ADHD alone and found that conditioning deficits were specific to those with additional CD (van Goozen et al., 2016). However, this was the first study to find such an effect and further research on the topic is therefore warranted.

In addition to the above, another neglected area in the fear conditioning literature on antisocial behaviour relates to extinction learning. This process refers to how quickly an individual comes to 'lose' the association between a neutral stimuli and an aversive one. When the neutral stimuli is no longer accompanied by an aversive noise, the strength of the connection between the stimuli will dissipate, and therefore the neutral stimuli being presented alone will no longer result in a fear response. This process has received a large amount of attention in relation to anxiety disorders such as Post Traumatic Stress Disorder (PTSD) and Obsessive-Compulsive Disorder (OCD; (e.g. Milad et al., 2008, 2013). Typically, in these studies, those with high levels of anxiety (who show an exaggerated fear response) are found to have deficits in extinction learning such that the continuous presentation of the neutral stimuli without the pairing with the aversive stimulus continues to trigger a fear response. Given that those showing antisocial behaviour (including those with CD) are characterised by low levels of anxiety, it may be case that along with a reduced ability to acquire a fear response, they may also lose the association between a neutral stimuli and aversive stimuli at a faster rate. The studies that examine fear conditioning in those with CD typically only compared mean skin conductance responses over the entire extinction period (Fairchild et al., 2010; Syngelaki, Fairchild, Moore, Savage, & van Goozen, 2013; van Goozen et al., 2016) and found

no differences between those who display antisocial behaviour and those who do not. However, no studies on CD have examined the rate in which the association between a neutral stimuli and aversive stimuli dissipates. Support for the idea that there may be a difference in extinction rate in those with CD comes from Syngelaki et al., (2013), who found that deficits in fear conditioning in young offenders were more pronounced in the later phases of the conditioning paradigm. The authors speculated that this effect may have been due to young offenders extinguishing the conditioned response more rapidly than controls. Further, as well as the acquisition of fear responses, the amygdala is also thought to be involved in the extinction process (Davis, 1992). Given that those with CD are supposed to have a dysfunctional amygdala, this strengthens the idea that there may be abnormalities during the extinction phase in those with CD.

A final area of uncertainty in the fear conditioning literature, and a source of heterogeneity in those with CD is the role of Callous and Unemotional (CU) traits and their relevance will be reviewed below.

1.1.13. Factors of Degree, and Not of Kind

The majority of previous research has adopted a categorical approach to identifying risk factors for certain disorders by comparing clinical groups (Decety, Michalska, Akitsuki, & Lahey, 2009; Herpertz et al., 2008; Luman, Tripp, & Scheres, 2010). Up until very recently most research used this framework when conceptualising psychopathology. However, there are problems with such an approach (Walton, Ormel, & Krueger, 2011). Symptoms of disorders at a non-clinical level, comorbidities (as have been previously described) and heterogeneity within disorders are all common. Such is the extent of such variability that it is theoretically possible that two individuals with the same diagnosis may share no more than one

common feature, but that two individuals with distinct diagnoses may share many symptoms (Vanheule, 2014). As a result, research has increasingly explored impairments on a continuous scale, using correlation and regression analyses (e.g. Fairchild et al., 2009; Marsee, Silverthorn, & Frick, 2005; Passamonti et al., 2010). Two particular sources of variability in those with CD are relevant to the current thesis (although see studies that highlight the importance of age of onset; Moffitt, 1993). The first is CU traits and the second is symptom severity of CD and ADHD.

1.1.13.1. Callous and unemotional traits.

As described earlier in this chapter a recent addition to the DSM-V (APA, 2013) in the diagnosis of CD is the presence or not of Callous and Unemotional (CU) traits. These traits describe an individual who displays limited empathy, shallow affect and a general disregard for others (Frick & White, 2008). This closely corresponds to a core construct of psychopathy, the interpersonal-affective dimension (Hare & Neumann, 2008). Research has suggested that these traits add further prognostic value that is not explained by CD symptomology alone (Frick, Marsee, & Patrick, 2006). It appears that antisocial behaviour accompanied by CU traits is more strongly explained by genetic factors (Fontaine, Rijdsdijk, McCrory, & Viding, 2010), while those with CD and low CU traits are better explained by environmental factors (Viding, Jones, Paul, Moffitt, & Plomin, 2008). In addition, those with CD who additionally display CU traits exhibit a more chronic, severe and aggressive pattern of conduct problems and delinquency than those without such traits (Frick, Stickle, Dandreaux, Farrell, & Kimonis, 2005; Scheepers, Buitelaar, & Matthys, 2011). The strength of evidence for the role of CU traits in different domains of emotion processing varies and can also depend on the sample used. The majority of evidence for CU traits playing a role in emotion processing impairments has been found in

community samples who do not show clinical levels of conduct problems (Bedford, Pickles, Sharp, Wright, & Hill, 2015; Dadds et al., 2006; Sharp, Vanwoerden, Van Baardewijk, Tackett, & Stegge, 2015) while there is some evidence in clinical samples (Pasalich, Dadds, & Hawes, 2014; Sebastian et al., 2014). However, when exploring the role of CU traits in those with clinical levels of CD evidence for CU traits is less convincing. For example Martin-Key, Brown, and Fairchild (2016) found no difference between those with CD who had been divided into high and low CU traits in empathic accuracy, while other studies, when considering both CU traits and severity of CD together find CU traits do not add anything over that explained by CD severity (e.g. Northover et al., 2015a; Northover, Thapar, Langley, Fairchild, & van Goozen, 2016; Sully, Sonuga-Barke, & Fairchild, 2015). However, as alluded to earlier, a domain in which there is uncertainty and relevant to the current thesis is fear conditioning. The original finding of deficits in fear conditioning in those displaying antisocial behaviour was in those with psychopathy (Lykken, 1957), and subsequent findings in the adult psychopathy literature have since found that such deficits seem to be most strongly associated with the interpersonal-affective dimension (also known as primary psychopathy; e.g. Birbaumer et al., 2005; Veit et al., 2013)). Therefore, one would expect to find that deficits in fear conditioning observed in adolescents would be related to CU traits. However, studies in CD have failed to find such a relationship (Fairchild et al., 2008; van Goozen et al., 2016) , or have not explored their influence (Gao et al., 2010). Therefore, given the negative results to date, the role of CU traits in relation to fear conditioning, and the role of another aspect of psychopathy, Impulsive-Irresponsibility, warrant further investigation.

In addition to fear conditioning, CU traits have also been implicated in

impairments in emotion recognition and empathy. Work that suggested that a lack of attention to the eye-region of the face explained emotion recognition deficits was conducted on individuals high in CU traits (Dadds et al., 2008); therefore it may be that these traits specifically account for such attentional deficits. However, that study was conducted in undergraduate students who are unlikely to show the clinically severe sorts of behaviours observed in those with CD, and it is therefore important to test this further. The same can be said for the role of attention to the eye region in empathy. CU traits have been found to play a moderating role in empathy deficits observed in those with CD. Anastassiou-Hadjicharalambous & Warden (2008) found cognitive and affective deficits in those with CD and low CU traits, while only affective deficits were found in those with CD and high CU traits. However, other studies have found that those high in CU traits have problems in both cognitive and affective empathy over and above problems associated with CD (Pasalich et al., 2014).

From what was described above, it is clear that the role of CU traits in emotional processing and in abnormal attention allocation requires further exploration.

1.1.13.2. Symptom severity.

As opposed to an age of onset dichotomy (Moffitt, 1993) or the presence of CU traits, other researchers have found that it is the severity of symptoms (that transcend categorical boundaries) that are important in explaining emotional deficits and vice versa. For example, when examining brain structure abnormalities Fairchild et al., (2011) found that CD symptom severity (rather than CU traits) was positively correlated with reduced structural integrity of important emotional processing regions, while classifying subjects into child or adolescent onset CD was not

important in explaining such abnormalities. In addition, Northover et al., (2016) found that severity of CD symptoms predicted both lower levels of cortisol at baseline and reduced cortisol stress reactivity, while van Goozen, Snoek, Matthys, van Rossum, & van Engeland (2004) found severity of conduct problems predicted reduced startle response and Syngelaki et al., (2013) found a similar relationship in a sample of young offenders. There is also further evidence that severity of conduct problems predict both fear conditioning and affective empathy impairments (van Goozen et al., 2016). The extent of structural abnormalities in those areas involved in executive functioning in those with ADHD has also been found to be positively correlated with ADHD symptom severity (Hwang et al., 2015). In addition, Northover, Thapar, Langley, & Goozen (2015b) found ADHD symptom severity was related to lower pain tolerance, while CD severity was related to higher pain tolerance. Therefore, given both the evidence presented above and the mixed evidence of CU traits it is clear further research is needed considering these dimensions and their contribution to emotional processing deficits.

1.1.14. Goals and Hypotheses of the Current Thesis

This chapter has discussed what CD and ADHD are, as well as exploring the overlap between the two diagnoses in key areas. It then went on to review key literature suggesting that impairments in emotion processing, particularly emotions such as fear that communicate distress, are important in explaining risk of antisocial behaviour and therefore potential mechanisms that may increase risk of antisocial behaviour in those with comorbid CD. However, it is clear that there are still areas of uncertainty in regards to the specificity of emotion processing impairments in CD and whether these are also observed in those with ADHD. In addition, it is still unclear why those with antisocial behaviour problems have impairments in emotion

processing. One recurring theme throughout the chapter related to attention (or lack of this) to key regions of the face. There are inconsistencies in the literature and given the overlap with ADHD it is unclear if these attention problems are a feature of CD, or associated with ADHD symptomology or the presence of CU traits.

As a result of current uncertainties in the literature, the overarching aim of the current thesis was to explore differences in emotion processing – particularly for fear related stimuli – in a sample of adolescents with ADHD with and without comorbid conduct disorder, while also exploring the role of attention and specific clinical characteristics (i.e., CD and ADHD severity, CU traits) in emotion processing ability.

The rationale for choosing adolescents as the study sample is important to mention given that CD and ADHD can be diagnosed in childhood, while as mentioned earlier, the vast majority of adults with a diagnosis of ASPD meet criteria for CD as adolescents. Adolescence represents a key stage of development both socially and in terms of brain development. The increase in social functioning and interactions with peers apparent during adolescence requires increased need to accurately process the emotions of others as well as a need to independently regulate their own affect at a distance from parents who would have previously played a role in helping regulation (Brown, 2004). This increased social pressure happens in the context of the relatively late development of the PFC and as a result can lead to difficulties with cognitive functioning and behavioural self-regulation (Yurgelun-Todd, 2007). Steinberg,(2005) suggests that antisocial behaviour (which peaks during adolescence; Monahan, Steinberg, Cauffman, & Mulvey, 2009) could be reflective of difficulties in the coordination of intellectual, emotional and behavioural capabilities.

Therefore if the specific mechanisms underpinning antisocial behaviour of those with CD can be identified during adolescence it is hoped that this will highlight which types of interventions would be most beneficial and how they can be appropriately tailored and targeted during this key stage of development to reduce antisocial behaviour.

This will be done by investigating the following areas of emotion processing: (1) emotion recognition, (2) empathy, (3) emotion regulation, and (4) fear conditioning. Each experimental chapter will start with a discussion of the literature that has explored these deficits in CD and ADHD samples, and where appropriate it will discuss the role of attention, severity of clinical symptoms and CU traits.

Chapter two will assess emotion recognition performance in a sample of ADHD participants with and without comorbid CD and a sample of age matched typically developing controls. The chapter had two primary aims: the first was to determine whether emotion recognition deficits to negative emotions (particularly fear) were specific to those with ADHD and comorbid CD or generally present in those with ADHD. As a secondary aim we explored whether emotion recognition deficits could be explained by a lack of attention to the eye region (as measured by eye-tracking) of the face or whether these were related to misinterpretation of the features of the faces. In addition, we explored the contribution of CD and ADHD severity and CU traits to emotion recognition and attentional impairments.

It was hypothesised that only participants with ADHD and comorbid CD would show deficits in the recognition of negative emotions in general, and in these would be most pronounced for fear, while in line with the hostile attribution bias, it was also predicted that these participants would be more inclined to attribute neutral faces as angry than both ADHD alone and control participants. It was further

predicted that these impairments would be most strongly negatively correlated with CD severity.

Second, it was predicted that both ADHD subgroups would demonstrate attention problems in comparison to controls, but that the ADHD and comorbid CD subgroup would have specific impairments in focusing on the eye region of the face of fear faces. While it was predicted that while ADHD severity would be related to eye-looking impairments in general, CD severity would be related to specific impairments in looking towards the eyes of fear faces.

Chapter 3 will explore cognitive and affective empathy in response to emotional film clips in those with ADHD alone and those with ADHD and comorbid CD. Eye-tracking was used to assess preference towards looking at the eyes of the main actor and whether this explained empathy performance. As with Chapter 2, the chapter will also explore the contribution of CD and ADHD severity and CU traits to such impairments.

It was predicted that participants with ADHD and comorbid CD would show reduced empathy for negative emotions, and that such reduced empathy would be limited to affective empathy. In addition, it was predicted that both CD severity and CU traits would be negatively related to empathy. Finally, it was predicted that adolescents with comorbid ADHD and CD would show less of a preference to look at the eye region during negative emotional clips and this would be related to amount of affective empathy.

Chapter 4 will explore emotion regulation and includes two experiments. Given the lack of an objective and experimental measure of emotion regulation available in the literature, in the first experiment in Chapter 4 we aimed to test a new attentional measure of emotion regulation, employing a pictorial emotional Stroop

(Cardiff Emotional Pictorial Stroop; CEPS) paradigm that utilised emotional faces and feature-matched objects as distractors, as well as positive and negative valenced symbols as targets. We tested this paradigm in undergraduate students. In the second experiment of this chapter we explored differences in emotion regulation in a small sample of adolescents with ADHD and comorbid CD and those with ADHD alone using this new paradigm.

For the first experiment it was predicted that undergraduate students' reaction time would not differ between target stimuli conditions *overall*, but they would respond faster to congruent (distractor-target) trials than to incongruent (distractor-target) trials, and faster to emotional face distractors than to object distractors. In relation to the second experiment, the thesis predicted a significant congruency effect in all ADHD participants such that they would respond faster to congruent trials than to incongruent trials, but we also predicted that this effect would be less strong in the comorbid ADHD and CD group based on the hypothesis that this subgroup has greater impairments in emotion processing.

Chapter 5 will assess differences in fear conditioning, and rate of extinction learning, in those with ADHD and CD and those with ADHD alone. Furthermore, a third aim of this chapter was to explore the contribution of CU traits, CD severity and ADHD severity to fear conditioning impairments and rate of extinction.

In line with previous research in a similar sample, we predicted that ADHD and comorbid CD participants would show less fear conditioning and an increased rate of extinction learning compared to those with ADHD alone. Secondly, we predicted that CD severity and CU traits explained variation in fear learning and extinction deficits, such that higher CD severity and more CU traits would be related to less differential fear conditioning and a faster rate of extinction.

Finally, Chapter 6 will summarise the findings from each of the experimental chapters, relating them back to the previous literature that motivated the current research questions while also contextualising the findings in relation to emotional and neurobiological theories of antisocial behaviour. Limitations of the current research will be discussed along with suggestions as to where further research is required. Finally, potential implications of the research findings will be considered in relation to issues of heterogeneity and implications for treatment and intervention.

The data collection for this thesis was carried out by a small research team (which included the author) but all analyses were undertaken solely by the author of this thesis, with advice provided by the supervisory team. The write-up of the thesis was solely the work of the author, with the supervisory team (co-authors) providing feedback and final approval of submitted journal papers.

1.1.15. The Current Sample

In Chapters 2, 3, 4 (experiment 2), and 5 the same sample of adolescents with ADHD or ADHD+CD were used which were recruited as part of a second recruitment phase of a wider study (COMT). This study explored the role of the Catechol-O-methyltransferase (COMT) Val158Met gene in aggression in those with ADHD (van Goozen et al., 2016). Participants from the COMT study were recruited from an earlier genetic study of ADHD (Study of ADHD Genes and Environment – SAGE). SAGE participants were recruited from Child and Adolescent Mental Health services in England and Wales. All the adolescents in the sample were of British Caucasian Origin and met criteria for a lifetime diagnosis of DSM-III-R or DSM-IV ADHD, which was confirmed using the Child and Adolescent Psychiatric Assessment (CAPA, Angold et al., 1995), a semi-structured research diagnostic interview administered during the SAGE study. Cognitive ability was assessed using

the Wechsler Intelligence Scale for Children IV (WISC-IV). Children with any known clinical or CAPA research diagnosis of Schizophrenia, bipolar disorder, Autistic Spectrum Disorder (ASD), Tourette's syndrome, or with an IQ < 70, epilepsy, brain damage or any other neurological or genetic disorder were excluded from the study. Ethical approval for SAGE and follow-up studies described here were obtained from the Wales Multicentre Research Ethics Committee.

For the current study participants were invited to Cardiff University with their parents/carers for a testing day which took between 4 and 5 hours. The day consisted of a number of computer tests and questionnaires which included the four paradigms described in the current thesis. Families were paid £25 in vouchers for taking part.

Due to data on all paradigms being collected contemporaneously (within the same testing day), rationale and hypotheses for each chapter are not influenced by the findings of the previous chapters.

The paradigm described in Chapter 2 was the first to be completed on the day, hence the sample size of the ADHD and ADHD+CD groups in this chapter is the largest. The paradigm used in Chapter 4 was added towards the end of the testing phase and therefore has a much reduced sample size compared to the other chapters.

Chapter 2 additionally includes a sample of typically developing adolescents recruited from local secondary schools. The schools sent out letters which included information about the study, as well as opt-in, consent and assent forms and parent Strengths and Difficulties Questionnaires (SDQs). The children whose forms were returned with the parents and children opting into the study were approached during school hours to complete the paradigm in a quiet classroom. Any participants who reporting having a diagnosis of either ADHD, CD or ASD were excluded. The ethics

for this part of the study was approved by Cardiff University School of Psychology Ethics Committee. Participants were paid a £5 voucher for taking part.

Chapter 4, experiment 1, used a sample of typically developing undergraduate students from Cardiff University. They completed the paradigm in a small experimental testing room at university. Any participants reporting that they had a diagnosis of ADHD, CD or ASD were excluded. They were paid a £5 voucher for taking part.

2. Chapter 2: Emotion Recognition

Paper under revision

This chapter is based on Airdrie, J. N., Langley, K., Thapar, A., van Goozen, S. H. M. (2017). Facial emotion recognition and eye gaze in attention-deficit/hyperactivity disorder and conduct disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*. *Under revision*

2.1. Introduction

Conduct disorder (CD) is costly to individuals and society (Scott et al., 2001). Thus developing and providing effective interventions are a priority. Parenting interventions represent one well-established treatment but there is growing appreciation that interventions which target underlying cognitive and biological mechanisms might also be important.

One mechanism proposed to be involved in the development of antisocial behaviour in CD is problems in recognising facial emotion expressions. Such deficits have been observed in several studies of antisocial adolescent and adult populations. (Blair et al., 2004; Bowen, Morgan, Moore, & van Goozen, 2014; Dadds et al., 2006; Fairchild, van Goozen, Calder, Stollery, & Goodyer, 2009; Stevens, Charman, & Blair, 2001). Although antisocial behaviour observed in those with CD has been linked with deficits in recognising a range of negative emotions (Blair & Coles, 2000; Dawel, O’Kearney, McKone, & Palermo, 2012; Woodworth & Waschbusch, 2008), the most consistent impairment found is in recognising fear (Blair & Coles, 2000; Dadds et al., 2006; Dadds, El Masry, Wimialaweera, & Guastella, 2008; Marsh & Blair, 2008). Distress related cues such as fear are argued to serve as inhibitors for aggressive acts (Blair, 2005); therefore an inability to recognise fear cues reduces the likelihood that aggressive acts are inhibited and also adversely affects development of empathy (Dadds, Cauchi, Wimalaweera, Hawes, & Brennan, 2012). One recent study applied an intervention in an attempt to improve emotion recognition in juvenile offenders (Hubble et al., 2015). Not only was there an improvement in emotion recognition, but there was also a significant reduction in severity of offences committed 6 months following the intervention. While these findings are promising, it is unclear if these deficits are specific to CD or generalize

to commonly comorbid disorders. Attention Deficit Hyperactivity Disorder (ADHD) is an especially common comorbidity of CD and is prevalent in offender populations (Young, Moss, Sedgwick, Fridman, & Hodgkins, 2015).

ADHD is comorbid with CD in about 30-50 % of cases (Biederman, 2005). To date there is a scarcity of research and a lack of consensus on emotion recognition impairments in ADHD. Although two studies observed impairments in ADHD (Pelc et al., 2006; Singh et al., 1998), other studies found no deficits when participants with comorbid CD were excluded (Schwenck et al., 2013). The first aim of the current study was to investigate whether in those with ADHD, emotion recognition deficits are specific to those with comorbid CD or are a feature of ADHD itself.

It is also uncertain why individuals with CD have emotion recognition impairments, and therefore what elements of the intervention are responsible for bringing about emotion recognition improvements and subsequent improvement in behaviour (e.g. reduction in crime (Hubble et al., 2015)). Dadds and colleagues (Dadds et al., 2006; Dadds et al., 2008) hypothesized that a lack of attention paid to the eye-region of the face, as evidenced by fewer fixations to the eye-region, leads to poorer recognition. This could be especially impaired in those with ADHD as inattention is a core symptom. Others proposed the existence of interpretational biases (Dodge et al., 1990). Aggressive individuals supposedly have a hostile attribution bias (Dodge & Newman, 1981; Dodge et al., 1990) whereby neutral expressions are misattributed as hostile or threatening, with further evidence of this found in violent adult offenders (Schönenberg & Jusyte, 2014).

Therefore, the second aim of this study was to examine both attention during emotion recognition performance and the specific errors made, to help disentangle

the role of these mechanisms in emotion recognition impairments.

Adolescents with a clinical diagnosis of ADHD with or without comorbid CD and typically developing controls matched on socioeconomic status completed an emotion recognition task whilst visual attention and scanning patterns were recorded via eye-tracking. We predicted that ADHD participants with CD would show impairments in negative emotion recognition, and would be more inclined to identify neutral faces as angry compared to those with ADHD without CD and typically developing controls.

Secondly, we predicted that both ADHD subgroups would demonstrate attentional problems compared to the healthy controls, but that the ADHD+CD subgroup would have a specific problem in focusing on the eye region of the face over and above ADHD alone.

2.2. Methods

2.2.1. Participants

Sixty three adolescents (16 females) between 11 and 18 years ($M = 14.2$, $SD = 2.09$) with a clinical diagnosis of ADHD were recruited from psychiatric and paediatric clinics in Wales (UK) as part of a larger genetics study (van Goozen et al., 2016).

Those with any known clinical diagnosis of schizophrenia, bipolar disorder, autism, Tourette's syndrome, or with an $IQ < 70$, epilepsy, brain damage or any genetic disorder were excluded. All participants had normal or corrected to normal vision.

No participants were stimulant naïve, but those currently being prescribed stimulant medication were asked to refrain from taking it at least 24 hours prior to testing.

Forty one (20 females) typically developing and healthy control participants (HC) between 11 and 18 years ($M = 15.5$, $SD = 2.7$) were recruited from local schools. HC

participants had normal or corrected to normal vision and excluded if they had received an ADHD, CD or ASD diagnosis.

Ethical approval was obtained from the Wales Multicentre Research Ethics Committee. Informed written consent was obtained from parents and adolescents aged over 16 years, while written assent was obtained from adolescents below 16 years of age.

2.2.2. Measures and Materials

2.2.2.1. Clinical Measures.

Child psychopathology in ADHD participants was assessed using the Development and Well Being Assessment (DAWBA) structured psychiatric research diagnostic interview using both parents and children as informants (Goodman, Force, Richards, Gatward, & Meltzer, 2000). ADHD and CD symptom scores and CD diagnosis were generated from the DAWBA interview. Total symptom scores and diagnoses were computed according to DSM-IV criteria (the DSM-V had not been published at the start of the study (American Psychiatric Association, 2000)) and further verified by a trained clinician. CD symptoms were considered present if endorsed by either parent or child. A research diagnosis of CD was given when more than or equal to 3 CD symptoms were present over the preceding 6 months and this interfered with functioning in at least one area of their lives. Internal validity of CD ($\alpha = .78$) and ADHD severity was high ($\alpha = .88$).

ADHD participants were allocated to one of two groups according to whether they met DSM-IV diagnostic criteria for Conduct Disorder or not: ADHD only ($n = 36$) and ADHD with comorbid CD (ADHD+CD; $n = 27$).

CU traits were measured using the Youth Psychopathic traits Inventory (YPI) (Andershed, Kerr, Stattin, & Levander, 2002). The CU subscale of this measure has

15 items, and each item is answered on a 4-point Likert scale (score range 0 – 45). The internal validity of the YPI overall ($\alpha = .94$) and the CU traits subscale ($\alpha = .80$) was high. In order to compare across ADHD and control participants, CD symptoms were also assessed via the parent and child Strengths and Difficulties Questionnaire (SDQ; $\alpha = .60$). The SDQ is a 25 item brief behavioural screening questionnaire. The CD subscale consists of 5 items which are scored on a 3 point Likert scale (range 0 – 15). Average CD symptoms score across parent and child ratings was calculated for each participant.

2.2.2.2. Socioeconomic status (SES)

SES was estimated using UK's Office of National Statistics estimates of average house-hold weekly income based on participants' postcodes (Low=0–£520; Middle=£521–£670; High=£671+).

2.2.2.3. Intelligence Quotient (IQ)

In the ADHD participants IQ was estimated using the Wechsler Abbreviated Scale of Intelligence (Wechsler, 2011) – 2 subset form (vocabulary and matrix reasoning).

2.2.2.4. Emotion Recognition.

Emotion recognition was examined using the Facial Emotion Recognition (FER) task (Bowen et al., 2014), consisting of 60 faces taken from the Ekman and Friesen facial affect battery (Ekman & Friesen, 1976), representing four basic emotions (Happiness, Anger, Fear, Sadness) and Neutral. An equal number of male and female targets appeared, and slides contained an equal number of each emotion presented at medium intensities (50 % and 75 %; see Figure 2.1 for example of stimuli). Each trial presented a target image along with numbered options of 1 to 5, representing, "Happy", "Sad", "Fear", "Anger", and "Neutral".

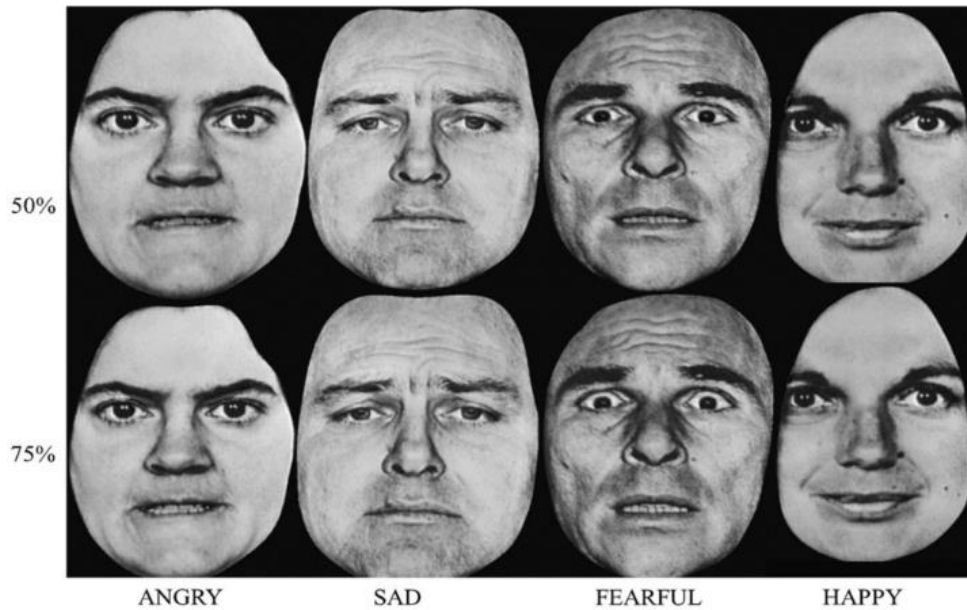


Figure 2.1. Examples of emotional face stimuli at 50 and 75% intensity

2.2.2.5. Eye-tracking.

Participants were positioned 60-65 cm from a laptop screen and a 9-point calibration was performed. The quality of calibration was checked and repeated as required. Calibration was followed immediately by facial stimuli. Eye-movements were recorded with a portable Tobii X2-60 compact eye-tracker sampling at 60 Hz with a screen resolution of 1920 x 1080. This equipment is robust to changes in head-position, negating the need for a chin rest. An I-VT fixation filter with a minimum fixation criterion of 60msec sampled average raw data of both eyes to produce information on eye position and duration. Eye-gaze validity was checked using a sample rate percentage that gives a rough estimate of quality of eye-tracking in a recording by providing a percentage score of successfully recorded data.

2.2.3. Procedure

ADHD and ADHD+CD participants were tested in a dimly lit laboratory room in a university clinic while HC participants were tested in a dimly lit room at school.

Following eye-tracker calibration, each face was presented in a set of three slides. First, a noise screen was used to prevent visual carryover effects from the previous trial; second, a fixation cross appeared in the top right hand corner of the screen in order to control starting eye position, and third, the face stimulus was presented. Noise and fixation screens were presented for one second each. The face stimulus had no time restraint, lasting for as long as it took to select an emotion.

2.2.4. Data Analyses

2.2.4.1. Emotion recognition accuracy.

Percentage correct recognition and percentage incorrect emotion selected for each emotion and each intensity level were calculated.

2.2.4.2. Eye-tracking.

Tobii Studio was used to analyse eye-gaze. Areas of interest (AOI's) were created around the eyes, mouth and face as a whole; one AOI was created around the emotion options and another around the entire screen (see Figure 2.2). In order to calculate percentage dwell time to the eyes for each participant, we first calculated dwell time to the entire face by summing all fixation durations to the face AOI area (area D, figure 2.2). We then calculated percentage dwell time to the eyes when looking at face by summing all fixation durations to the eye AOI divided by the total duration of time spent looking at the face AOI and multiplying by 100. We also analysed time to first fixation on the eye (TFF). Values more than 2 standard deviations above the mean and trials with values of zero were excluded for TFF analyses.

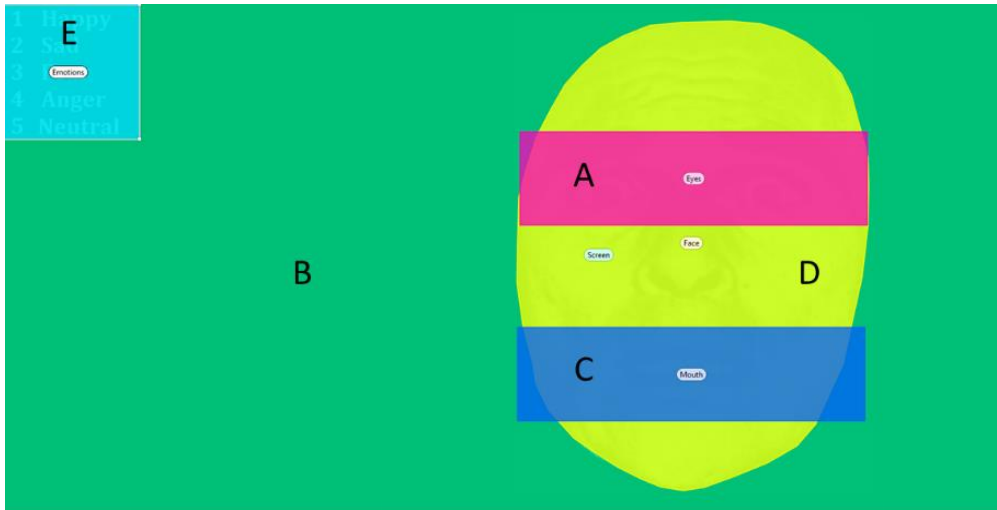


Figure 2.2. Defined areas of interest. A. Eye region. B. Non-affective region. C. Mouth Region. D. Rest of face region. E. Emotion options region.

Analyses were carried out using SPSS 20 (SPSS Inc., Chicago, Illinois). Differences in demographic and clinical characteristics between groups were analysed with one-way ANOVAs for continuous variables with student t-tests used to determine specific differences between groups and X^2 tests for binary variables. Pearson's (Spearman's rho where necessary) correlations were used to examine relationships between demographic and clinical characteristics with recognition and eye-gaze measures. Mixed model analysis of variance (ANOVA; ANCOVAs where demographics differed between groups and were independently related to outcome measures) with group (ADHD+CD, ADHD, HC) as a between subjects factor and emotion (Fear, Happy, Sad, Angry) and intensity (50% and 75%) as within subjects factors were used for the analysis of emotion recognition and eye-tracking measures. Where intensity was not found to interact with group, ANOVAs were rerun without intensity as a factor and the addition of Neutral to the emotion factor. In order to limit multiple comparisons, statistical analysis of confusion errors, using one way ANOVAs, were limited to emotions where a significant group difference was found. Where the assumption of sphericity was violated, Greenhouse-Geisser corrections

were used. Where follow-up tests were required, Bonferroni corrections were used. Effect sizes were calculated as partial eta squared (η_p^2 ; small ≥ 0.01 , medium ≥ 0.06 , large ≥ 0.14 ; J. Cohen, 1988)

2.3. Results

Table 2.1 presents demographic and clinical characteristics. Groups differed in gender, where controls had the highest percentage of female participants, followed by ADHD, while there were least females in the ADHD+CD group ($X^2(2) = 7.4, p = .025$) and age ($F(2, 103) = 4.94, p = .009$). Emotion recognition accuracy did not differ between genders ($F(1,103) = 2.57, p = .11$); however, the relationship between age and emotion recognition approached significance ($r(104) = .054$). ANOVAs with and without age as covariate found the same pattern of results in terms of direction and significance, therefore results are presented without age as a covariate. ADHD+CD participants (as expected by definition) had significantly more CD symptoms than the ADHD alone group and controls ($p < .001$) and more ADHD symptoms than ADHD alone ($p = .042$). There was a significant group difference in CU traits ($p = .02$), with ADHD+CD having higher CU scores than ADHD ($p = .002$) and controls ($p = .035$), while ADHD and controls did not differ ($p = .66$)

Table 2.1. Demographic and clinical characteristics of sample

	ADHD+CD (n = 27)	ADHD (n = 36)	HC (n = 41)	P value	Posthoc
Age	13.7(2.18)	14.6(2.02)	15.51(2.68)	.009	ADHD+CD < HC
IQ _{Wasi}	80.5(13.72)	86.17(16.93)	NA	.17	
%_female	17.9	30.6	48.8	.03	
SES				.46	
% Low	60.7	41.7	43.6		
% Medium	28.6	44.4	35.9		
% High	10.7	13.9	20.5		
CD _{Dawba}	6.44(2.34)	1.0(1.03)	NA	<.001	
CD _{SDQ}	5.26(2.25)	2.74(1.44)	1.81(1.38)	<.001	ADHD+CD > ADHD ADHD+CD, ADHD > HC
ADHD _{Dawba}	14.15(3.61)	11.89(4.6)	NA	.042	
CU _{YPI}	36.59(8.03)	30.03(7.89)	32.04(5.68)	.002	ADHD, HC < CD

Note: Means are presented with standard deviations in brackets (except where indicated otherwise). Both ADHD_{Dawba} and CD_{Dawba} represent number of symptoms and are restricted to the ADHD groups. IQ is also restricted to ADHD groups. CD_{SDQ} is CD score as measured by SDQ. CU_{YPI} is the CU subscale for the YPI. *Key:* ADHD = Attention Deficit Hyperactivity Disorder. CD = Conduct Disorder. CU = Callous Unemotional. DAWBA = Development and wellbeing assessment. HC = Healthy controls. IQ = Intelligence quotient (2 subtest WASI). NA = Not Applicable. SDQ = Strengths and Difficulties Questionnaire. SES = Socioeconomic Status. YPI = Youth Psychopathy Inventory.

2.3.1. Facial Emotion Recognition

There was a main effect of Intensity ($F(1,101) = 382.4, p < .001, \eta^2 = .79$), with more accurate responses for higher intensity emotions ($M = 78.2, SD = 10.77$ vs. $M = 61.06, SD = 12.52$); there was no interaction between Intensity and Group ($F(2,101) = .443, p = .64, \eta^2 = .009$). Intensity was therefore dropped from further analysis. Recognition accuracy scores are presented in Figure 2.3.

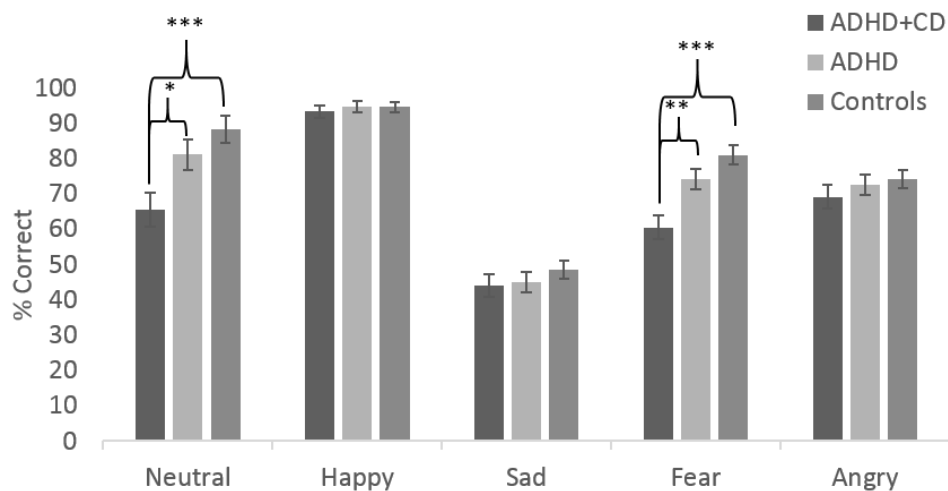


Figure 2.3. Percentage correct responses to each emotion for each group. * $p < .05$, ** $p < .01$, *** $p < .001$. Error bars denote ± 1 Standard error.

There was a main effect of Emotion ($F(2.76, 278.9) = 110.2, p < .001, \eta^2 = .52$), with highest accuracy scores to Happy ($M = 94.23, SD = 9.65$), followed by Neutral ($M = 79.81, SD = 26.75$), Fear ($M = 73.23, SD = 19.14$), Angry ($M = 72.28, SD = 16.97$) and Sad ($M = 45.99, SD = 16.63$). There was a main effect of Group ($F(2,101) = 9.6, p < .001, \eta^2 = .16$), and a significant interaction between Group and Emotion ($F(5.52, 278.9) = 2.98, p = .01, \eta^2 = .056$). Groups differed in Fear ($p < .001$) and Neutral ($p = .002$) recognition but not in other emotions ($ps > .05$). ADHD+CD were worse in Fear and Neutral recognition than ADHD ($p < .003$ and $p = .018$ respectively) and HC ($p < .001$ and $p < .001$ respectively), while there was no

difference in Fear or Neutral recognition between ADHD and HC ($ps > .05$).

Because most studies focused on recognition accuracy in males only, we conducted a sensitivity analysis restricted to males and found the same pattern of results with a significant interaction between Emotion and Group ($F(5.06, 164.3) = 2.71, p = .02$) and specific differences in fear recognition accuracy in Fear and Neutral between ADHD+CD and ADHD and controls ($ps < .05$), while there was no difference between ADHD and HC ($ps > .05$).

2.3.2. Confusion Matrix

2.3.2.1. Fear.

Patterns of errors made by the groups are presented in Table 2.2. There was a significant group difference in incorrect identification of Fear faces as Sad ($F(2,103) = 6.39, p = .002, \eta^2 = .11$). ADHD+CD made this error more than HC ($p < .002$). There was no difference between ADHD+CD and ADHD. There was a significant group difference in tendency to misinterpret Fear faces as Angry ($F(2,103) = 5.95, p = .004, \eta^2 = .11$); ADHD+CD made this error more than ADHD ($p = .04$) and HC ($p = .003$), with no difference between ADHD and HC ($p > .05$).

2.3.2.2. Neutral.

There was a significant group difference in tendency to judge Neutral faces as Happy ($F(2,103) = 5.17, p = .007, \eta^2 = .09$). ADHD+CD was more likely to make this error than both ADHD and HC ($ps < .01$), while there was no difference between ADHD and HC ($p > .05$). While there was also a group difference in propensity to judge Neutral faces as Sad ($F(2,103) = 5.24, p = .007, \eta^2 = .09$), pairwise comparisons indicated ADHD+CD committed this error more than HC ($p < .01$); the differences with ADHD approached significance ($p = .057$). There were no group

difference in tendencies to misattribute Neutral faces as Angry ($F(2,103) = .18, p = .98, \eta^2 < .001$), or Fearful ($F(2,103), p = .72, \eta^2 = .05$).

Chapter 2: Emotion Recognition

Table 2.2. Confusion matrices depicting the mean percentage groups selected each option for each presented emotion

	Emotion selected														
	Neutral			Happy			Sad			Fear			Angry		
	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3
Neutral	65.4^{ab}	81.0^a	88.21^b	11.1 ^{ab}	2.8 ^a	3.7 ^b	12.4 ^a	5.6	1.2 ^a	3.7	3.7	0.0	7.4	6.9	6.9
Happy	4.6	3.4	4.8	93.2	94.7	94.2	.93	.46	.41	.93	.93	0.0	.31	0	.20
Sad	37.0	38.9	41.36	4.6	2.3	1.2	43.8	44.9	48.4	8.0	9.3	5.9	6.5	3.7	3.3
Fear	12.7	8.1	8.9	1.5	1.6	0.81	13.3 ^b	10.0	5.5 ^b	60.5^{ab}	74.1^a	80.9^b	11.4 ^{ab}	5.6 ^a	3.9 ^b
Angry	10.5	10.9	17.1	.31	1.2	.61	9.3	3.7	3.1	10.5	10.9	5.1	69.1	72.5	74.2

Notes: Bold values depict correct responses. 1 = ADHD+CD. 2 = ADHD. 3 = Controls ^a Significant difference between ADHD+CD and ADHD ^b Significant difference between ADHD+CD and Control

2.3.3. Eye-tracking

Examination of participants' eye-gaze validity led to the exclusion of three ADHD+CD, three ADHD, and one HC participant.

2.3.3.1. Percentage of time spent looking at the eyes.

A three way ANOVA with Emotion, Intensity and Group revealed a main effect of intensity ($F(1, 94) = 5.22, p = .025, \eta^2 = .053$), with participants spending more time focusing on the eyes for higher ($M = 56.07, SD = 17.48$) compared to lower intensity ($M = 54.71, SD = 17.48$) faces. There was no interaction between Emotion and Intensity ($F(3, 282) = .57, p = .64, \eta^2 = .006$), nor an interaction between Intensity and Group ($F(2,94) = .602, p = .55, \eta^2 = .013$). Emotion intensities were therefore collapsed.

A main effect of Emotion was found ($F(3.54, 332.86) = 26.72, p < .001, \eta^2 = .22$; see Figure 2.4, Panel A). Participants focused most on the eyes for Sad faces

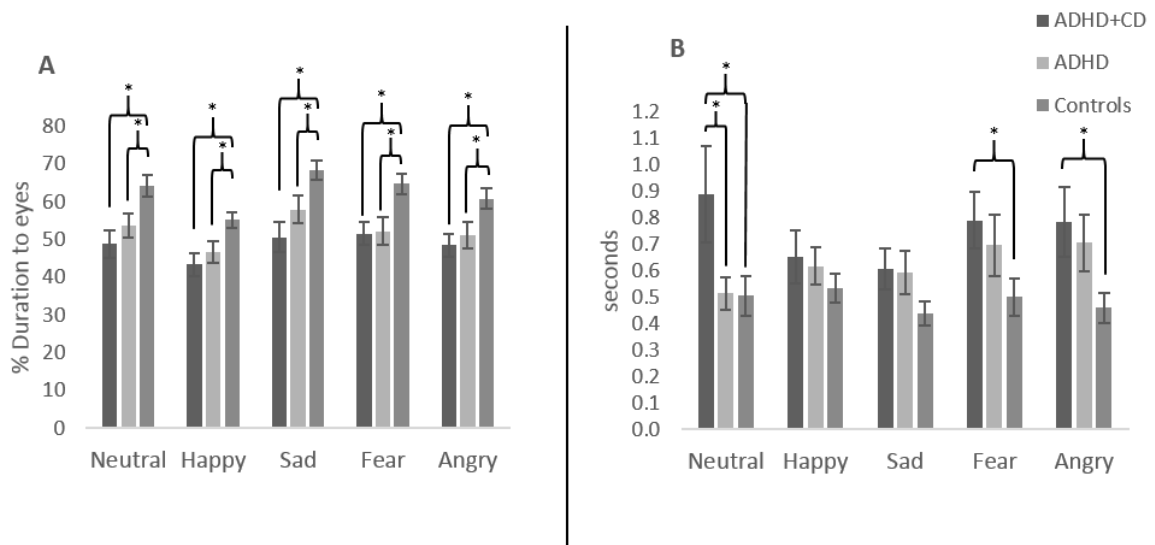


Figure 2.4. Panel A. Percentage fixations on the eye region out of all fixations on the face. Panel B. Mean time to first fixation on eye region. $*p < .05$, $**p < .01$, $***p < .001$. Error bars denote ± 1 Standard error.

($M = 60.49, SD = 19.65$), followed by Fear ($M = 57.23, SD = 18.89$), Neutral ($M =$

56.79, $SD = 18.54$), Angry ($M = 54.47$, $SD = 18.07$), and Happy ($M = 49.4$, $SD = 15.71$) faces. There was also a main effect of Group ($F(2, 94) = 6.81$, $p = .002$, $\eta^2 = .13$) with both ADHD+CD and ADHD groups spending less time focusing on the eyes than HC ($ps < .05$) while there was no difference between ADHD+CD and ADHD ($p > .05$). There was no interaction between Emotion and Group ($F(7.08, 332.86) = 1.33$, $p = .24$, $\eta^2 = .03$).

2.3.3.2. Time to first fixation (TFF) on eye-region.

After removing participants with a mean TFF of zero; 22 ADHD+CD, 31 ADHD and 35 HC participants remained. There was no main effect of Emotion ($F(3.25, 275.86) = 1.76$, $p = .15$, $\eta^2 = .02$; see Figure 2.4, Panel B), a main effect of Group ($F(2,85) = 3.25$, $p = .04$, $\eta^2 = .07$), but no interaction between Group and Emotion ($F(6.49, 275.86) = 1.82$, $p = .089$, $\eta^2 = .04$). ADHD+CD were slower to fixate to the eyes than HC ($p < .05$), but there was no difference between ADHD+CD and ADHD, nor between ADHD and HC ($ps > .05$). Pairwise comparisons indicated ADHD+CD were slower to fixate the eye region than HC for Fear and Angry faces ($ps < .05$), while ADHD+CD was slower than both ADHD and HC for Neutral faces ($ps < .05$). There were no differences between groups for the other emotions ($ps > .05$).

2.3.4. Relationships between Clinical Characteristics, Emotion Recognition and Fixation to Eyes

Table 2.3 presents correlations between clinical characteristics, emotion recognition and fixation duration for those emotions for which there was a significant group difference. A negative relationship was found between severity of CD and emotion recognition within those with ADHD. CD symptoms did not relate to time spent looking at the eyes. ADHD symptom severity was negatively related to percentage of time spent looking at the eyes, but was not related to emotion recognition

accuracy. Across the sample as a whole, there was a positive relationship between emotion recognition and percentage time spent looking at the eyes.

Table 2.3. Relationships between clinical variables, emotion recognition accuracy and percentage fixation duration to the eyes

	1	2	3	4	5	6	7	8	9
1. CD _{Dawba}	-								
2. CD _{SDQ}	.53***	-							
3. CU _{YPI}	.51***	.37***	-						
4. ADHD _{Dawba}	.33*	.22 ^a	.17	-					
5. Emotion accuracy overall	-.23	-.29**	-.19*	-.15	-				
6. Fear accuracy	-.27*	-.29**	-.20*	-.19	.67***	-			
7. Neutral accuracy	-.24	-.24*	-.08	-.23	.48***	.28**	-		
8. % Fear eyes	-.11	-.25*	-.19	-.35*	.37***	.34**	.35***	-	
9. % Neutral eyes	-.12	-.26*	-.16	-.45**	.32**	.28**	.28**	.81***	-
10. % Eyes all	-.17	-.26*	-.19	-.41**	.31**	.31**	.32**	.95***	.91***

Notes: Correlations involving CD_{Dawba} or ADHD_{Dawba} only include participants in either of the ADHD groups, r = Pearson's r , CD_{Dawba} = conduct symptoms measured by the dawba, CD_{SDQ} = conduct score measured by SDQ, CU_{YPI} = Callous unemotional traits measured by youth psychopathy inventory. ADHD_{Dawba} = Attention deficit hyperactivity symptoms measured by the DAWBA. DAWBA = Diagnostic and Wellbeing Assessment. % = Percentage fixation duration * p < .05, ** p < .01, *** p < .001.

2.4. Discussion

We compared emotion recognition between ADHD+CD, ADHD alone and healthy control participants to ascertain if emotion recognition impairments were generally a problem for those with a diagnosis of ADHD. We found support for the hypothesis that these impairments are specific to CD, finding specific Fear and Neutral recognition impairments. Although a null result is not evidence of equivalent performance, the lack of significant differences between ADHD and control participants taken together with previous studies of those with conduct disorder supports the idea that recognition deficits are specific to those with CD. Our findings are in line with studies showing deficits in fear recognition in antisocial populations (Dadds et al., 2006; Dadds et al., 2008; Marsh & Blair, 2008), but not consistent with some studies in antisocial or CD samples that found additional impairments in Sadness (Bowen et al., 2014; Hubble et al., 2015) or Anger (Fairchild et al., 2010; Sully et al., 2015). However, differences in sample composition and/or design specifics of the emotion recognition tasks may explain this.

Absence of a difference in emotion recognition performance between ADHD alone and typically developing controls is inconsistent with some studies that have previously found recognition deficits in ADHD (Pelc et al., 2006; Singh et al., 1998). However, participants in Pelc et al., (Pelc et al., 2006) were considerably younger (7 – 12 years), whereas Singh et al.'s (Singh et al., 1998) protocol did not include a pure measure of facial emotion recognition. Our findings are in line with other studies that found emotion (recognition) impairments are limited to those with comorbid CD (Northover et al., 2015a; Schwenck et al., 2013; van Goozen et al., 2016).

The study's second aim was to gain a clearer understanding of the mechanisms involved in emotion recognition impairments. Although it has been claimed that those with aggressive behaviour have a hostile attribution bias (Dodge et al., 1990), participants in the ADHD+CD group were not more prone to confuse Neutral with Anger. As the hostile attribution bias was originally found in a study examining the proposed intention of a character in a situational context (Dodge et al., 1990), it is possible that the bias is specific to attributions of intent. However, ADHD+CD participants were more likely to misattribute Fear as Anger. If in a confrontational situation Fear is misinterpreted as Anger, the inhibitory cue to an (already) aggressive individual is absent and may lead to more aggression and violence. We also found that those with ADHD+CD had a tendency to confuse fear with sadness, which does lead to an inhibited response, but may be a less potent inhibitor. It may thus be the case that there is a general difficulty in the interpretation of fearful features. Interestingly, we found an inverse relationship between CD severity and emotion recognition accuracy, providing further evidence that emotion recognition in general, and fear recognition in particular, is a problem for those with ADHD and comorbid CD.

We also measured participants' eye-gaze, finding participants in ADHD groups looked towards the eyes less for all emotions compared to healthy controls. This suggests lack of attention to the eye-region is a problem for ADHD generally rather than a specific problem for CD. The fact that a lack of focus on the eye region was present in both ADHD groups rather than specifically in those with CD suggests that emotion recognition problems are not uniquely due to a lack of attention to the eye region. This is inconsistent with a study that found directing participants to look towards the eyes eliminated fear recognition impairments (Dadds et al., 2006).

However, participants in that study were typically developing undergraduates, divided into those with high or low CU traits and therefore a different sample from the current study. Relatedly, CD severity was more strongly related to recognition impairments than were CU traits.

Although we found a positive relationship between looking towards the eyes and emotion recognition across the entire sample, ADHD groups did not differ in their percentage of time looking at the eyes and yet differed in emotion performance, suggesting other factors are involved. Lack of attention to the eyes is only one explanation, and those with ADHD without CD may have alternative strategies to help them interpret faces from information they do process.

Our findings are important for the design of interventions targeting emotion recognition deficits as a cognitive mechanism underlying conduct disorder including in those with ADHD. In the juvenile offender emotion intervention study (Hubble et al., 2015), participants were not only taught to pay attention to salient features of the face, but hints were also given to assist with interpretation of features (Hubble et al., 2015). The current findings suggest the improvements observed may have been due to help with the interpretation of features rather than redirecting attention.

In support of the finding that neglect of the eye-region is a problem for those with ADHD generally, we found a stronger negative correlation between ADHD severity and time spent looking at the eyes than with CD severity. This suggests that ADHD symptomatology rather than CD is driving this attentional problem.

This is the first study to examine eye-gaze in ADHD when completing an emotion recognition paradigm. The finding that ADHD participants look less at the eye region of a face is in line with a recent study that found ADHD participants were less distracted by the eye-gaze of distractor faces while performing a word

classification task compared to controls, suggesting the attention of those participants with ADHD was captured less by eye-gaze (Marotta et al., 2017).

We also examined time to first fixation on the eye-region; although they were no different to ADHD alone, ADHD+CD participants were slower to engage attention to this region than controls. This suggests it takes longer for those with CD to engage with this important aspect of the face. If the paradigm had been time limited, ADHD+CD participants might have shown stronger recognition impairments due to not having the opportunity to process this region.

2.4.1. Limitations

First, although we used a sample of adolescents with ADHD, categorised them into those with or without CD, and compared them to typical developing healthy controls matched for SES, the study would be strengthened further if a sample of adolescents with CD alone had been included.

Second, time limitations prevented collection of IQ data in controls. It is possible that performance was influenced by IQ. However, there was no difference in IQ between the two ADHD subgroups, yet they performed differently in emotion recognition.

Finally, one could argue that the paradigm lacks ecological validity. Participants had unlimited time to process the emotion and respond. This decision was initially made so participants would not need to respond from memory. However, real life scenarios may not allow for such an opportunity. It is therefore possible that participants' emotion recognition accuracy was overestimated and larger differences between groups might have emerged if less time had been given.

2.4.2. Conclusions and Implications

Using a clinical sample of ADHD participants split into those with and without comorbid CD and compared to a sample of typically developing controls, we found that Fear and Neutral face recognition is specifically impaired in those with conduct problems. We found that a lack of attention to the eye-region of faces is a general problem for those with a diagnosis of ADHD and is not specific to those with additional conduct problems. Our findings add to discussions about the hostile attribution bias theory, finding no evidence of an increased inclination to interpret Neutral faces as Angry in those with ADHD with or without CD. Instead, there was evidence to suggest that ADHD+CD misattributed Fear for Anger, which could lead to further behavioural problems (Blair, 2005). Individuals with ADHD and comorbid CD have problems with the interpretation of specific emotional features and this is relevant for interventions seeking to reduce their conduct problems by improving emotion recognition. These interventions should target the interpretation of the facial configuration of an emotional expression by individuals at risk of future conduct problem.

3. Chapter 3: Empathy

Paper in preparation

This chapter is based on Airdrie, J. N., Langley, K., Thapar, A., van Goozen, S. H. M. (2017). Heterogeneity and the role of social attention in empathy impairments in Attention-Deficit/Hyperactivity Disorder and comorbid Conduct Disorder.
In prep

3.1. Introduction

Chapter 2 found that impairments in the recognition of fearful and neutral faces were specific to those with ADHD+CD. It also found that a lack of attention to the eye region did not explain the impairment apparent in this group. Next, the current chapter will explore cognitive and affective empathy performance and the role of attention to the eye region in those with ADHD and ADHD+CD. In addition, it will consider the role of key clinical characteristics in empathy performance. As mentioned in Chapter 1, tasks from all chapters were conducted contemporaneously and therefore results of Chapter 2 did not influence our hypotheses for the current chapter.

Humans are social beings. Their behaviour is shaped and directed toward other people in a social group (Batson, 1990). One mechanism via which such social behaviour is facilitated is empathy (Decety et al., 2016). Many definitions of empathy have been proposed in the literature (see Preston & de Waal, 2002), but broadly, it refers to the understanding and sharing of another's emotional state (Eisenberg & Strayer, 1990). Empathy is said to occur where one is in the same affective state as another person, said state has resulted from the observation or imagination of that other person's emotional state and one is able to identify the source of their own state as resulting from the other person (de Vignemont & Singer, 2006).

The above definition is a broad and overarching view of empathy, however, it is not a unitary construct and can be broken down further into its subcomponents of affective (or emotional), cognitive and motor empathy (Blair, 2005b). Affective empathy involves the vicarious experience of emotions consistent with those of

another (Bons et al., 2013). Cognitive empathy is the ability to understand what another person is thinking or feeling, and involves representing another's thoughts, desires, beliefs, intentions and knowledge (Blair, 2005b). Finally, motor empathy refers to the ability to automatically and unconsciously mirror the facial expression of another person (also known as facial mimicry; Blair, 2005).

One important function of empathy is to inhibit aggressive and antisocial behaviour (Hoffman, 1987; Miller & Eisenberg, 1988). A lack of empathy has long been considered a core feature of antisocial behaviour (Redl & Wineman, 1951). As outlined earlier, one group of individuals who demonstrate antisocial behaviour are adolescents with a diagnosis of CD (Cohen & Strayer, 1996). Studies have found that those with CD are lower in empathy (both cognitive and affective) than typically developing controls (Anastassiou-Hadjicharalambous & Warden, 2008; Cohen & Strayer, 1996). Such lack of empathy has been found to be important in the prediction of aggressive behaviour in these individuals. In a recent genetic study, van Goozen et al., (2016) found that a lack of affective empathy, specifically for fear, mediated the relationship between genetic risk and aggressive behaviour in adolescents with Attention/Deficit Hyperactivity Disorder (ADHD) and CD. In addition, Miller, Johnston, and Pasalich, (2014) found that child empathy moderated the relationship between parenting style and conduct problems such that mother praise was found to be negatively related to child conduct problems, but only for children with low levels of empathy. On the other hand, mother criticism was positively related to child conduct problems only for those children high in empathy.

ADHD has also been found to be associated with empathy deficits is ADHD. The self-regulatory skills that adolescents with ADHD are supposed to lack are a prerequisite for the perspective taking required for empathy, as individuals need to

inhibit their responses long enough to consider how someone else's perspective might differ from their own (Barkley, 2006). As a result, due to poor inhibitory control, those with ADHD would be less responsive to the needs, feelings and opinions of others stemming from their reduced ability to interpret events from the perspective of another. Indeed, studies have found that adolescents with ADHD were less empathic than typically developing controls (Braaten & Rosen, 2000; Dyck et al., 2001). However, due to the high comorbidity with CD, it is not clear if such empathy deficits result from problems associated with ADHD symptoms or CD symptoms. For example, Marton, Wiener, Rogers, Moore, and Tannock (2009) found that ADHD children were rated as less empathic by their parents, but this was accounted for by co-occurring conduct problems.

A further source of heterogeneity in those with CD is the presence of callous-unemotional (CU) traits (Frick & Ellis, 1999). Such traits are analogous to the interpersonal-affective dimension of adult psychopathy and have recently been included in the Diagnostic and Statistical Manual of Mental Disorders (DSM-V; APA 2013) as a specifier in the diagnosis of CD. Adolescents with a diagnosis of CD who also possess CU traits are characterised by a lack of guilt and empathy and make use others for their own gain and have a particularly stable, severe and aggressive pattern of antisocial behaviour (see Frick & White (2008) for a review). It has been argued that the presence or absence of such traits in those with CD accounts for the rather moderate relationships between aggression and lack of empathy in systematic reviews and meta-analyses (Lovett & Sheffield, 2007; Miller & Eisenberg, 1988). Lovett and Sheffield (2007) suggest that empathy deficits may only be related to proactive types of aggression, while reactive forms of aggression result from problems with emotion regulation. It is proactive aggression that is

observed in those with CU traits. Other studies have found that CU traits may have a role in different profiles of empathy deficits. For example, Anastassiou-Hadjicharalambous and Warden (2008) found both cognitive and affective deficits in those with CD and low CU traits but only problems with affective empathy in those with CD and high CU traits. While other studies have found that those high in CU traits have problems with both cognitive and affective empathy over and above those problems associated with CD, or the quality of family relationships (Pasalich et al., 2014).

As a result of heterogeneity in those with ADHD, the primary aim of the current study was to compare those with a diagnosis of ADHD with and without comorbid CD in their cognitive and affective empathy in response to emotional film clips, and to assess the contribution of CU traits and CD and ADHD severity in explaining such differences.

Another open question in the empathy literature is the mechanism via which empathy impairments occur. It has been suggested that a lack of attention to the eye region of the face results in empathy impairments in those with CD (Bons et al., 2013). Indeed, the eyes are crucial in social processing (Gliga & Csibra, 2007). Healthy individuals first fixate, and spend more time looking at the eyes than other features of the face (Itier & Batty, 2009), while eye to eye exchanges are considered vital in establishing or extinguishing social interactions (Mason, Tatkow, & Macrae, 2005). Cowan, Vanman and Nielsen (2014) found a positive relationship between trait empathy and looking towards the eyes during an emotional film clip. In addition, the amygdala has been found to be less responsive in those with CD and a hypothesised role of the amygdala is the direction of attention to salient features of the face such as the eyes (Gamer & Buchel, 2009). A lack of attention to the eyes has

also been suggested as important in impairments in the recognition of emotional expressions (Bowen, Morgan, Moore, & van Goozen, 2014; Dadds, El Masry, Wimialaweera, & Guastella, 2008; Hubble, Bowen, Moore, & van Goozen, 2015; although this was not supported by the findings of Chapter 2) and such recognition is thought to be a precursor to empathy (Blair, 2005). Therefore, it seems a lack of attention to the eye-region is a good candidate mechanism to explore in terms of empathy impairments. To date only one very recent study has explored the relationship between attention to the eye region and empathy (van Zonneveld et al., 2017). The authors in this study explored differences in empathy and social attention in a group of children at high risk of criminal behaviour. Despite finding evidence of reduced affective empathy in the high-risk group compared to controls, groups did not differ in social attention. However, this group of children were only the younger siblings of young offenders or children of delinquent parents and did not have a diagnosis of CD themselves, therefore no study has yet examined social attention and empathy in those with CD.

The role of attention in explaining empathy performance in those with ADHD and CD is complicated further by the presence of comorbidities and different subtypes in those with CD. CU traits have also been implicated in attentional deficits to emotional stimuli. Kimonis, Frick, Fazekas, and Loney (2006) found that children high in CU traits exhibited less attentional orientating to distressing pictorial stimuli, while Moul, Killcross, and Dadds (2012) suggest that emotional stimuli that would grab the attention of non-psychopaths, fail to elicit the same allocation of attention in psychopaths. Attentional deficits are also a core problem for those with a diagnosis of ADHD (APA, 2013). Therefore, it is still unclear if attentional deficits to the eye region found in those with a diagnosis of CD are driven by CU traits, ADHD

symptoms or if they are a problem specific to CD. Therefore, the secondary aim of the current study was to investigate the difference between ADHD with and without comorbid CD in preference to look towards the eyes during an emotional film clip, while exploring the impact of CU traits on such differences, and assessing the relationship between eye preference and cognitive and affective empathy.

In order to explore these open issues, adolescents with a diagnosis of ADHD with or without comorbid CD had their cognitive and affective empathy in response to emotional film clips assessed while their attention was measured via eye-tracking. On the basis of findings in the emotion recognition literature (Bowen et al., 2014; Blair, 2005) we predicted participants with additional CD would only show empathy impairments for negative emotions, and that these would be confined to affective empathy (van Goozen et al., 2016), while we predict no difference in positive emotions. We predicted that both CD severity and CU traits would be negatively related to affective empathy ability while ADHD severity would not. Similarly, in terms of attention, we predicted that those with ADHD+CD would have less of a preference towards the eyes for negative emotions (Kimonis et al., 2006; Moul et al., 2012). Again, we predicted that this difference would be explained by higher CD severity and CU traits. We also predicted that that a preference towards the eyes would positively predict level of affective empathy.

3.2. Method

3.2.1. Participants

Fifty two adolescents (16 females) between 11 and 18 years ($M = 13.8$, $SD = 2.2$) with an ADHD diagnosis participated in the current study. 30 participants were in the ADHD alone group while 22 were in the ADHD+CD group.

See section 2.2.1 for information on recruitment, exclusion criteria and ethical approval.

3.2.2. Measures and Materials

3.2.2.1. Empathy task: emotion-eliciting video clips

We used six clips depicting main characters feeling sad, happy or fear (van Goozen et al., 2016; van Rijn, Barendse, van Goozen, & Swaab, 2014; van Zonneveld et al., 2017). The clips were edited from commercially available films to have a duration of approximately 120 seconds. Where possible these clips showed real people experiencing real-life emotional events. Two clips matched for duration and intensity (as determined by a prior pilot study) represented each target emotion. A description of these can be found in Appendix A. Sad (1), happy (1) and fear (1 and 2) were edited from cinematic films whilst sad (2) and happy (2) depicted real people describing real events. Films were shown via Tobii Studio. Participants were simply asked to watch each clip and were not provided with any other information or instructions until the end of the clip.

3.2.2.2. Explicit empathy test

After each clip participants completed two questionnaires, one concerning the emotions of the main character and the other concerning their own emotions while viewing the clip. Participants indicated how strongly they or the main character felt a range of eight emotions (labelled as follows: anger, sad, upset, fear, happy, scared, cheerful, surprise) on a scale of 0 (not at all) to 10 (very much). Participants were asked to give the reason for the emotion they identified in the main characters and in themselves. The experimenter went through each emotion that was identified as being present and asked the participant “what happened in the clip to make you (or the main character) feel [insert emotion]?” These responses were coded for cognitive

and affective empathy using the Cardiff Empathy Scoring System (CESS; Van Goozen et al., 2016; Van Rijn, Barendse, Van Goozen, & Swaab, 2014; van Zonneveld, Platje, de Sonnevile, van Goozen, & Swaab, 2017). This took into consideration four elements of empathy: (1) whether the target emotion was correctly identified; (2) whether other similar emotions were identified; (3) the intensity of the emotion(s) identified and (4) the explanation for the causes of the emotion. Participants could score between 0 and 9 for cognitive empathy while they could score between 0 and 6 for affective empathy. For all scales, a higher score was indicative of greater empathy (for a full explanation as to scoring criteria see Appendix B). Interrater reliability ranged from .91 (cognitive) to .99 (affective).

Participants' previous experience of the film clip was checked after each clip by informing the participants which film it was from and asking whether they had seen the film before. Three participants had seen sad (1), 20 had seen happy (1), 34 had seen fear (1), three had seen sad (2), five had seen happy (2), and eight had seen fear (2). The effect of film familiarity was checked before analysis to examine whether previous experience with a film affected the intensity of the emotions observed in the main character or experienced by the participant. Between subjects *t*-tests revealed that there were no significant differences in emotional intensity experienced by participants or observed in the main character (all *p*'s >.05) between those who had or had not seen the clips before; as a result, film familiarity was not included in the main analysis.

3.2.2.3. Clinical Measures.

See section 2.2.2.1. The internal reliability of CD ($\alpha = .74$) and ADHD symptom severity ($\alpha = .86$), YPI overall ($\alpha = .93$) and the CU traits subscale ($\alpha = .81$) was high.

3.2.2.4. Socioeconomic Status (SES)

See section 2.2.2.2

3.2.2.5. Intelligence Quotient (IQ)

See section 2.2.2.3

3.2.2.6. Eye-tracking.

Participants were positioned 60-65cm from a laptop screen and a 9-point calibration was performed before each of the two sets of three videos. The quality of calibration was checked and repeated as required. Following successful calibration a set of three videos was shown. See section 2.2.2.5 for additional information on eye-tracker recording.

3.2.3. Procedure

Participants were tested in a dimly lit laboratory room in a university clinic. The empathy eliciting clips were shown in two sets of three videos each, the order of which was counterbalanced across participants. Each set included a clip with characters depicting sad, happy and fear as the target emotion. Eye-tracking calibration was performed before each of the video sets. Participants watched the video first, following this they rated how strongly they thought the character felt each emotion (cognitive empathy), they then rated how much they felt each emotion (affective empathy), and finally participants were asked for reasons as to why they (affective) and the character (cognitive) felt each emotion.

3.2.4. Data analysis

3.2.4.1. Empathy

Factorial ANOVAs (and ANCOVAs where necessary) were run separately for cognitive and affective empathy with Group (ADHD or ADHD+CD) as a between-subjects factor, and Emotion (sad, happy, and fear) as a within-subject factors.

3.2.4.2. Eye-tracking

Eye-gaze was analysed during a 4-second segment that was independently judged (via a pilot study) to have included the highest emotional content in each clip. Tobii analysis software was used to analyse eye-movements, which allowed dynamic areas of interest (AOI) to be created and a variety of summary reports generated. The eyes were grouped into one area. A second AOI was created around the mouth. A third AOI was around the face as a whole to allow for analysis of when participants were looking at the face. A final AOI was created around the screen as a whole in order to determine the percentage of time participants were looking at the screen during the analysis segment. Percentage dwell time to the eyes and mouth were calculated by summing all fixations to the AOIs and divided by the sum of fixation durations to the face AOI. Percentage dwell time to the face was calculated by summing fixations to the face AOI and dividing by sum of fixations to the screen AOI. Preference to look at the eyes over the mouth (eye-preference) was calculated by subtracting percentage dwell time to the mouth from percentage dwell time to the eyes. Eye-gaze validity for each film clip was calculated by summing the fixation durations to the screen during the segment and dividing it by the total segment analysis time. Participants whose validity fell below 60% for each clip were excluded from analyses related to that clip. Due to the different numbers of participants excluded from each clip, analyses for eye-tracking variables for each film clip were analysed separately with univariate ANOVAs with Group as a between-subjects factor. For the same reasons regression analyses including eye-tracking measures from each clip and clinical characteristics differ in sample size from the main ANOVA analyses.

Analyses were carried out using SPSS 20 (SPSS Inc., Chicago, Illinois).

Where the assumption of sphericity was violated, the Greenhouse-Geisser correction

was used. Where follow-up tests were required, Bonferroni corrections were used. Effect sizes were calculated as partial eta squared (η^2 ; small $\geq .01$, medium $\geq .06$, large $\geq .14$; Cohen, 1988). Where differences in empathy or eye-gaze were found between groups, correlational and linear regression analyses were carried out to assess the relative contribution of personality and eye-gaze measures in explaining empathic abilities and also to assess the contribution of personality and clinical measures in explaining eye-gaze behaviour. Where differences in empathy were found between groups, the role of key clinical and eye gaze variables in such differences were explored with mediation and exploratory moderation analyses using the PROCESS procedure developed for SPSS (Hayes, 2013). This technique allows the implementation of a bootstrapping method. The indirect (mediated) effects were estimated by computation of bias-corrected bootstrap confidence intervals (CI; 95%) based on multiple resamples of the data. This procedure overcomes problems of non-normal distribution. Standardised beta coefficients are reported.

3.3. Results

Table 3.1 presents demographic and clinical characteristics of the sample. As expected, groups differed in CD severity, while ADHD+CD were higher in ADHD severity and also reported more CU traits. ADHD+CD participants had lower IQ than the ADHD group, but did not differ in any other demographic characteristics. IQ did not correlate with either cognitive ($p = .38$) or affective ($p = .15$) empathy and therefore it was decided not to enter IQ into subsequent analyses. Due to group differences in ADHD severity, this was included as a covariate in analysis of differences in Empathy to ensure any difference were reflective of additional conduct problems and not severity of ADHD.

Table 3.1. Demographic and clinical characteristics of the sample

	ADHD+CD (n = 22)	ADHD (n = 30)	P value
Age	13.6(2.3)	13.9(2.2)	.66
IQ _{Wasi}	85.4(11.2)	93.3(12.0)	.02
% female	20.0	18.2	.87
SES			.14
% Low	57.1	37.0	
% Medium	23.8	51.9	
% High	19.0	11.1	
CD _{Dawba}	5.9(2.5)	.87(.97)	<.001
ADHD _{Dawba}	14.1(3.6)	11.5(4.9)	.047
CU _{YPI}	36.3(7.8)	28.3(7.9)	.001

Note: Means are presented with standard deviations in brackets (except where indicated otherwise). Both ADHD_{Dawba} and CD_{Dawba} represent number of symptoms and are restricted to the ADHD groups. IQ is also restricted to ADHD groups. CD_{SDQ} is CD score as measured by SDQ. CU_{YPI} is the CU subscale for the YPI. *Key:* ADHD = Attention Deficit Hyperactivity Disorder. CD = Conduct Disorder. CU = Callous Unemotional. DAWBA = Development and wellbeing assessment. IQ = Intelligence quotient (2 subtest WASI). SES = Socioeconomic Status. YPI = Youth Psychopathy Inventory.

3.3.1. Empathy

Seven participants were excluded for the ANOVA of empathy scores due to missing data in one or more videos, leaving 17 ADHD+CD and 28 ADHD participants.

Figure 3.1 presents mean scores for cognitive and affective empathy.

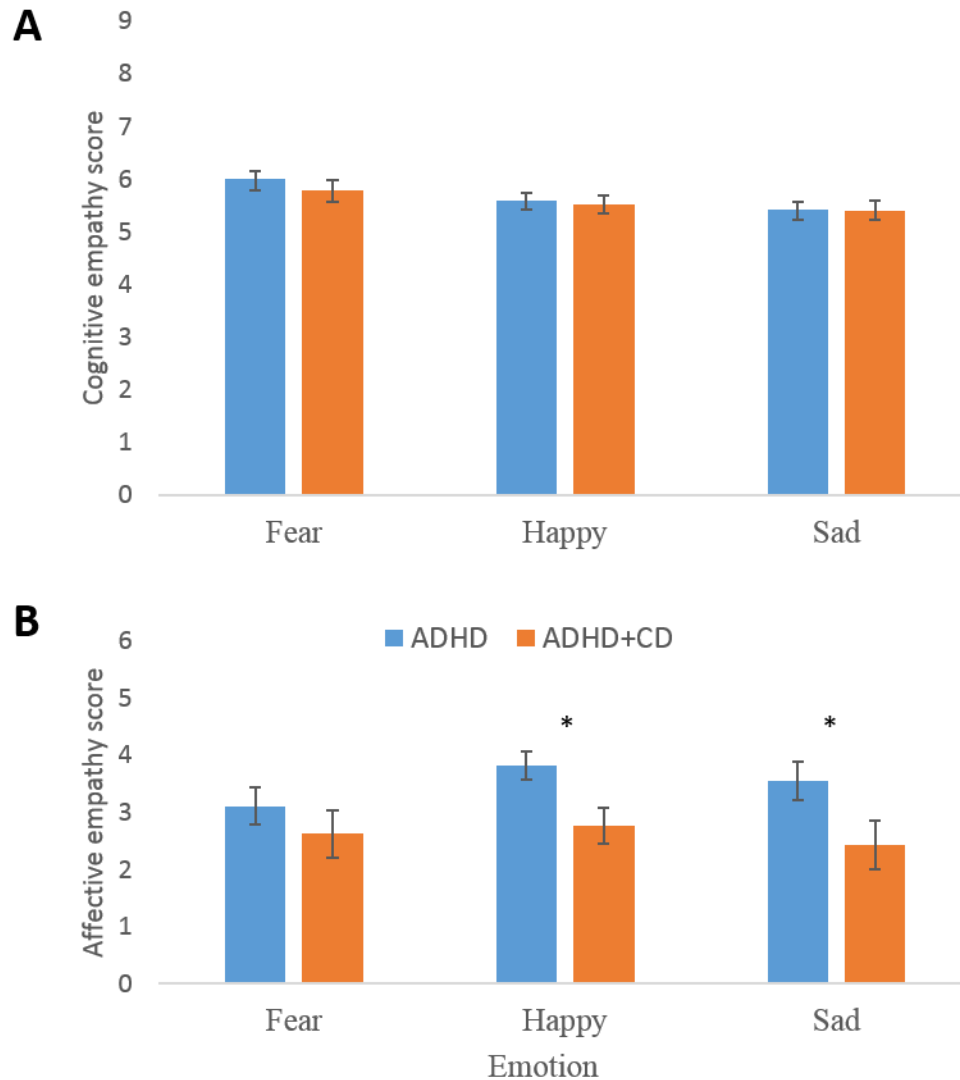


Figure 3.1. Mean scores for cognitive (Panel A) and affective empathy (Panel B). * $p < .05$ Error bars depict ± 1 Standard error

3.3.1.1. Cognitive Empathy.

The ANOVA for cognitive empathy revealed no differences between Groups ($F(1, 42) = .35, p > .05, \eta^2 = .008$), no main effect of Emotion ($F(1, 84) = .60, p > .05,$

$\eta^2 = .014$), nor an interaction between Emotion and Group ($F(2, 84) = .27, p > .05, \eta^2 = .006$).

3.3.1.2. Affective Empathy.

The ANOVA for affective empathy revealed a main effect of Group ($F(1, 42) = 4.35, p < .05, \eta^2 = .09$), but the main effect of Emotion ($F(2, 84) = .16, p > .05, \eta^2 = .004$) and the interaction between Emotion and Group were not significant ($F(2, 84) = 1.15, p > .05, \eta^2 = .027$). Follow-up tests revealed that the ADHD+CD group scored lower than ADHD group and that the significant group effect was specifically caused by a difference on the Happy ($p = .012$) and Sad ($p = .049$) film clips, but not the Fear ($p > .05$) clip (See Figure 3.1).

3.3.2. Eye-Gaze

Eye-gaze data was not useable for two of the six video clips. For one of the Happy clips there was interference in the video at the point of AOI analysis while for one of the Sad clips a high number of participants' (35%) eye-tracking validity fell below 60%. For the remaining videos, mean eye-preference was calculated across both the Fear clips while the single Happy and Sad clips were analysed separately. Eight participants' each for the Fear and Sad clips, and 12 participants' for the Happy clip fell below 60% and were excluded from analysis.

Differences between groups in eye-preference (positive values represent preference for the eyes over the mouth while negative values indicate preference to the mouth over the eyes) are presented in Figure 3.2.

We found no difference between groups for Fear ($F(1, 42) = 1.03, p = .32, \eta^2 = .024$) but found ADHD+CD participants looked at the eyes less than ADHD alone participants for both the Happy ($F(1,38) = 4.74, p = .036, \eta^2 = .11$) and Sad ($F(1, 42) = 4.44, p = .041, \eta^2 = .096$) clips.

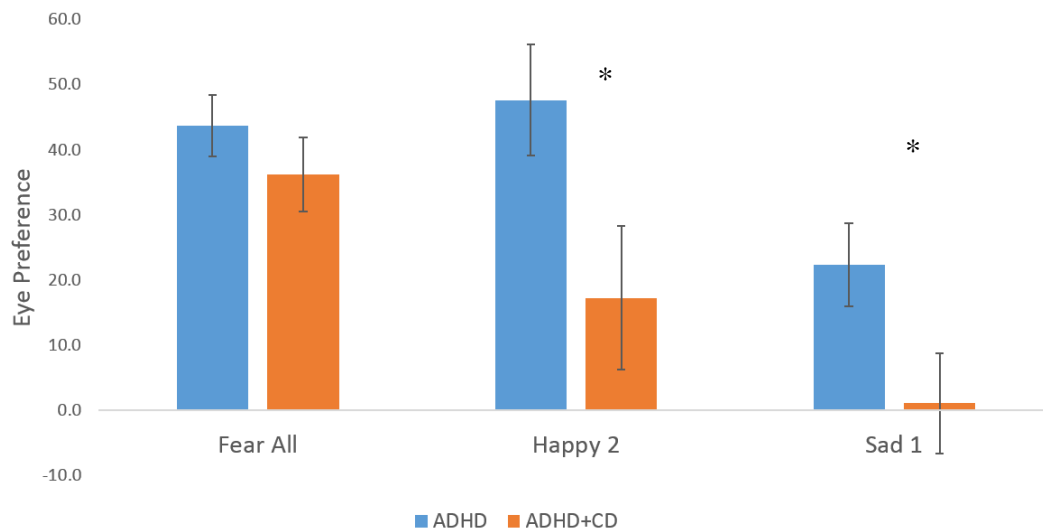


Figure 3.2. Difference in eye preference between ADHD groups. $*p < .05$. Positive scores reflect a preference to look at the eyes over the mouth. Error bars depict ± 1 Standard error.

3.3.3. Exploring Group Differences Using Clinical Characteristics and Eye-Gaze

3.3.3.1. Clinical characteristics explaining empathy impairments.

Relationships between clinical characteristics and affective empathy for Fear, Sad and Happy were explored with correlational analyses (see Table 3.2) and subsequently, mediation and moderation analysis. As we did not anticipate group differences in empathy for Happy, no predictions were made about the relationship between Happy empathy and clinical characteristics and eye gaze, however since group differences did emerge this difference warranted further exploration. It is important to be clear that these analyses were run post-hoc and therefore should be interpreted in light of this.

Table 3.2. Correlation matrix of affective empathy scores and clinical characteristics

	1	2	3	4	5
1.Fear	-				
2.Happy	.51***	-			
3.Sad	.69***	.52***	-		
4.CD severity	-.17	-.37**	-.36*	-	
5.CU	-.29#	-.11	-.47**	.50***	-
6.ADHD severity	-.05	.15	-.03	.26	.33*

* $P < .05$. ** $p < .01$. *** $p < .001$. # $p = .056$

Correlations are Pearson's r

When entered into a mediation analysis the observed difference between ADHD+CD and ADHD in affective empathy for Sad was mediated through CU traits (see Figure 3.3, Panel A). The estimate of the indirect effect (Path A*B) reached significance as indicated by the 95% CI excluding zero ($\beta = -0.22$, 95% CI [-.46, -.06]) with the negative relationship indicating that affective empathy for Sad decreased as the number of CU traits increased. Due to follow-up tests not finding significant differences between groups in Fear we could not conduct a mediation analysis for the fear clips specifically. However, when running a regression predicting affective empathy for Fear based on CU traits and controlling for ADHD score (because of the significant difference between groups), CU significantly negatively predicted affective empathy for Fear ($\beta = -0.33$, $t = -2.1$, $p < .05$). However, the difference between groups in affective empathy for Happy was not mediated by CU traits (see Figure 3.3, Panel B) with the estimate of the indirect effect bootstrap confidence intervals including zero ($\beta = 0.05$, 95% CI [-.08, .20]).

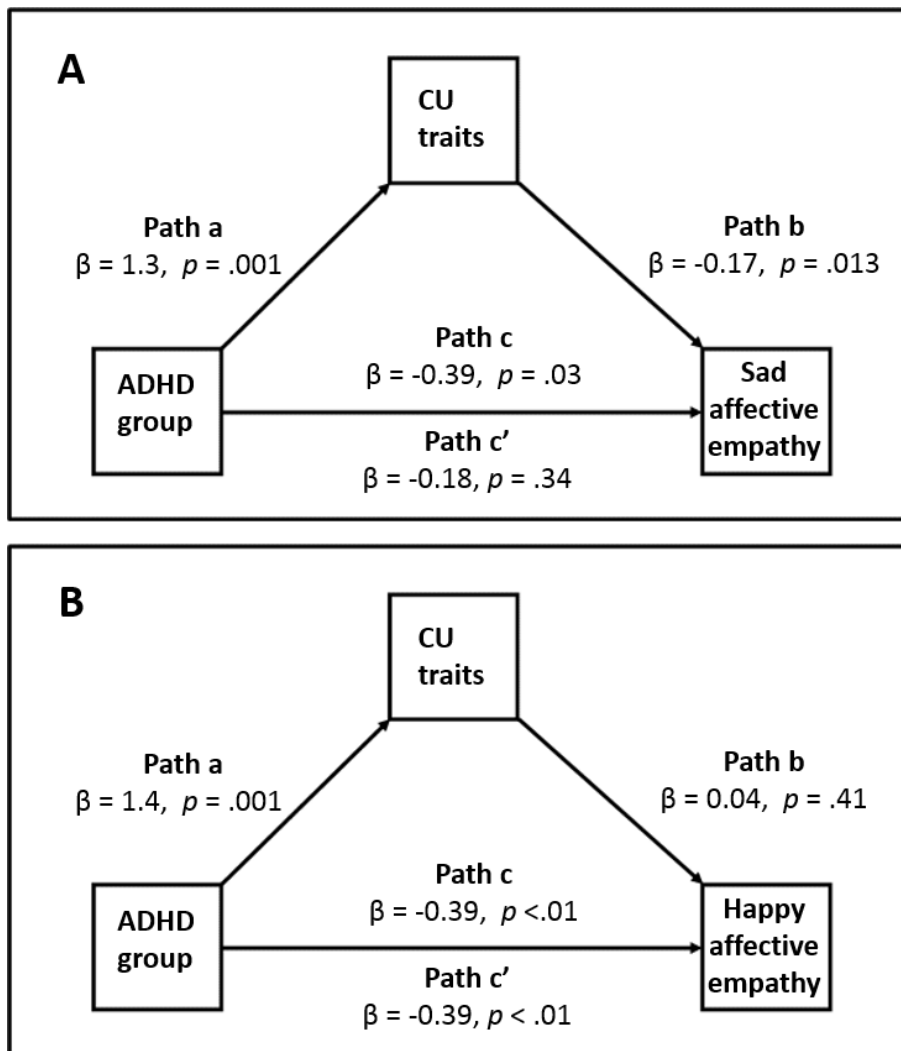


Figure 3.3. Mediation of ADHD group difference in Sad (Panel A) and Happy (Panel B) affective empathy through CU traits. β represents standardised coefficients. Path c (total effect) depicts the relationship between ADHD group and empathy without accounting for specific effect of the mediator (indirect effect), while Path c' (direct effect) depicts the relationship between ADHD group and empathy accounting for the specific effect of the mediator (indirect effect).

In order to further explore differences between groups in affective empathy for Happy we examined the moderation effect of ADHD severity. While controlling for CU traits, we found that the relationship between CD severity and affective empathy for Happy was moderated by ADHD severity as indicated by a significant interaction between CD and ADHD severity ($\beta = 0.06, p = .03, 95\% \text{ CI } [.005, .11]$). A significant negative relationship between CD severity and affective empathy for

Happy was only found in those low ($\beta = -0.73, p < .001, 95\% \text{ CI } [-1.1, -.37]$) and at average levels ($\beta = -0.47, p < .001, 95\% \text{ CI } [-.72, -.22]$) of ADHD severity, but not in those high in ADHD severity ($\beta = -0.21, p = .22, 95\% \text{ CI } [-.54, .13]$; see Figure 3.4).

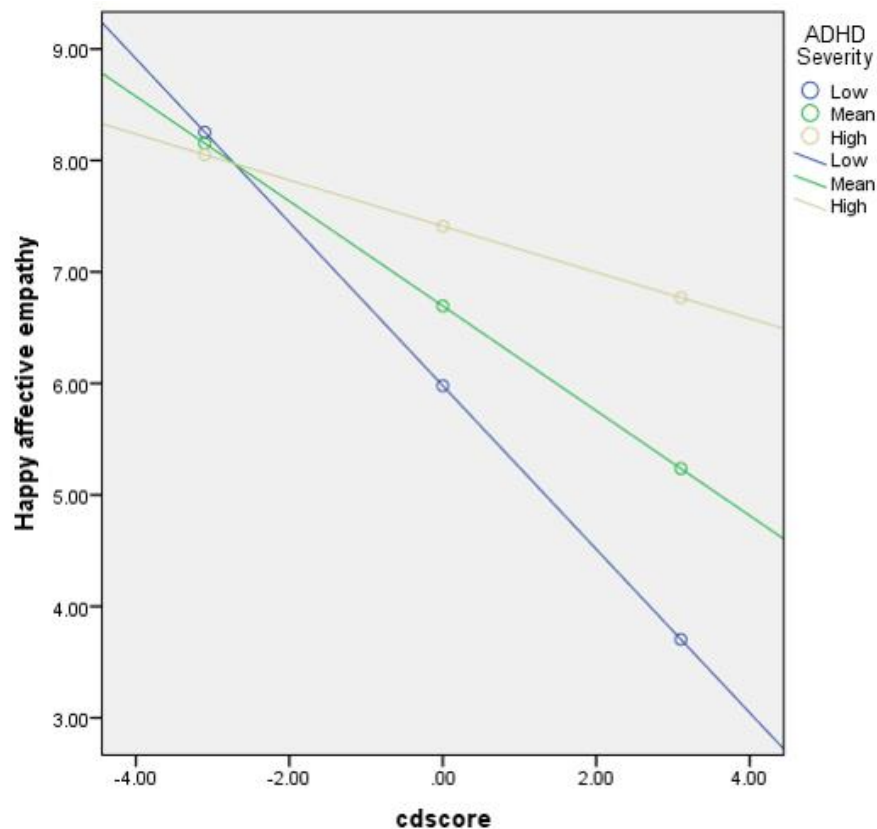


Figure 3.4. The moderating effect of ADHD severity on the relationship between CD severity and Happy affective empathy

3.3.3.2. The relationship between clinical characteristics, eye-gaze and empathy impairments

As with clinical characteristics, the relationship between eye-gaze and Happy and Sad empathy scores were explored with correlational analyses (see Table 3.3 and Table 3.4, respectively). Affective empathy for Happy was not related to eye preference. However, we did find that CD severity was negatively related to Happy eye preference (even after controlling for ADHD severity; $\beta = -0.35, p = .038$), and

Happy eye-preference was positively correlated with Happy cognitive empathy.

However, the negative correlation between CD severity and Happy cognitive empathy only approached significance ($p = .055$).

Table 3.3. Correlation matrix between Happy affective and cognitive empathy, eye gaze and clinical characteristics.

	1	2	3	4	5
1. Happy 2 Eye Preference	-				
2. Happy 2 Affective Empathy	.23	-			
3. Happy 2 cognitive empathy	.39*	.13	-		
4. CD score	-.39*	-.31 [#]	-.31 [#]	-	
5. CU traits	-.20	.05	.09	.42**	-
6. ADHD score	-.21	-.19	-.21	.34*	.34*

* $p < .05$. [#] $p < .06$. Correlations are Pearson's r

Table 3.4. Correlation matrix between sad affective and cognitive empathy, eye gaze and clinical characteristics

	1	2	3	4	5
1. Sad 1 Eye Preference	-				
2. Sad 1 Affective Empathy	.27	-			
3. Sad 1 cognitive empathy	-.03	.03	-		
4. CD score	-.24	-.23	-.05	-	
5. CU traits	-.28	-.52**	-.09	.49**	-
6. ADHD score	-.40**	-.06	-.06	.27	.30 [#]

** $p < .01$. [#] $p = .061$
Correlations are Pearson's r

When conducting a mediation analysis the inclusion of Happy eye preference did reduce the strength of the direct effect between CD severity and Happy cognitive empathy (see Figure 3.5), but the indirect effect included zero ($\beta = 0.04$, 95% CI [- .12, .0009]).

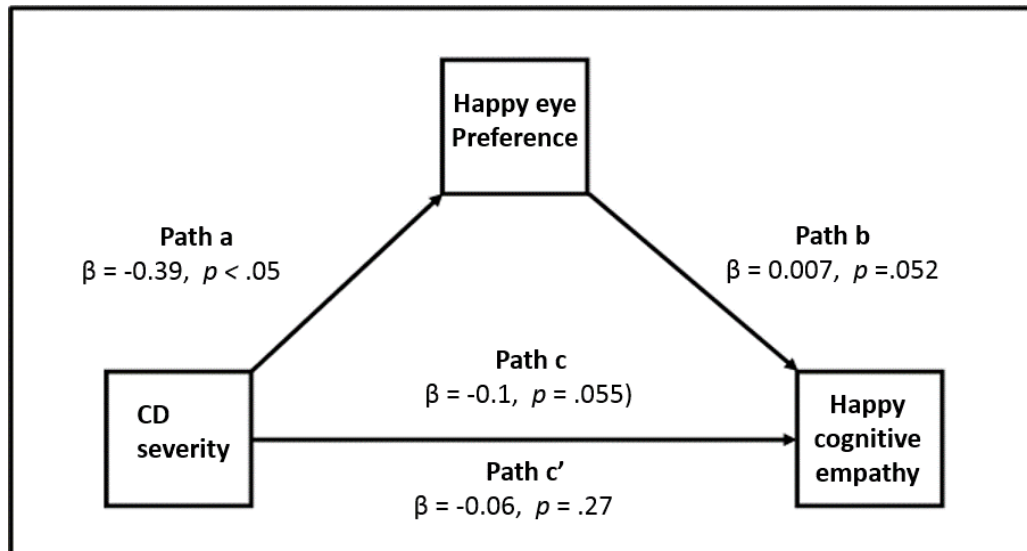


Figure 3.5. Mediation of the relationship between CD severity and Happy cognitive empathy through happy eye preference. Path c (total effect) depicts the relationship between CD severity and empathy without accounting for specific effect of the mediator (indirect effect), while Path c' (direct effect) depicts the relationship between CD severity and empathy accounting for the specific effect of the mediator (indirect effect).

Affective empathy for Sad was significantly negatively correlated with CU traits while ADHD severity was significantly negatively correlated with preference to look towards the eyes for sad faces.

Due to the fact that ADHD severity was found to be significantly correlated with preference for the eyes on sad clips and due to the fact that groups significantly differed in ADHD severity, we ran simple linear regression analysis in order to explore whether ADHD severity contributed to the group difference in preference to look towards the eyes. Group significantly predicted preference towards the eyes for the Sad clip ($\beta = -0.31$, $p = .041$) replicating the difference illustrated in Figure 3.2,

such that ADHD+CD participants looked towards the eyes less than ADHD alone. When adding ADHD severity in block two, we found that ADHD severity accounted for the difference between groups. There was a significant negative relationship between ADHD severity and Sad eye preference ($\beta = -0.35, p = .021$) while the effect of Group on eye preference was reduced to non-significance ($\beta = -0.19, p = .20$) with the model being significant overall ($R^2 = .19, R^2 \text{ change} = .12, F(2,42) = 4.8, p = .014$).

Further, due to the earlier found influence of CU traits in affective empathy for Sad, we then explored whether CU traits accounted for the relationship between ADHD severity and Sad eye preference. Although when entered alone there was a trend towards significance for CU predicting eye preference ($\beta = -0.30, p = .056$); when entering ADHD severity into the second block, ADHD severity was the only significant predictor of eye preference ($\beta = -3.9, p = .021$) and CU traits no longer predicted it ($\beta = -0.19, p = .21$) with the model overall significant ($R^2 = .17, R^2 \text{ Change} = .17, F(2,39) = 5.1, p = .01$). Finally, we explored whether Sad eye preference predicted affective empathy for Sad when also including CU traits and ADHD severity in a model. We entered CU in block one and then added ADHD severity and Sad eye preference in the second block. As expected, CU traits significantly predicted affective empathy for Sad ($\beta = -.52, p = .001$) while the addition of ADHD severity ($\beta = .08, p = .63$) or Sad eye preference ($\beta = .11, p = .49$) to the model did not explain any more variance in affective empathy for Sad ($R^2 \text{ Change} = .011, F(2, 36) = .27, p = .76$).

3.4. Discussion

The current study examined empathy and social attention in response to emotional film clips in a sample of adolescents with a diagnosis of ADHD with or without

comorbid CD. We found a general deficit in affective but not cognitive empathy in those with ADHD and comorbid CD compared to those with ADHD alone as opposed to a specific deficit in affective empathy for negative emotions. This general deficit in affective empathy for all emotions (Happy, Sad, and Fear) is in contrast to the findings of Chapter 2 where emotion recognition impairments were only apparent in those with ADHD+CD for fear and neutral faces. The findings of the current chapter are in contrast those of Cohen and Strayer (1996) who found both cognitive and affective empathy impairments in those with CD. However, both our groups had a diagnosis of ADHD therefore it may have been the case that both groups showed impaired cognitive empathy but that those with CD had additional affective impairments, but due to the lack of a typically developing control group we can only speculate that this is the case. Impairments specifically in affective empathy are partially in line with van Goozen et al., (2016), who found that impairments in affective empathy for fear within CD, mediated the link between genetic risk and aggressive behaviour. In relation to ADHD, our findings suggest that reduced empathy in those with ADHD in past studies (Braaton & Rosen 2000; Dyck et al., 2001) may be attributable to associated conduct problems as was the case in Marton et al., (2009).

In Chapter 2 we found that emotion recognition impairments were more strongly related to CD severity than CU traits, however in the current chapter, as predicted, we found that Sad and Fear affective impairments in ADHD+CD were explained by the presence of a higher numbers of CU traits which supports studies claiming that affective empathy impairments but intact cognitive empathy are related to psychopathic traits (Blair, 2005).

Suppressor effects have been found when examining the responsiveness of

brain regions thought to be involved in emotional processing when taking these traits into account. For example, Sebastian, Mccrory, Cecil, & Lockwood, (2017) found that the unique variance associated with conduct problems was positively associated with amygdala responsiveness, whereas the unique variances associated with CU traits was negatively associated with amygdala activity. In addition, Frick and Viding (2009) and Dadds et al., (2006) both found that CD without additional CU traits was associated with an exaggerated affective response to perceived social threat rather than the attenuated one expected in those high in CU traits.

Interestingly, the lower affective empathy in response to the happy clips in the ADHD+CD group was not explained via CU traits. However, we found the relation between CD severity and affective empathy for happy was moderated by ADHD severity such that there was only a negative relationship in those with less severe ADHD. It is possible that CD with low ADHD severity may have a different aetiology than CD with high ADHD severity. One could speculate that ADHD symptoms are driving the associated conduct problems in those high in ADHD severity but for those with lower ADHD severity there are different factors at play (other than the additional presence of CU traits since these were not related to affective empathy for happy). Although not considered in this study, perhaps those with CD and less severe ADHD come from a particularly disadvantaged background where there is less opportunity for happiness, making it more difficult to learn how to share in the happiness of others. Impairments in affective empathy for happiness may be important in explaining antisocial behaviour. The appropriate recognition of and capacity to experience happiness could be helpful in the regulation of more negative emotions. Indeed, Penton-Voak et al., (2013) found that improving the recognition of happiness reduced angry and aggressive behaviours highlighting the

need to explore specific impairments and implement targeted interventions. Due to all our participants having a clinical diagnosis of ADHD, future studies should seek to also include a group of CD without associated ADHD to test this further.

While in Chapter 2 there was no difference between ADHD alone and ADHD+CD groups in proportion of time spent looking at the eyes, in relation to our second aim of the current chapter, we found that ADHD+CD participants had less of a preference to look towards the eyes in response to sad and happy clips compared to ADHD alone. However, when performing regression analyses it was found that ADHD severity predicted this difference in eye-looking behaviour in response to said videos and not CD severity or number of CU traits. This relationship is in line with the findings of Chapter 2 where proportion of time spent looking at the eyes was significantly negatively correlated with ADHD severity. On the other hand, when examining happy eye preference in the current chapter, we found that this was negatively related to CD severity and not ADHD. We found no evidence eye looking behaviour was related to affective empathy (although we did find a trend in the relationship between eye preference and happy cognitive empathy). CU traits strongly predicted affective empathy for Sad even when controlling for eye looking behaviour, while ADHD severity seemed to be a strong predictor of eye-preference during the sad clips even when considering for CU traits. Our findings go against the hypothesis of Bons et al., (2013) who proposed that lack of attention to the eye region of the face results in empathy impairments in those with CD and those of Cowan et al., (2014) who found a positive relationship between trait empathy and looking towards the eyes during an emotional clip. We measured state as opposed to trait empathy in our study. It is possible that these different operationalisations of empathy have separate relationships with eye preference. Our findings are also in

contrast to those in the emotion recognition literature which suggests that attention towards the eyes is important for emotion recognition, particularly for fear expressions (Dadds et al., 2008). We used dynamic emotional situations rather than static facial expressions and therefore included additional vocal, gestural and contextual information. It may have been attention towards the eyes was rendered less important due to other cues of emotionality being available. However, the increased ecological validity of the paradigm should not be taken as a limitation. The fact that impairments in eye looking behaviour were explained via ADHD severity rather than CU traits is in contrast to Kimonis et al., (2006) who found that children high in CU traits exhibited less attentional orientating to distressing pictorial stimuli. Due to the relation with ADHD, in future studies it would be useful to include a non-emotional attentional control test to explore whether individuals with ADHD have problems with attention to emotional stimuli over and above the usual inattention problems associated with ADHD. Our finding that CU traits were related to affective empathy for Sad while eye preference deficits were related to ADHD severity is consistent with a study that found differential associations between different behaviours associated with conduct problems. Waller, Hyde, Grabell, Alves, and Olson (2015) found CU traits and oppositional and ADHD behaviours were separable by confirmatory factor analysis. CU traits were found to be related to lower empathy while ADHD was associated with lower attentional focusing. This suggests that it is important to consider specific mechanisms that may be dysfunctional in each individual rather than treating problems as isolated disorders.

Despite finding no evidence for a relationship between eye preference and affective empathy, our study is cross sectional and therefore cannot determine if eye looking deficits are not involved at any point during empathy development. It is

possible that lack of attention to the eyes earlier in development prevents one from developing the capacity to affectively empathise. Indeed, Johnson and Griffin (2005) suggest that it is plausible that atypical face preferences early in infancy contribute to later impairments in socioemotional behaviour. Eye contact occurs during interactions between caregiver and child and forms a crucial component of infants early social communication, influencing the development of the social brain (Bedford et al., 2015). Early in typical development, social interaction (attention to faces/reciprocal smiling) facilitates bonding and learning from the caregiver during the protracted period of postnatal development (Csibra & Gergely, 2006; Morton & Johnson, 1991). In support of this view, Dadds et al., (2014) found that children between 4 and 8 years who were high in CU traits showed impaired eye contact with attachment figures. In order to explore this further, future longitudinal studies are needed to measure eye preference at a young age and conduct problems later in life.

There were a number of strengths to the current study. Assessing empathy in response to dynamic clips has greater ecological validity than questionnaire-based measures of empathy. Such videos more closely reflect the kind of every day situation in which empathic responses may be triggered (Karow & Connors, 2003). In addition, by including participants with a clinical diagnosis of ADHD splitting them into those with and without comorbid conduct problems, while also measuring CU traits and social attention we were able to better delineate which specific problems relate to one another. Nevertheless, our findings should be interpreted in light of the study's limitations.

Due to not having a typically developing control group (as was the case in Chapter 2), we cannot determine if groups had impairments in cognitive empathy, only that additional CD was associated with additional affective impairments. It may

be that both groups show cognitive empathy impairments compared to typically developing controls. Therefore, future studies should include a typically developing control group. In addition, due to drop out associated with different aspects of the experiment, a consistent group of the same participants could not be used at all stages of analyses, it is possible that this reduced our power to find certain effects. A further limitation in relation to the eye gaze measure is that empathy was assessed in relation to the entire clip while social attention was assessed during a small segment of the clip. Therefore, even though this section of the clip was judged via a pilot study to be the most emotionally intense, it is possible that all the affective information needed was already retrieved from the eyes earlier in the clip and therefore the eyes became less salient at the point of analysis. Future studies should explore if eye looking deficits are found depending on where in the clip the analysis segment is conducted. In addition, there is evidence to suggest that antisocial adults may have difficulties verbalising their personal thoughts and feelings (Quiggle, Garber, Panak, & Dodge, 1992); future research should therefore employ physiological measures to further examine affective empathy difficulties as was the case in van Zonneveld et al., (2017).

There are also aspects of empathy that our study did not explore. We did not assess motor empathy, an important component of empathy (van der Graaff et al., 2016). Information on motor empathy could confirm if affective empathy is the key component related to the antisocial behaviour associated with conduct problems. In addition, since motor empathy is supposed to precede affective empathy (Preston & de Waal, 2002), a study including motor empathy could help determine where the break down in empathy processing occurs. Further, our study only examined the capacity to experience empathy and not the responses which occur as a result of

empathy. It may be that there are certain individuals who do not experience affective empathy and yet have learnt appropriate ways of responding or have other protective factors present to prevent antisocial behaviour. Future studies should therefore assess participants' responses to a social situation following an empathy eliciting task.

Our findings add to the literature suggesting that there might be different pathways to conduct problems. Wootton, Frick, Shelton, and Silverthorn (1997) found that CU traits moderated the relationship between ineffective parenting and conduct problems. Ineffective parenting was only related to CD in those low in CU traits. We found that ADHD severity moderated the relationship between affective empathy and CD, while we found that the presence of CU traits only mediated the difference in affective empathy for Sad between groups. In addition, Miller et al., (2014) found that empathy impairments moderated the effects of parenting on conduct problems. Taken together these findings suggest that the heterogeneity of those with different problems interact resulting in varied presentations. This further highlights the need to look at problems from a dimensional perspective as advocated by the research domain criteria approach to mental health problems (NIMH; Insel et al., 2010) as opposed to considering problems as isolated disorders. It also further supports the need for targeted interventions based on the assessment of mechanisms leading to such problems. Interventions focusing on enhancing emotional awareness have shown promise in reducing aggressive behaviour in clinical (van Baardewijk, Stegge, Bushman, & Vermeiren, 2009) and young offender populations (Hubble et al., 2015). While there is also evidence that empathy and compassion training results in increased affective responses and functional activity in brain areas involved in emotion processing (Klimecki, Leiberg, Ricard, & Singer, 2013). However, our finding that eye preference deficits are related to ADHD severity only, along with

the results of van Zonneveld et al., (2017) who found no difference in eye preference in those at risk of antisocial behaviour and controls suggests that interventions specifically changing where individuals look may only be helpful for those with additional ADHD symptoms.

In conclusion, the present chapter indicates that adolescents with ADHD+CD have more problems with affective empathy than those with ADHD alone, and that the impairments for sad and fear empathy seem to be driven by an increased number of CU traits. We did not find evidence that a lack of attention to the eye region was associated with empathy impairments although a lack of attention was related to ADHD severity. Our study highlights the need to consider the heterogeneous nature of ADHD and conduct problems and assign interventions based on specific impairments as opposed to the blanket approaches used in the past.

4. Chapter 4: Emotion Regulation

Paper in preparation

This chapter is based on Airdrie, J. N., Langley, K., Thapar, A., van Goozen, S. H. M. (2017). Emotion regulation impairments in adolescents with Attention-Deficit/Hyperactivity Disorder and Conduct disorder: A novel emotional Stroop paradigm. *In prep*

4.1. Introduction

Chapters 2 and 3, in emotion recognition and cognitive and affective empathy, explored aspects of emotion processing that are somewhat conscious. Next, Chapter 4 will examine a more automatic aspect of emotion processing, namely emotion regulation, using a novel pictorial emotional Stroop task.

The role emotions play in our lives has long been debated. Early arguments were contradictory, with some highlighting the value that emotions have in functioning and responding (Hume, 1739/1978), while others viewed emotions as distractors, interfering with our rationality and reason, and in need of strict control (Ryle, 1949). Nevertheless, more recent theorising highlights the important functions that emotions serve in areas such as decision-making (Lerner, Li, Valdesolo, & Kassam, 2015), preparing the response system for action (Ekman, 1992; Frijda, 1986) and informing us of the mismatch between our current state and a desired goal state (Bagozzi, Baumgartner, Pieters, & Zeelenberg, 2000). Arguably the most important functions of emotions are in the social realm, portraying both our own intentions and informing us of the behavioural intentions of others (Fridlund, 2014). Nevertheless, emotions have the potential to be harmful or maladaptive. They can be of the wrong duration or intensity, or incompatible with the current situation, leading to biases in our cognition and behaviour (Gross, 2015). Such examples highlight the necessity of regulatory processes to ensure emotions are adaptive.

Emotion regulation (ER) has been defined as “the processes by which individuals influence which emotions they have, when they have them, and how they

experience or express these emotions” (Gross, 1998, p. 275), while it also encompasses “an individual’s ability to modify an emotional state so as to promote adaptive, goal-orientated behaviours” (Shaw et al., 2014, p. 276). Emotion regulation includes both top-down and bottom-up influences, and can be conscious or unconscious (Gross, 1998). An example of conscious emotion regulation would include being motivated to attenuate an angry response in order to avoid harshly disciplining a child for something which they did on accident, or suppressing an inappropriate laugh on a sombre occasion, while an example of unconscious emotion regulation could be hastily turning away from upsetting material (Gyurak, Gross, & Etkin, 2011).

Effective ER is vital for healthy functioning. This is highlighted by the number of disorders in which maladaptive ER (or emotion dysregulation) is implicated. Emotion dysregulation occurs when ER processes are impaired, resulting in behaviour that is maladaptive to that individual. This could include rapid and poorly controlled shifts in emotion, emotional expressions and experiences which are out of proportion for the context or are against social norms, and the irregular allocation of attention to emotional stimuli (Shaw et al., 2014). Gross and Levenson (1997) highlight how emotion dysregulation is implicated in over half of DSM-IV Axis I and in all of Axis II disorders. For example, difficulty regulating sadness is associated with depression whereas problems regulating anxiety (or fear) are associated with anxiety disorders (e.g. Caspi, Henry, McGee, Moffitt, & Silva, 1995). Deficits in emotion regulation have been associated with a number of other impairments such as reduced prosocial behaviour (Blair et al., 2004), academic success (Graziano, Reavis, Keane, & Clakins, 2007), social competence (Denham, Way, Kalb, Warren-Khot, & Bassett, 2013) and quality of social relationships

(Lopes, Salovey, Côté, & Beers, 2005).

A disorder where emotion dysregulation is seen as particularly important is ADHD. A recent meta-analysis (Shaw et al., 2014) revealed prevalence estimates of emotion dysregulation in those with ADHD of between 24% and 50% in clinic based studies. Adolescents with ADHD are proposed to show problems in emotion regulation due to their reduced capacity for inhibition making it difficult for them to withhold a response for long enough to gather the information necessary for understanding emotionally charged situations (Barkley, 1997). Those with emotion regulation impairments in combination with ADHD have a worse clinical prognosis (Biederman et al., 2012) while they also show more difficulties in peer and family relationships, and occupational and academic achievement compared to those with ADHD alone (Wehmeier, Schacht, & Barkley, 2010). Despite the prevalence of emotion dysregulation being high in ADHD, a recent review conducted by Shaw et al., (2014) suggests that it is still unclear if emotion dysregulation and ADHD are merely correlated, whether emotion dysregulation is a core feature of ADHD, or whether the presence of both ADHD and emotion dysregulation represents a distinct nosological entity. Importantly, the review by Shaw et al., (2014) could not conclude if there was stronger evidence for either of these relationships between ADHD and emotion regulation, while the problem is further complicated by a key a prevalent comorbidity of ADHD, conduct disorder (CD).

Emotion dysregulation theory has also been used to explain antisocial behaviour in individuals with CD (Cole et al., 1994). Aggressive behaviours have been argued to reflect abnormalities in neurobiological circuits (including the orbito-frontal cortex, ventromedial prefrontal cortex, dorsolateral prefrontal cortex, amygdala and anterior cingulate cortex) which are involved in both the top-down

regulation of negative emotions and the bottom-up processing of environmental cues that ordinarily produce emotional restraint (Davidson et al., 2000; Frick et al., 2003). Numerous studies have found impairments in emotion regulation in adolescents with CD compared to control groups (Calkins & Dedmon, 2000; Deborde et al., 2015; Kostiuk & Fouts, 2002; McLaughlin et al., 2011).

Given the overlap between CD and ADHD, it is still unclear if emotion regulation difficulties are associated with a diagnosis of ADHD, or are only found in those with ADHD who have additional conduct problems. For example, if the combination of ADHD and emotional dysregulation represents a distinct nosological entity, which was one of three models suggested by Shaw et al., (2014), it may be that the co-occurrence of ADHD and CD explains such a relationship. There is evidence to suggest that when both ADHD and CD are considered together, emotion regulation impairments are specific to additional CD (Maedgen & Carlson, 2000; Melnick & Hinshaw, 2000). For example, in a study by Northover, Thapar, Langley, and van Goozen (2015a) that included participants with an ADHD diagnosis who were divided into those with or without additional CD, it was found that those with additional CD showed significantly more difficulties in emotion regulation. Similarly, Schoorl, van Rijn, de Wied, van Goozen, and Swaab (2016), in a study of those with a diagnosis of CD (and not ADHD), found that the CD group showed increased emotion dysregulation compared to controls, and that these deficits were not associated with inattention, a core feature of ADHD. In addition, Hinshaw and Melnick (1995) when comparing those with ADHD and high aggression to those with ADHD and low aggression, it was found that those with high aggression displayed increased emotional reactivity and reduced emotion regulation abilities.

However, other studies have reported on a specific link between emotion

regulation difficulties and ADHD symptoms even when controlling for additional conduct problems (Bunford, Evans, Becker, & Langberg, 2014; Seymour, Chronis-Tuscano, Iwamoto, Kurdziel, & MacPherson, 2014; Villemonteix, Purper-Ouakil, & Romo, 2015).

In addition to the lack of consistency above, much research exploring emotion regulation difficulties is reliant on self- or (more often than not) informant-reported problems in emotion regulation (Anastopoulos et al., 2012; Sobanski et al., 2010; Strine et al., 2006; Bunford et al., 2015; Seymour et al., 2014). Such methodology has long been suggested to lack reliability and validity (Nisbett & Wilson, 1977), especially in judgements that are made ‘offline’, at a time after an emotional situation may have occurred (Robinson & Clore, 2002).

Further, the more recent studies above that explored emotion regulation in CD and ADHD (Northover et al., 2015a; Schoorl et al., 2016), did so using the ultimatum game (UG) paradigm. Primarily, this is an economic decision making game. The game involves a proposer (in the case of the above studies this was a computer program with pre-set responses) who must choose how a 10 point reward must be shared between themselves and another (the participant in the study). The proposer’s offer can be fair (e.g. 5 points each) or very unfair (9 points allocated to the proposer and 1 point to the participant) or fall between those ends of the scale. The job of the participant is to choose whether to accept or reject this offer. If the participant accepts, the share of points suggested by the proposer is received, while if the participant rejects neither player receives any points. As a result of this, the rational response to all offers is to accept the proposal. Therefore, any rejection of an offer is considered a failure of emotion regulation. In the above two studies (Northover et al., 2015a; Schoorl et a., 2016), those with CD were more likely to

reject more moderately unfair offers than those without CD, and this was taken as evidence of more impairments in emotion regulation. However, there are problems with the validity of this paradigm as a measure of emotion regulation.

Firstly, although the rejection of unfair offers is considered irrational because it results in personal loss, from a social perspective, the rejection of unfair offers can be seen as a rational, altruistic action to preserve social norms. Rather than maximising self-interest, the participant chooses to punish the socially inappropriate action, and therefore the decision to reject an offer is not necessarily a result of a failure in regulation (Fehr & Fischbacher, 2003; Knack, Pascual-Leone, Meyer, Treyer, & Fehr, 2006). This is supported by the fact that similar rates of rejection are found in studies in which participants play on behalf of a third party (Civai, Corradi-Dell'Acqu, Gamer, & Rumiati, 2010). Secondly, it is assumed that the proposition of unfair offers will result in an emotional response in participants that is in need of regulation; however, no physiological measurements were taken in the above studies, so it remains unclear whether participants experienced an emotional reaction when confronted with the proposition.

4.1.1. A Need for a New Emotion Regulation Paradigm

As a result of the previous reliance on questionnaire-based studies to measure emotion regulation and the questionable validity of the UG as a measure of emotion regulation, the current study (experiment 1) sought to use an objective, experimental measure of emotion regulation in order to acquire a more valid picture of its role in clinical disorders.

Attentional deployment is suggested by Gross (1998) to be central to emotion regulation. It includes concepts such as distraction, where an individual may focus on a non-emotional aspect of a situation to decrease negative affect (Nix, Watson,

Pyszczynki, & Greenberg, 1995). The emotional Stroop task has been suggested to be a valid measure of the attentional aspect of emotion regulation because it incorporates an affective dimension into a cognitive paradigm (Shaw et al., 2014). Emotional Stroop tasks typically consist of words (both emotional and neutral) presented in different colours, with the aim of the task to identify the colour of the text. The common finding is that participants are slower to respond when the word is emotional compared to neutral, particularly in clinical populations where the emotional word relates to their disorder (Buhle, Wager, & Smith, 2010), reflecting an attentional bias to emotional stimuli which interferes with performance of the current goal. However, the presentation of words is not representative of the scenarios individuals with ADHD and CD will be faced with in everyday life. We therefore sought to use more ecologically valid stimuli in a new emotional Stroop task.

It is now generally accepted that pictorial emotional stimuli capture attention during concurrent task performance and activate key neural structures including the amygdala (see Carretié, 2014, for a review). It is thought that because attentional capture by emotional stimuli happens so rapidly and automatically they reflect attentional processes at an involuntary, pre-conscious level. Ashwin, Wheelwright, and Baron-Cohen (2006) made use of a pictorial emotional Stroop task that presented emotional faces as distractors to explore interference in individuals with Autism Spectrum Disorder (ASD) and controls. This paradigm presented participants with photographs of angry faces, neutral faces and chairs, which were overlaid with a transparent film of one of three colours. Participants were required to ignore the content of the picture and name as quickly as possible the colour. It was found that children with ASD showed increased reaction time latencies to facial

stimuli compared to objects, while typical controls were slower to respond to angry faces compared to both neutral faces and chairs, which suggests emotional interference by the stimuli. Ashwin et al., (2006) argued that interference represents emotional capture by the stimuli, while facilitation (a faster response in the presence of the emotional stimuli) would be a reflection of participants disengaging from the content of the picture to be better able to respond to the colour. We conducted our own pilot study in order to replicate the findings of Ashwin et al., (2006) in a sample of undergraduates, finding that participants were faster to name colours in the presence of an emotional face distractor compared to a neutral face and an object (chair) distractor.

While the above paradigm shows promise as a measure of the attentional aspect of emotion regulation, pictorial Stroop tasks which rely on colour naming have been criticised for the relationship between the target (colour) and distractor (emotional face) being arbitrary (Buhle et al., 2010) and as a result typically have small effect sizes (Phaf & Kan, 2007). Due to the arbitrary nature of the relationship between colours and emotions, one cannot conclude from the pilot mentioned above whether faster responses when the distractor was an emotional face represents facilitation by emotional stimuli or that the neutral images were distracting and therefore causing interference. Inferences from this paradigm are only made on the basis of responding to one type of trial being faster than another and therefore cannot be named facilitation or interference. Stroop tasks with true interference require both congruent and incongruent trials. Congruent trials are those for which the irrelevant aspect of the task facilitates responding to the target due to a semantic match, while incongruent trials are those in which the irrelevant aspect of the task interferes with responding to the target due to semantic mismatch. Stroop tasks that include this true

Stroop interference typically show robust interference effects (e.g. Anes & Kruer, 2004; Egner, Etkin, Gale, & Hirsch, 2008).

As a result of the above, we conducted a second pilot study, again on university students. This time presenting the distractor stimuli, emotional faces of different valences (happy and fearful) and an object (photographs of houses) with target stimuli (symbols) that were congruent or incongruent (Ticks and Crosses) with the emotional distractors but shared no semantic connection with the house. The symbols were presented around the four corners of the distractor image and the distractor and target images were presented simultaneously.

Using this paradigm there were no significant differences in reaction time to the different distractor conditions. However, problems with this paradigm included the fact that the presentation of the symbols were at the four corners of the distractor image while we also presented both the target and distractor simultaneously. Therefore, participants could adopt a strategy whereby they never gave any attention to the features of the distractor image due to the symbols not appearing near the key features of the distractor images, nor the distractor image appearing on its own first. In addition, by having two emotional face conditions but only one object condition, faces appeared at twice the rate of objects, and therefore this may have impacted on results.

In addition to the problems identified with the previous pilots, Sebastian, Mccrory, De Brito, & Viding (2017) have found that amygdala responses to task irrelevant fearful faces were attenuated under high difficulty compared to low difficulty conditions. This highlights the importance of making the target aspect of the Stroop task relatively simple, and having all stimuli equally difficult, as the above study shows that unbalanced difficulties result in differential processing of

emotional stimuli.

Experiment 1 therefore aimed to explore performance on a novel pictorial emotional Stroop task (Cardiff Emotional Pictorial Stroop; CEPS) which had been adapted to deal with the problems identified with previous Stroops in the literature and those from earlier piloting. It was important for the current Stroop to meet the following requirements: (1) it produces a significant congruency effect (reaction times to congruent trials are faster than to incongruent trials), and (2) it produces no difference in reaction time to target stimuli combined across all distractor stimuli. In order to do this undergraduate students completed a reaction time based emotional Stroop task (CEPS) which presented emotional faces (happy and fearful) and neutral objects (feature matched images of houses and flowers) as distractor images and symbols (ticks or crosses) as target stimuli which were presented over the features (eyes or mouth areas) of the distractor stimuli. Participants were told to ignore the distractor stimuli and to respond verbally to the target (see Figure 4.1 for a depiction of a single trial of the task). The pairing of emotional faces and symbols created the congruency conditions such that a happy face paired with a tick was a congruent trial, while a happy face paired with a cross was an incongruent trial, and vice versa for fearful distractor images.

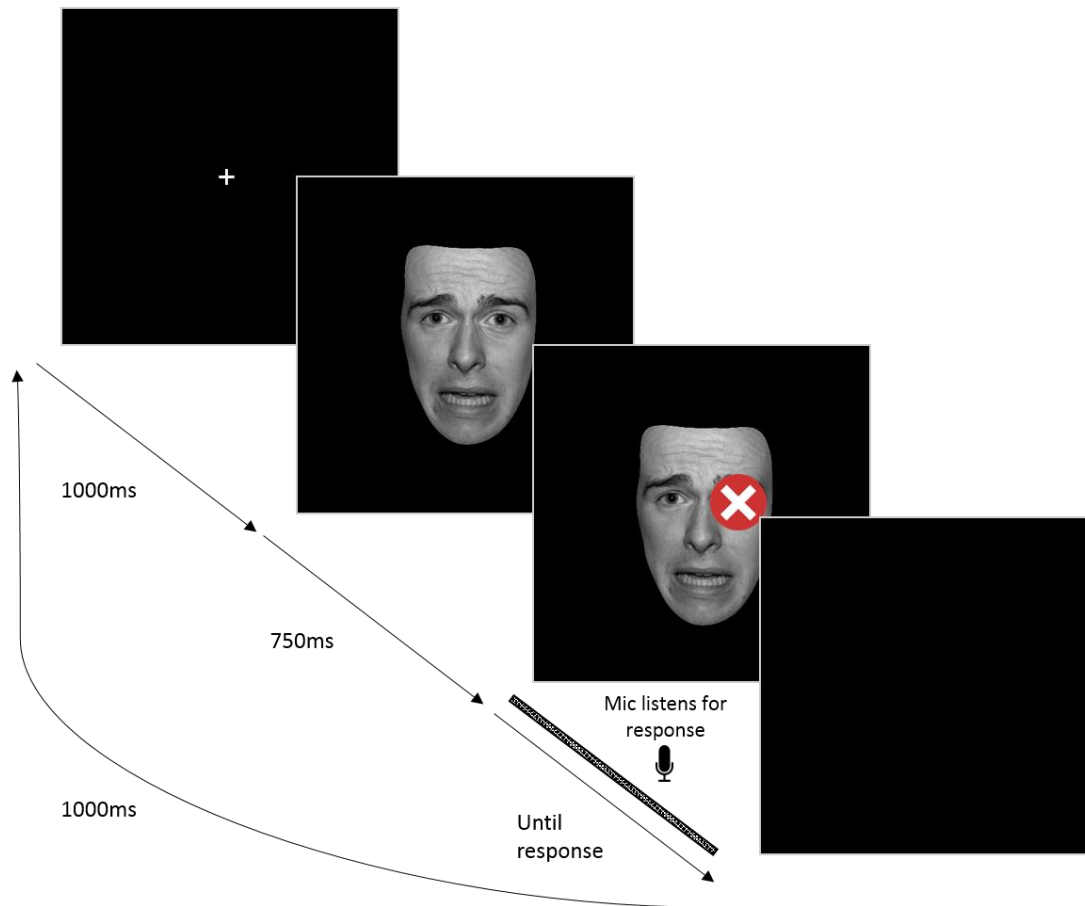


Figure 4.1. Emotional Pictorial Stroop trial depiction.

It was hypothesised that reaction time would not differ when responding to the ticks and crosses overall (therefore signifying that each of the target stimuli were equally difficult/easy to respond to). In addition, it was predicted that participants would have faster response times to congruent trials compared to incongruent trials. Further, given research showing that the eyes are an attentional grabbing feature of the face (Bassili, 1979), we explored whether the congruency effect was more pronounced when targets were presented over the eyes compared to the mouth. In addition, we explored the emotional interference and facilitation effects by comparing congruent and incongruent emotional trials to those of the neutral objects. We predicted that congruent emotional trials (e.g. fear/cross) would be responded to

faster than object trials, while we also predicted that incongruent emotional trials (e.g. fear/tick) would be responded to slower than object trials.

4.2. Method

4.2.1. Participants

A sample of 50 undergraduate students (42 females, 8 males) between the ages of 18 and 40 ($M = 20.0$, $SD = 3.4$) years from the Psychology degree programme at Cardiff University took part in the study. Participants volunteered to take part in the study via online sign up in return for a £5 voucher. All participants had normal or corrected to normal vision. Participants were excluded if English was not their first language.

4.2.2. Materials and Measures

4.2.2.1. Emotional Stroop task.

4.2.2.1.1. Stimuli.

The task involved two types of stimuli: distractor and target stimuli. The distractor stimuli consisted of 12 human faces (retrieved from the NimStim database; Tottenham, Borscheid, Ellertsen, Marcus, & Nelson, 2002), six of which portrayed fearful emotional expressions, and six portrayed happy emotional expressions (half of each emotion were female faces and the other half were male). Neutral objects, six houses and six flowers (created using CorelDraw), were also used as distractor images and these were made to have the same number of features as the faces (see Figure 4.2, Panel A).

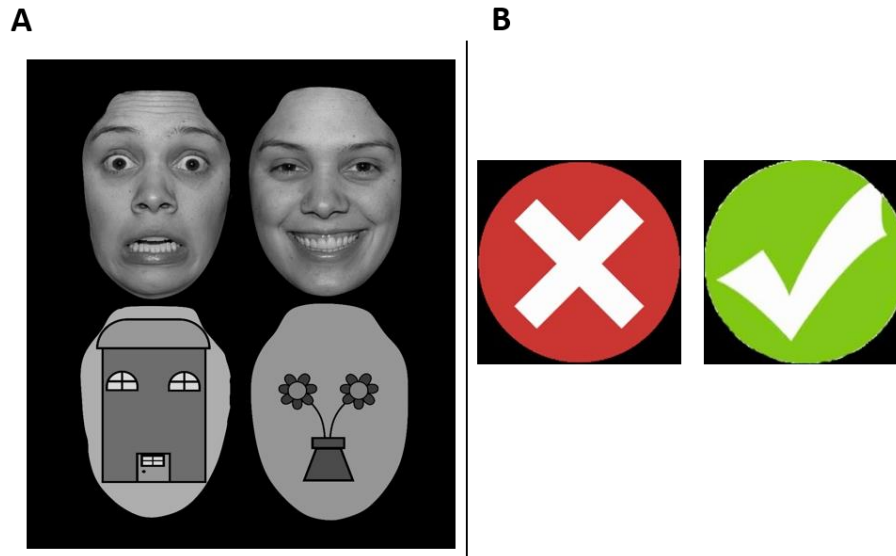


Figure 4.2. Panel A: Distractor stimuli examples. Panel B: Target stimuli examples. Image sizes are not to scale

The houses had two windows in same locations as the eyes were positioned on the faces, while there was a door in the same region as the mouth. For the flowers, there were two flowers at the location of the eyes and a plant pot at the same location as the mouth. The houses and flowers were positioned on blank cut-outs (silhouettes) of the face stimuli in order to control for the overall size and shape of the stimuli.

The target stimuli consisted of a white tick with a green circular background and a white cross with a red circular background (see Figure 4.2, Panel B).

4.2.2.1.2. Presentation and data recording equipment.

The experimental paradigm was programmed using Matlab in conjunction with the open source add-on, Psychtoolbox (Kleiner et al., 2007) and presented on a 13” Toshiba Portege laptop. The background of the screen was always black and the fixation cross presented in white. A free Psychtoolbox function, SimpleVoiceTriggerDemo was adapted to record reaction time. The function works by continually monitoring a microphone’s sound input level (a Samson Go mic™ was used) to the computer. When the sound level surpasses a pre-defined threshold,

a trigger is sent, which was used to stop a stopwatch. The experimenter recorded the accuracy of the participant's verbal responses with a response sheet marking the relevant response that the participant made for each trial while also recording if the microphone failed to register the participants' response. Participants were positioned approximately 60cm from the laptop screen and 30cm away from the microphone.

4.2.2.1.3. Experimental procedure.

A pictorial depiction of the CEPS paradigm is presented in Figure 4.1 (above). Each trial began by presenting a white fixation cross (presented against a black background) for 1000msec. Following this, one of the distractor stimuli (either a fear face, happy face, flower, or house) first appeared alone on the screen for 750msec (a delay between presentation of the distractor and the onset of the target symbol was used in order to deal with the issue identified in piloting whereby the simultaneous presentation of the distractor and target would mean the participant would never need to process the features of the distractor). Participants were to ignore this distractor image. Next, one of the two symbols (target stimuli; Tick or Cross) appeared on top of one of the distractor stimuli's three features (left or right eye, or mouth, or the neutral objects' equivalent feature). This remained on the screen until the participants' response. Participants responded verbally to the target stimuli. They were required to say "good" if a tick appeared, and to say "bad" if a cross appeared. The screen then was blank for a further 1000msec. until the beginning of the next trial. There were 96 trials, with each distractor stimulus being paired an equal number of times with the tick or cross, while the tick or cross appeared on each of the features an equal number of times. The order of trials was randomised for each participant. Participants were instructed that the task was a reaction time test, in which they were required to respond as fast as they could, whilst avoiding errors.

They were also told that they had to ignore the first image and only respond to the symbols.

Before the main Stroop task there were two practice blocks of 16 trials each; in the first practice only the distractor stimuli were presented and they were required to name the distractor stimuli (“house”, “flower”, “happy”, “fear”). In the second practice block only the target stimuli were presented and they had to name them (“good”, “bad”). The practice blocks familiarised the participants with the procedure, while also ensuring participants were able to recognise each of the distractor stimuli conditions. The main experimental task was split into three blocks (with an equal number of stimulus combinations presented in each) to prevent fatigue effects. At the end of each block, participants were given feedback about their average reaction time and the number of errors for the preceding block and encouraged to try to improve on the next block.

4.2.3. Data analysis

Four participants were excluded due to having a first language other than English, leaving 46 remaining participants.

4.2.3.1. Reaction time data processing.

For each participant, trials in which their reaction time was less than 250msecs or more than three standard deviations above their overall mean reaction time were excluded. Trials in which participants made an error and trials after error trials were to be excluded as reaction time on trials after an error has been committed have been found to be consistently slower than those following correct trials (Rabbitt, 1966). However, in the current experiment participants did not make any errors therefore it was not necessary to exclude trials after errors. Only a small percentage of trials were excluded. 0.3% of trials were excluded for being faster than 250ms and 0.9% of

trials were excluded for being more than 3 standard deviations above the mean.

There were no systematic differences between percentages of trials excluded between the different trial types.

Analyses were carried out using SPSS 20 (SPSS Inc., Chicago, Illinois). Reaction time analyses consisted of a number of planned comparisons. For analyses of the reaction time, differences in response time to target stimuli (ticks and crosses), and congruent and incongruent trials overall and when presented over specific regions, paired sample t-tests were used. In order to compare the eyes and the mouth the average reaction time across both the left and right eye location was calculated and then paired sample t-tests were used to compare conditions. To examine the effect of emotional congruency compared to objects, we used paired sample t-tests to compare individual emotion congruency conditions (i.e. happy and tick (congruent), happy and cross (incongruent), fear and cross (congruent), fear and tick (incongruent) to reaction time when an object distractor (with its corresponding matched target (e.g. fear tick would be compared to object distractors with tick targets)) was presented. Finally, we merged congruent and incongruent emotion trials in order to explore overall reaction time differences between objects and emotional faces.

4.3. Results

Overall reaction time to the ticks ($M = 457.6$, $SD = 45.8$) and crosses ($M = 451.9$, $SD = 43.3$) did not differ significantly ($t(45) = 1.64$, $p > .10$). Participants responded significantly faster to congruent trials ($M = 449.1$, $SD = 41.0$) than to incongruent trials ($M = 456.3$, $SD = 45.0$; $t(45) = -2.7$, $p < .01$, $d = 0.17$).

4.3.1. Congruency Effect at Eyes

We then explored whether the congruency effect differed according to whether the target stimuli appeared over the eyes or the mouth. When examining congruent and

incongruent trials at the eye region, participants were significantly faster to respond to congruent trials ($M = 446.3$, $SD = 43.4$) than to incongruent trials ($M = 453.0$, $SD = 45.8$; $t(45) = -2.1$, $p < .05$; $d = 0.15$).

4.3.2. Congruency Effect at Mouth

When exploring the reaction time to congruent and incongruent trials at the mouth region, participants were faster to respond to congruent trials ($M = 453.8$, $SD = 48.0$) than to incongruent trials ($M = 461.0$, $SD = 47.2$), but the difference was not significant; $t(45) = -1.3$, $p = .20$, $d = 0.15$).

4.3.3. Emotional Congruency Effects

4.3.3.1. Fear.

4.3.3.1.1. Congruent. Participants responded significantly faster to trials in which a fear face was paired with a cross (congruent; $M = 445.8$, $SD = 44.2$) than to an object paired with a cross ($M = 455.9$, $SD = 46.1$; $t(45) = -2.8$, $p < .01$, $d = .22$).

4.3.3.1.2. Incongruent. There was no difference between trials in which a fear face and tick were paired (incongruent; $M = 459.0$, $SD = 50.0$) and trials that paired an object with a tick ($M = 461.0$, $SD = 47.8$; $t(45) = -0.57$, $p = .57$, $d = 0.04$).

4.3.3.2. Happy.

4.3.3.2.1. Congruent. There was no difference between trials in which a happy face and tick were paired (congruent; $M = 459.4$, $SD = 51.1$) and trials presenting an object and tick ($M = 461.0$, $SD = 47.8$; $t(45) = -.39$, $p = .70$, $d = 0.03$).

4.3.3.2.2. Incongruent. Similarly, there was no difference between trials in which a happy face and cross were paired (incongruent; $M = 455.4$, $SD = 46.5$) and trials presenting an object and crosses ($M = 458.5$, $SD = 45.3$; $t(45) = 0.85$, $p = .40$, $d = 0.07$).

4.3.4. Emotional Interference Effects

When merging congruent and incongruent trials for fearful stimuli, there was an overall emotional facilitation effect such that responses were faster in the presence of fearful faces ($M = 452.4$, $SD = 44.3$) compared to objects ($M = 458.5$, $SD = 45.3$; $t(45) = -2.33$, $p = .024$; $d = 0.14$).

When merging the happy face data, there was no facilitation effect: responses were not faster in the presence of happy faces ($M = 457.3$, $SD = 46.5$) compared to objects ($M = 458.5$, $SD = 45.3$; $t(45) = -0.4$, $p = .69$, $d = 0.03$).

4.4. Experiment 1 Discussion

The aim of Experiment 1 was to create a pictorial emotional Stroop task in which responding to the target stimuli overall was of equal difficulty, while also creating a task which produced a true Stroop effect, such that responses would be significantly faster to congruent trials than to incongruent trials. In line with our hypotheses we found no differences in reaction time when responding to ticks and crosses overall. Further, we found that participants were significantly faster to respond to congruent than to incongruent trials. Interestingly, when examining the congruency effect when the target was presented over the eyes and mouth separately, there was only a significant difference in reaction time to the congruency conditions when the targets were presented over the eyes. However, we followed this up and found that congruency and location did not interact, therefore it was not the case that the congruency effect was significantly stronger at the eye than the mouth location. In addition, we compared the emotional congruency effect to object trials. The strongest effect was found for congruent fear stimuli, such that participants were

faster to respond to these trials than when an object was presented as a distractor, suggesting a strong facilitation effect. While we found no difference between incongruent fear trials and objects, and no difference between either congruent or incongruent happy trials and objects. This suggests that fear stimuli are particularly attention grabbing and facilitate responding. Importantly, when merging emotional distractors across congruency conditions, both emotions produced overall facilitation compared to object trials, this was significant for fear stimuli, but not for happy ones.

Having established that the newly developed CEPS can produce a significant congruency effect, while also employing target stimuli which were equally difficult to respond to, we next applied this task to a sample of ADHD participants with or without comorbid CD.

4.5. Experiment 2

As outlined above, there is inconsistency in the literature as to whether emotion regulation is a problem for ADHD in general, or a problem specifically for those with ADHD and a comorbid diagnosis of CD. Therefore, the primary aim of experiment two was to utilise the newly developed CEPS to explore whether automatic attentional processes of emotion regulation differ between these groups.

As stated earlier, in order to explore emotion processing in Stroop tasks, it is important that responses to the target stimuli do not significantly differ overall between groups (Sebastian et al., 2017). Therefore, in experiment 2, as well as replicating the results from experiment 1 in regards to a non-significant difference between reaction time to ticks and crosses, we also aimed for groups of participants not to differ in their reaction times to ticks and crosses overall across distractor conditions. The second aim of experiment 2 was to replicate the congruency effect in a clinical sample such that overall congruent trials resulted in faster responses than

incongruent trials. The central aim was to explore whether emotional interference (or facilitation) as a result of irrelevant emotional distractors differed between those with ADHD alone and those with ADHD and additional CD. To that end, adolescents with ADHD, divided into subgroups based on whether they had an additional research diagnosis of CD or not, performed the CEPS (described above). We hypothesised that overall, participants' reaction times would not differ when responding to ticks or crosses, nor would this differ between subgroups of participants. Second, we predicted that participants' responses would be faster for congruent trials (fear and cross, happy and tick) compared to incongruent trials (fear and tick, happy and cross). In relation to our third aim, the prediction for the direction of the results was more complicated. Due to the observation of problems with emotion regulation in both individuals with ADHD alone and those with comorbid CD, we predicted that both groups of participants would show interference by the emotional stimuli compared to objects, although we predicted this to be more pronounced for fearful faces. As an additional aim, because research shows that different regions of the face are more important for certain emotions (e.g. the eyes are more important for fear recognition; the mouth is more important for happiness recognition; Bassili, 1979), we also explored whether interference between groups differed depending on which part of the face the target symbol was presented over.

4.6. Method

4.6.1. Participants

A sample of 16 adolescents (7 females; 9 males) took part in this experiment. All participants were between 11 and 18 years ($M = 14.0$, $SD = 2.0$). 9 participants were in the ADHD alone group while 7 were in the ADHD+CD group. For more information on recruitment, exclusion criteria and ethics see section 2.2.1.

4.6.2. Materials and Measures

4.6.2.1. Clinical measures.

See section 2.2.2.1. The internal reliabilities of ADHD severity ($\alpha = .87$) and CD severity ($\alpha = .72$), the YPI overall ($\alpha = .93$) and CU traits subscale ($\alpha = .76$) was good.

4.6.2.2. Socioeconomic Status

See section 2.2.2.2.

4.6.2.3. Intelligence Quotient (IQ)

See section 2.2.2.3.

4.6.2.4. Emotional Stroop task

See section 4.2.2.1

4.6.3. Data Analysis

Three (one ADHD alone and two ADHD+CD) participants were excluded due to problems with the administration of the Stroop task (the microphone did not register their vocal response). Six participants fell below 70 for estimated for IQ, but due to the already small sample size these participants were included in the analysis (their reaction time scores did not differ significantly from the rest of the sample $p > .05$).

4.6.3.1. Reaction time data processing.

As in experiment 1, trials in which the reaction time was below 250msec or above 3 standard deviations above the mean were excluded along with error trials and trials immediately following errors (Rabbitt, 1966). However, data were also analysed including trials after errors and results were identical to when they were excluded. Nevertheless, results excluding trials after errors only are reported for ease of presentation. Only a small percentage of trials were excluded. On average participants made errors on 3.7% of trials, 1.04% of trials were excluded for being

faster than 250ms, while 3.6% of trials excluded for being more than 3 standard deviations above the mean. This demonstrates that participants were well engaged with the task. There were no systematic differences between percentage of trials excluded for each trial condition or between ADHD groups.

Interference scores in Stroop tasks are typically calculated in order to compare between groups (e.g. van Honk et al., 2000). We calculated standardised difference scores in order to determine extent of interference by the distractor stimuli. Fear interference was the reaction time to fear stimuli minus reaction time to non-social stimuli (houses and flower) divided by the pooled standard deviation of the reaction times. Positive scores on this measure indicated interference by fear stimuli (slower reaction times in the presence of a fear face), while negative scores indicated facilitation (faster reaction times in the presence of fear faces). The same method was used to calculate happy interference. In order to examine the influence of positioning of target stimuli over the mouth or eyes, we also calculated interference scores for the eyes and mouth separately for each emotion, this was done in the same way as above, but limited to the trials where the stimuli were either over the eyes or mouth locations of each distractor stimuli.

The congruency effect was calculated by subtracting average reaction time for incongruent trials (happy/cross and fear/tick) from congruent trials (happy/tick and fear/cross) and dividing by the pooled standard deviation. Negative scores indicated faster responses to congruent trials, while positive scores indicated faster responses to incongruent trials.

Analyses were carried out using SPSS 20 (SPSS Inc., Chicago, Illinois). Differences in demographic and clinical characteristics between groups were analysed with one-way ANOVAs for continuous variables (due to small sample sizes

these were also run non-parametrically) and X^2 test for binary variables. Pearson's (Spearman's rho where necessary) correlations were used to examine relationships between demographic and clinical characteristics with interference scores. Mixed model analysis of variance with Group (ADHD+CD, ADHD) as a between-subjects factor and Emotion (fear and happy interference), Location (eyes and mouth), and Gender of the stimuli (female and male) as within-subjects factors were conducted. Where sphericity was violated, Greenhouse-Geisser corrections were used. Where follow-up tests were required, Least Square Difference adjustments (equivalent to no corrections) were made. Due to the small sample size, follow-up comparisons were also run non-parametrically. Effect sizes were calculated as partial eta squared (η_p^2 ; small ≥ 0.01 , medium ≥ 0.06 , large ≥ 0.14 ; Cohen, 1998).

4.7. Results

Table 4.1 presents demographic and clinical characteristics. As expected groups differed in CD severity ($p < .001$) where ADHD+CD participants scored higher than ADHD alone participants, but not in any other characteristics ($ps > .05$). The same pattern of results was found using non-parametric tests.

Table 4.1. Demographic and clinical characteristics of the sample

	ADHD+CD (<i>n</i> = 7)	ADHD (<i>n</i> = 6)	<i>P</i> value
Age	14.4(2.2)	13.5(1.8)	.42
IQ _{Wasi}	81.1(16.8)	83.3(22.0)	.83
% female	57.1	50.0	.80
SES			.17
% Low	16.7	75.0	
% Medium	66.7	25.0	
% High	16.7	0.0	
CD _{Dawba}	5.1(1.6)	.83(.98)	<.001
ADHD _{Dawba}	12.4(5.7)	13.8(4.4)	.63
CU _{YPI}	30.6(9.6)	29.7(7.4)	.86
ImIrr _{YPI}	44.1(5.1)	37.3(9.8)	.14

Note: Means are presented with standard deviations in brackets (except where indicated otherwise). Both ADHD_{Dawba} and CD_{Dawba} represent number of symptoms and are restricted to the ADHD groups. IQ is also restricted to ADHD groups. CD_{SDQ} is CD score as measured by SDQ. CU_{YPI} is the CU subscale for the YPI. *Key:* ADHD = Attention Deficit Hyperactivity Disorder. CD = Conduct Disorder. CU = Callous Unemotional. ImIrr = Impulsive Irresponsible. DAWBA = Development and wellbeing assessment. IQ = Intelligence quotient (2 subtest) WASI. SES = Socioeconomic Status. YPI = Youth Psychopathy Inventory.

4.7.1. Reaction Times to Target Stimuli

A paired sample t-test was performed on the reaction times to ticks and crosses across all distractor pairings. Reaction times to the ticks ($M = 522.2$, $SD = 73.6$) and crosses ($M = 517.2$, $SD = 87.9$) did not significantly differ ($t(12) = .81$, $p = .44$, $d = 0.06$). Independent samples t-tests also revealed reaction times to ticks ($t(11) = -.16$, $p = .87$) and crosses ($t(11) = -.13$, $p = .90$) did not differ significantly between ADHD and ADHD+CD participants.

4.7.2. Congruency Effect

A one-way ANOVA revealed a significant difference in congruency effect between groups (see Figure 4.3, Panel A), such that ADHD participants were facilitated by congruent trials (faster to congruent trials; $M = -0.41$, $SD = .56$) while ADHD+CD participants were slower to congruent trials ($M = 0.17$, $SD = .31$; $F(1,11) = 5.7$, $p =$

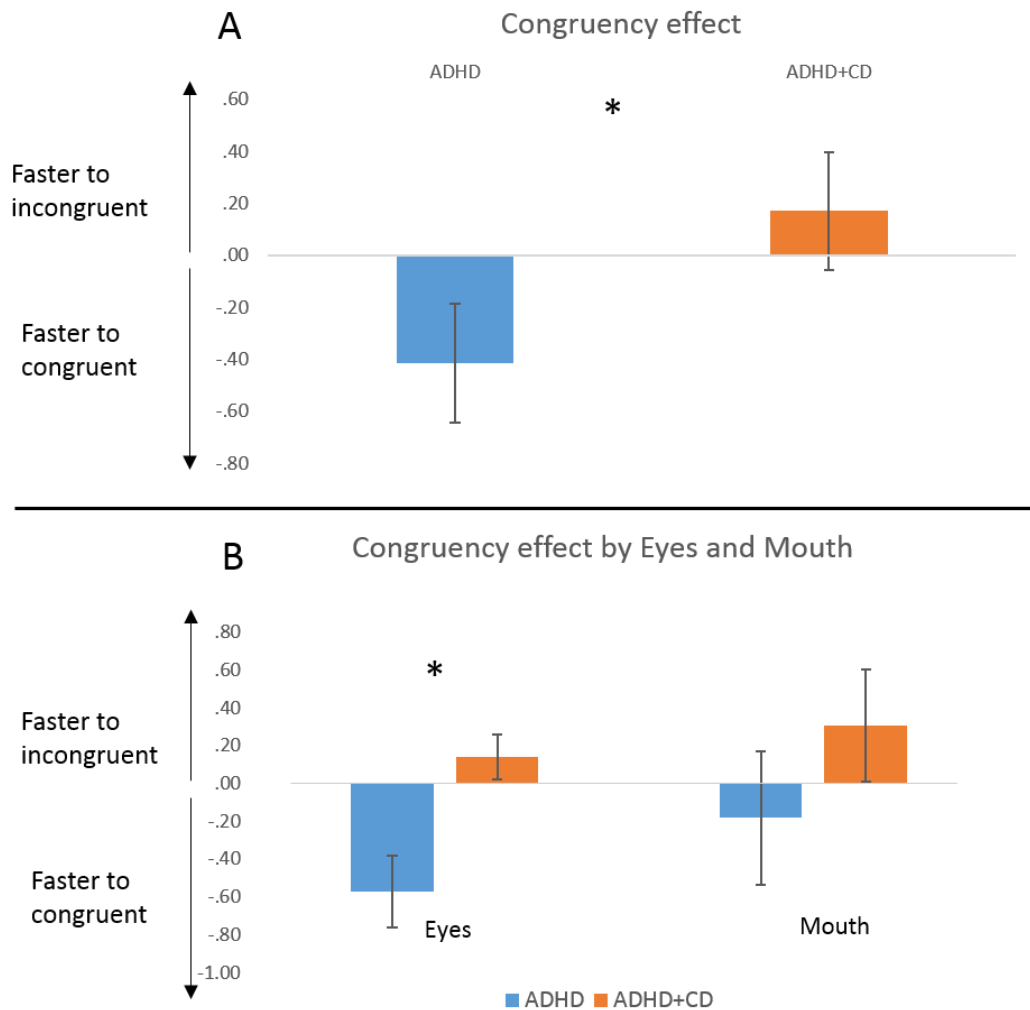


Figure 4.3. Panel A: Congruency effect overall. Panel B: Congruency effect split by whether the target appeared over the eyes or mouth. Negative scores indicate faster responses to congruent trials while positive scores indicate faster responses to incongruent trial. * $p < .05$

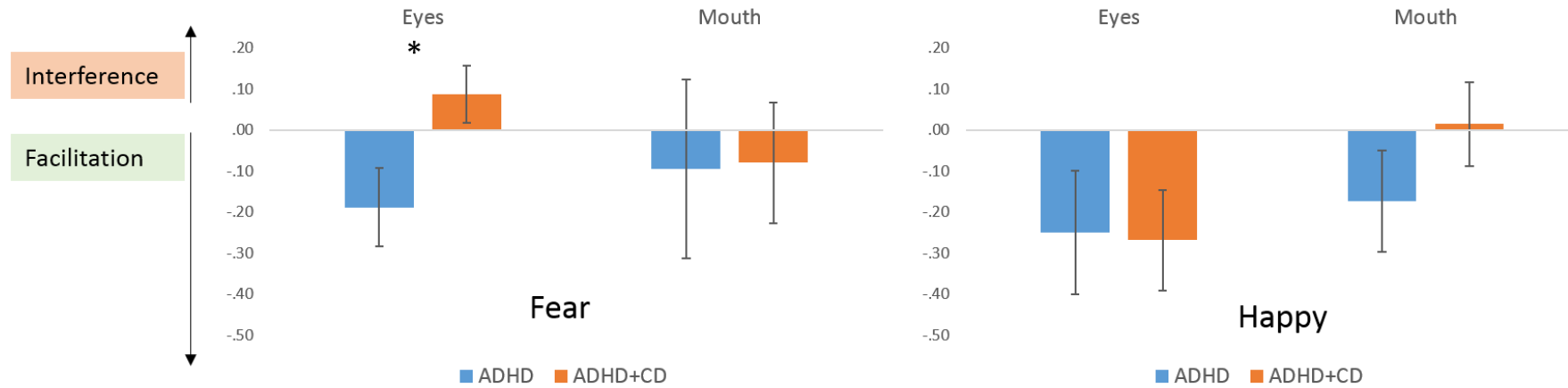
.036 $\eta_p^2 = .34$). We explored this effect further by examining the group difference in congruency to the eyes and mouth separately (see Figure 4.3, Panel B). The group difference was only present when the target stimuli were presented over the eyes

($F(1,11) = 10.6, p < .01, \eta_p^2 = .49$), but not over the mouth ($F(1,11) = 1.1, p = .31, \eta_p^2 = .09$).

4.7.3. Emotional Interference

The mixed model ANOVA revealed a main effect of Emotion ($F(1,11) = 5.5, p = .04, \eta_p^2 = .33$), such that happy faces resulted in more facilitation ($M = -0.33, SE = .13$) than fear faces ($M = -0.13, SE = .14$). There was no overall effect of group ($F(1, 11) = .24, p = .63, \eta_p^2 = .02$), nor an interaction between group and emotion ($F(1,11) = .01, p = .92, \eta_p^2 = .001$). There was no main effect of target location ($F(1,11) = .05, p = .83, \eta_p^2 = .004$) nor was there was an interaction between location and group ($F(1,11) = .03, p = .87, \eta_p^2 = .003$), or between emotion and location ($F(1,11) = .82, p = .38, \eta_p^2 = .07$). However, importantly, the three way interaction between emotion, location and group was significant ($F(1,11) = 4.9, p < .05, \eta_p^2 = .31$). Post hoc comparisons (see Figure 4.4) revealed a significant difference in interference scores between groups but only for trials where the distractor stimuli were fear and the target was presented over the eyes ($p < .05$). ADHD alone participants were facilitated in this condition ($M = -0.27, SE = .12$) while ADHD+CD participants showed interference in this condition ($M = 0.10, SE = .11$). When exploring this effect non-parametrically we found the same result ($U = 7.0, p = .046$). There were no significant differences between groups in either of the other distractor-target combinations ($ps > .05$; also confirmed with Mann-Whitney U tests).

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*Figure 4.4. Interference scores reflecting differences between reaction time to emotional faces compared to objects. Negative scores indicate faster responses to emotional stimuli, positive scores indicate faster responses to objects. Error bars represent +/- 1 Standard error. * $p < .05$*

When examining the effect of stimulus gender, we only found a significant interaction between stimulus gender and emotion ($F(1,11) = 5.4, p = .04, \eta^2 = .33$) such that male happy faces facilitated ($M = -0.33, SD = .33$) whereas female happy faces did not ($M = -0.06, SD = .21; p = .04$), while there was no effect of distractor gender for fear faces ($p = .96$). The main effect of gender of the distractor was not significant, nor were there any other significant interactions involving gender of the stimuli ($F_s(1,11) < 2.8, p_s > .12, \eta^2_s < .20$).

4.7.4. Relationship between Clinical Characteristics and Emotional Interference

A correlation matrix of the relationship between clinical characteristics and interference scores are presented in Table 4.2. There were no significant relationships between any of the clinical and interference variables. However, the relationship between CD severity and fear interference at the eyes ($r = .52, p = .07$) and between CD severity and congruency effect at the eyes ($r = .55, p = .054$) approached significance, indicating that as CD severity increased, facilitation via fear eyes decreased, and facilitation by congruent trials at the eyes decreased. These relationships were subsequently re-run as regression analyses in order to determine confident intervals. Again, the relationship between CD severity and fear interference at the eyes approached significance ($\beta = .52, p = .066, 95\% \text{ CI } [-0.004, 0.10]$), as did and the relationship between CD severity and congruency effect at the eyes ($\beta = .55, p = .054, 95\% \text{ CI } [-0.002, 0.23]$). Psychopathic traits or ADHD severity scores were not found to relate to any interference variables.

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Table 4.2. Correlation matrix of clinical characteristics and interference scores

	1	2	3	4	5	6	7	8	9	10	11
Clinical Characteristics											
1. CD _{Severity}	-										
2. ADHD _{Severity}	.12	-									
3. CU _{YPI}	.36	.49	-								
Interference scores											
4. Fear	.21	-.40	-.07	-							
5. Happy	.04	-.13	.13	.47	-						
6. Fear Eyes	.52	-.21	-.01	.86 ^{***}	.21	-					
7. Fear Mouth	-.27	-.46	-.07	.76 ^{**}	.63 [*]	.31	-				
8. Happy Eyes	.02	.08	.22	.31	.91 ^{***}	.18	.38	-			
9. Happy Mouth	.06	-.63 [*]	-.23	.45	.15	.21	.56 [*]	-.25	-		
10. Congruency	.35	-.13	-.25	.53	.34	.44	.43	.09	.59 [*]	-	
11. Congruency Eyes	.55	-.28	-.01	.48	.39	.43	.37	.18	.45	.88 ^{***}	-
12. Congruency Mouth	.08	-.26	-.49	.52	.13	.48	.33	-.04	.43	.78 ^{**}	.41

ADHD_{Severity} and CD_{Severity} represent symptom severity as measured by the DAWBA. *Key:* ADHD = Attention Deficit Hyperactivity Disorder. CD = Conduct Disorder. CU_{YPI} = Callous Unemotional. DAWBA = Development and wellbeing assessment. YPI = Youth Psychopathy Inventory. * $p < .05$. ** $p < .01$. *** $p < .001$

4.8. Experiment 2 Discussion

The aim of experiment 2 was to replicate the results from experiment 1 in that reaction times to the target stimuli would not differ by condition (tick vs. cross) while also confirming that reaction times to the ticks and crosses did not differ between groups. Secondly, we sought to replicate a congruency effect such that participants are faster to respond in the presence of congruent trials than incongruent trials, while also exploring whether the congruency effect differed between groups with ADHD+CD and ADHD alone. Further, we examined whether the congruency effect differed depending on whether target stimuli appeared over the eyes or the mouth of the distractor. Finally, we aimed to explore the general interference effect of emotional distractors compared to object distractors while also comparing if this differed by ADHD group.

In relation to the first aim, and in line with results from experiment 1 there was no difference between reaction times to the target stimuli overall, nor did this differ between groups, therefore, both groups of participants found the target aspect of the task equally difficult. This is important because it implies that any differences in reaction times between different distractor-target combinations can be attributed to processing of the irrelevant distractor stimuli and not the target.

Secondly, again replicating results from experiment 1, we found that overall, participants were faster to respond to congruent than incongruent trials, illustrating that the paradigm created a true Stroop effect. However, interestingly, it was found that the congruency effect was significantly stronger for the ADHD alone group than the ADHD+CD group. Examination of the mean interference scores highlighted that while those with ADHD alone were facilitated by congruent trials, while participants

with ADHD and CD showed interference (they were faster to incongruent than congruent trials). When further probing these differences by separating trials into those in which targets were presented over the eyes or the mouth, it was revealed that the significant difference between groups was being driven by trials in which the target appeared over the eyes.

Finally, we explored the extent of general interference by emotions compared to objects and found that compared to ADHD alone (who were facilitated), ADHD and comorbid CD participants showed interference when targets were presented over the eyes of a fear face, compared to when targets appeared over the same locations of the object stimuli. In addition, when examining the relationship between symptom severity, CU traits and interference measures, CD symptom severity was strongly positively related to fear interference and strongly negatively related to response time in the presence of congruent trials; no such relations were observed for CU traits and ADHD severity.

How these findings and the ones from experiment 1 relate to previous literature and what their implications are, will be discussed in the next section.

4.9. General Discussion

The current study sought to (1) test a pictorial emotional Stroop task (CEPS) in which responding to the each of the target stimuli was of equal difficulty, and that difficulty of the target task over all distractor-target pairings, did not differ between groups. (2) We also aimed to create a task which utilised true Stroop interference, in the form of congruent and incongruent trials and whether such an effect differed between ADHD and ADHD with comorbid CD participants, and (3) we explored whether interference by emotional distractors differed between adolescents with ADHD with or without comorbid CD.

In relation to our first aim, we were successful in creating a paradigm in which responding to the target stimuli conditions was equally difficult and equally difficult across clinical groups. This is important for studies using irrelevant emotional faces as distractors as evidence suggests that increased task difficulty reduces amygdala response to emotional stimuli, suggesting reduced processing (Sebastian et al., 2017), while in general there is also a consensus that increased cognitive load reduces irrelevant distractor processing (Lavie, Beck, & Konstantinou, 2014).

In relation to our second aim, we found that the current paradigm produced a significant congruency effect (participants, in general, responded faster in the presence of congruent trials compared to incongruent pairings). Finding a true congruency effect in this study was important due to the criticisms of earlier pictorial emotional Stroop tasks that have relied on coloured hues appearing over the distractor stimuli (e.g. Ashwin et al., 2006), which have typically only found small effect sizes (Buhle et al., 2010). In addition, by finding a true congruency effect, we can be more certain that emotional processing of the distractors is taking place and differences in reaction time are not due to other factors. However, we found that the significant congruency effect was being driven by ADHD only participants, while ADHD with CD participants showed slower reaction times during congruent compared to incongruent trials. Given that both ADHD alone and ADHD with comorbid CD have been associated with emotional dysregulation, it is important to contextualise the direction of results with reference to a healthy control group. Indeed, in experiment 1 of 50 undergraduate students, who can be considered typically developing, participants responded faster in the presence of congruent trials than incongruent trials, providing further evidence that the current paradigm was

successful in creating a congruency effect, and suggesting that those participants with ADHD with CD were performing atypically (because they were faster to incongruent trials than congruent trials; See Figure S4, Appendix C for comparison of congruency effect between all three groups). The fact that the ADHD+CD group performed atypically in this task is consistent with findings of Chapters 2 and 3 where this group also showed impaired performance compared to the ADHD alone group, suggesting emotion processing impairments are specific to those with ADHD with additional CD.

The third and final aim of the current study was to explore differences in interference between groups. The results of our study revealed specific differences between groups in interference effects for fear distractors when the target stimulus was presented over the eyes, such that ADHD alone participants were facilitated in the presence of fear faces compared to neutral objects, while ADHD and CD participants showed interference. As above in relation to the congruency effect, the direction of results in terms of whether interference or facilitation is reflective of more effective emotion regulation is difficult to contextualise. However, the finding that happy faces resulted in increased facilitation compared to fear faces, suggests that facilitation is not a reflection of looking away from the face (as suggested by Ashwin et al., 2006) and instead suggests that increased attention to the face facilitated responding to the target task (in the current paradigm at least). The fact that we found that the emotional distractors in the current paradigm facilitated responding, as opposed to interfered with it, is at odds with the suggestion by Carretié (2014) that emotional distractors cause interference. However, for many of the studies reviewed by Carretié (2014), distracting emotions were presented in a location other than where the targets appeared, therefore interference may have

reflected the fact that attention was directed away from the target task and towards the distracting faces. In our study, the targets appeared over the key features of the emotional faces; therefore, when attention was attracted to these areas, and target stimuli appeared in these same locations, this resulted in facilitation. In support of our finding of facilitation by emotional stimuli, Phelps, Ling, and Carrasco (2006) found that the presence of a fear emotional distractor increased contrast sensitivity, while Liu, Xin, Jin, Hu, and Li (2010) also found a facilitatory effect when emotional faces were paired with congruent emotional words, regardless of whether they were presented centrally on the face or in the periphery. Further, the fact that a sample of healthy undergraduate students in experiment 1 responded faster to congruent trials than incongruent trials, supports the idea that facilitation is a reflection of typical performance for the current paradigm. We found a significant congruency effect, suggesting that attentional capture by emotional stimuli facilitated performance when the distractor semantically matched the target, while the distractor interfered with performance when it was incongruent with the distractor. We also found that undergraduates in experiment 1 showed facilitation in the presence of emotions compared to objects, such that regardless of whether the emotional pairing was congruent or incongruent, participants responded faster in the presence of emotional faces compared to objects.

Together, the above reasoning can be taken as preliminary evidence that participants with ADHD and comorbid CD were performing atypically on the current task as they were both slower in the presence of congruent trials than incongruent trials, while they were also slower to respond when a target appeared over the eye region of a fear face. The results are in line with studies that have found emotion regulation abilities are compromised in those with ADHD and CD (e.g.

Hinshaw & Melnick, 1995; Northover et al., 2015a). The current findings suggest that participants with ADHD and CD were less susceptible to attentional capture by the emotional faces as reflected by the absence of facilitation in this group. These findings have relevance to those in Chapter 2, whereby the ADHD+CD group took significantly longer to focus the eye region than the typically developing control group (but not the ADHD alone group). Together, interpreting these results in line with emotional dysregulation theory (Cole et al., (1994), would suggest that those with ADHD and CD have impairments in the bottom-up processing of environmental stimuli which would ordinarily help participants automatically tune their emotions and behaviour to what is appropriate to a situation. Our results also have relevance to Blair's (2005) integrated emotion systems (IES) model, which suggests that a dysfunction in the amygdala is central to the behaviour of aggressive individuals. This dysfunction prevents these individuals from processing the distress cues of others and from receiving the inhibitory input required to desist from antisocial behaviour.

Our findings have relevance for studies that have suggested there is a neglect of processing of the eye region in those with CD (e.g. Dadds et al., 2006). We found a difference in interference between groups when the target was presented over the eye region of the fear face. The facilitation in the ADHD alone group suggests their attention was already focused on the eye region when the target appeared (the distractor appeared first), while the interference effect found in those with ADHD and CD suggests they were looking away. Indeed, as the interference score was calculated by subtracting reaction times to neutral objects when the target appeared over the eyes from those to fear when the target was presented over the eyes, it suggests that those with CD paid more attention to the eye region when the distractor

was an object than when it was a fear face. This provides some tentative evidence (given that the distractor appeared before the onset of the target) that CD participants automatically shifted their attention away from the eyes when the fear distractor face appeared. However, it is important to emphasise that all participants had a diagnosis of ADHD, therefore it is still currently up for debate if such attentional problems are a by-product of additional ADHD. Further research should seek to include a CD alone group to explore whether similar attentional patterns are found.

Barkley (1997) suggested that the inhibitory problems of those with ADHD lead them to lack the necessary time to adequately consider the emotional context before responding to situations. In light of the current findings, those with comorbid ADHD and CD may be particularly impaired (compared to CD alone) due to their lack of inhibition resulting in them acting before fully processing emotions. While their additional impairments in emotion processing suggest that even if problems with inhibition were reduced, these individuals would still have impairments in processing emotional situations and therefore responding appropriately.

The finding of emotion dysregulation in those with ADHD with additional CD is important given that a number of studies have found that problems in emotion regulation mediate the link between environmental risk and later aggressive behaviour (e.g. Kin & Cicchetti, 2010; McKenzie & Casselman, 2017), and because better emotion regulation skills are a protective factor against aggressive behaviour (Benavides, 2015; Calvete & Orue, 2012). Together, these findings suggest that targeting emotion regulation impairments in interventions for those with CD makes sense.

The current study has a number of strengths. By utilising a task in which the distractors share semantic associations with the targets, we have improved upon the

colour emotional Stroop task (e.g. Ashwin et al., 2006). Such a relationship has been argued to be crucial to the true Stroop interference effect (Buhle et al., 2010), which has proven to be robust over the last century. Indeed, when comparing the congruency effect between groups in experiment two, we found a large effect size ($\eta_p^2 = .49$). In addition, by equating the difficulty of the target task, both in terms of responding to the individual targets and the difficulty of performance between groups, we can be more confident that any interference or facilitation differences between groups reflect the influence of irrelevant emotional distractors and not responses to the target task (Sebastian et al., 2017). Finally, the current paradigm improved upon colour emotional Stroop tasks by presenting the target stimuli over the top of the features of the distractors. In colour Stroop tasks, participants could adopt a strategy whereby they never need to attend to the features of the face (for example by focusing on the forehead) and still perform well on the task and therefore this could explain the small effect sizes found in previous versions (Buhle et al., 2010). In the current paradigm, the locations of the features of the face were relevant, despite the stimuli themselves being irrelevant to the completion of the target task, therefore to perform well on the task participants had to engage with the features of the face.

It is also important to consider the limitations of the current study when interpreting the current findings. Firstly, a clear limitation of the current study is its small sample size. In order for the findings to be generalised, this task will require replication in larger samples. In addition, other than comparison undergraduates in experiment 1 who therefore were not matched for age or other environmental risk factors, our study did not include a typically developing control group or a CD alone group. These groups will be required in future studies to help determine if any

particular group shows impaired performance compared to others. It would also be interesting to establish whether those with CD alone show similar attentional deficits as those with ADHD and CD using this paradigm. If participants with CD alone were to show similar deficits it would suggest that such attentional deficits to emotional features are unrelated to ADHD and instead a general problem for those with CD, however if such impairments were not present, it would suggest that the combination of the two presentations result in more severe emotion processing impairments. Shaw et al., (2014) considered three possible conceptualisations of the relationship between ADHD and emotion dysregulation. The first being that emotion dysregulation is a core diagnostic feature of ADHD, second that they are distinct but correlated problems, and finally that the combination of the two represents a distinct entity. The results from the current study are against the first two ideas, as emotion dysregulation seemed to be driven by CD and therefore to some extent supports the idea that the combination is a distinct entity, however, because we had no CD alone group we cannot say for sure if it is in the combination, or CD itself which results in problems with emotion regulation. Finally, it is also important to keep in mind that the current study only explored one, albeit central (Gross, 1998; Shaw et al., 2014) component of emotion regulation, namely attention. The entire process of emotion regulation is more complex (see Gross, 2015). Our findings speak only to the attentional difficulties that may contribute to overall emotion regulation impairments; therefore, future research should also consider deficits in other components and how they may relate to one another.

In conclusion, the current study developed a novel pictorial emotional Stroop task (CEPS) in which the targets were equally difficult (or easy) to identify between groups of ADHD and ADHD with CD. A congruency effect was also successfully

observed, akin to true Stroop interference, which previous picture based emotional Stroops were unable to observe. In terms of a clinical difference in performance between ADHD and ADHD with CD, the study provides preliminary evidence that those with ADHD and CD were less attracted to the eye region of fear faces; this suggests these individuals would therefore not benefit from the facilitation that this type of information would ordinarily provide, which has implications for how they respond in emotionally charged situations.

5. Chapter 5: Fear Conditioning and Rate of Extinction

Paper in preparation

This chapter is based on Airdrie, J. N., Langley, K., Thapar, A., van Goozen, S. H. M. (2017). Fear conditioning impairments and expedited rate of extinction in Attention-Deficit/Hyperactivity Disorder and comorbid Conduct Disorder. *In prep.*

5.1. Introduction

Chapter 2 explored emotion recognition impairments in those with ADHD and ADHD+CD, finding specific impairments for fear and neutral faces in those with ADHD+CD compared to ADHD alone and controls. Chapter 3 found general impairments in affective empathy (and not cognitive) in those with ADHD+CD compared to ADHD, while Chapter 4 found that ADHD+CD showed impairments compared to ADHD alone in an attentional aspect of emotion regulation. The above three chapters focused on the processing of the emotional signals of another person. Next, the current chapter focuses on participants' ability to effectively learn an association between a neural stimuli and a fear response evoked in the individual themselves and the speed at which said association is lost again. Namely, fear conditioning ability and rate of extinction.

There are some key hypothesised risk pathways to antisocial behaviour that warrant further investigation (van Goozen et al., 2007). Impairments in the processing of emotionally aversive stimuli is one process that is strongly associated with antisocial behaviour in the general population (Blair, 2005; Raine, Venables, & Mednick, 1997; Snoek, van Goozen, Matthys, Buitelaar, & van Engeland, 2004; van Goozen et al., 2007). One such measure of emotion processing is fear conditioning. Fear conditioning is a form of Pavlovian conditioning where an individual learns the significance of a previously neutral stimuli through a process of association. In a typical fear conditioning paradigm, a neutral stimulus (conditioned stimulus; CS+) is paired with an aversive one (unconditioned stimulus; US) such as an aversive noise. After repeated pairings of the CS+ and US, the initially neutral stimulus will elicit a

response (conditioned response; CR), similar to that caused by the US. The response to this conditioned stimulus (CS+) is compared to the response to a control stimulus, which has not been paired with an aversive stimulus (CS-). The skin conductance response (SCR) is a reliable measure of emotional arousal and is often used as the unconditional response (UR) in fear conditioning paradigms because unusual skin conductance responding can help identify impairments in emotion processing (Dawson, Schell, & Filion, 1990).

Abnormalities in fear conditioning have been found in those displaying antisocial behaviour in adulthood (Birbaumer et al., 2005; Flor, Birbaumer, Hermann, Ziegler, & Patrick, 2002; Lykken, 1957; Raine, 1993). It has been suggested that impairment in this type of learning is a risk factor for antisocial behaviour, as individuals will not learn to associate particular situations, behaviours and contexts with punishment. If such individuals cannot learn in such a way, it may suggest certain intervention strategies would not be effective (Flor et al., 2002; Pine, 2010; Raine, 1993).

Underlying deficits in the amygdala are thought to play a role in the emotion processing impairments of those who display antisocial behaviours (Fairchild et al., 2011; Blair, 2005). For example, adolescents with early onset CD, when viewing affective pictures were found to have deficient amygdala activation compared to a control group (Sterzer, Stadler, Krebs, Kleinschmidt, & Poustka, 2005). The amygdala is also critically involved in emotional learning (Everitt, Cardinal, Parkinson, & Robbins, 2003). Studies investigating functioning in individuals with lesions to the amygdala have shown that it is required for the acquisition of fear conditioned responses (Bechara et al., 1995; LaBar et al., 1995). Neuroimaging studies in humans have also found that the amygdala is activated during fear

conditioning (Buchel, Morris, Dolan, & Friston, 1998). As a result, conditioning ability has often been used as an alternative measure of amygdala functioning (Fairchild et al., 2011).

While studies on fear conditioning ability in adults displaying antisocial behaviour has been well replicated, research in adolescents who display such behaviours is more limited. However, recent research has shown that fear conditioning is impaired in both males (Fairchild et al., 2008) and females (Fairchild et al., 2010) with CD. The severity of this emotional learning deficit has also been found to be associated with frequency of antisocial behaviour in a group of juvenile male offenders (Syngelaki et al., 2013). In addition, longitudinal studies have found that reduced fear conditioning ability in 3-year-old children predicted aggressive behaviour at age 8 (Gao, Raine, Venables, Dawson, & Mednick, 2010a) and criminal behaviour at age 23 (Gao, Raine, Venables, Dawson, & Mednick, 2010b) suggesting fear conditioning ability is an early risk marker for antisocial behaviour problems later in life.

Recent research has also illustrated that poor fear conditioning is a factor that distinguishes those who have ADHD and associated conduct disorder from those with ADHD alone (van Goozen et al., 2016). In this large study ($n = 108$) participants with ADHD were found to differentially condition to stimuli paired with an aversive noise versus those presented alone, but participants with ADHD and additional CD were unable distinguish between these two stimulus types. However, as this was the first study to find such an effect, and due to problems with replication currently overshadowing psychological research, the primary aim of the current study was to replicate these findings in a new and smaller sample to add further support to the generalisability of these findings.

A further issue is whether or not fear conditioning deficits are found universally in those with CD or are only found in specific presentations of CD. A recent specifier added to the Diagnostic and Statistical Manual of Mental Disorders (DSM-V; American Psychiatric Association, 2013) in the diagnosis of CD is the presence or not of CU traits. These traits found in childhood are analogous to the interpersonal-affective dimension of psychopathy found in adults. Typically, fear conditioning deficits in adult psychopathy are thought to be positively associated with the interpersonal-affective traits, or primary psychopathy (e.g. Birbaumer et al., 2005; Lykken, 1957; Veit et al., 2013). However, the contribution of CU traits to fear conditioning deficits in studies of children and adolescents is not so clear. Some studies do find a relationship to the interpersonal-affective dimension. For example, Gao, Tuvblad, Schell, Baker, and Raine (2015) found deficits in fear conditioning were related to instrumental (proactive) as opposed to reactive aggression. However, other studies have failed to find an association (Fairchild et al., 2008; Northover, 2015) or have not investigated the relationship (e.g. Gao et al., 2010a) between CU traits and fear conditioning deficits. In addition, various fMRI studies have found reduced amygdala reactivity in CD adolescents with CU traits in comparison to healthy controls, but in these studies comparisons were not made between CD individuals with and without CU traits (e.g. Jones, Laurens, Herba, Barker, & Viding, 2009). Indeed, in their review article on the role of CU traits in CD, Scheepers, Buitelaar, and Matthys (2011) suggest that the role of CU traits in fear conditioning requires further investigation. In a recent study in the adult psychopathy literature, fear conditioning deficits were only found in the more reactive and impulsive secondary psychopaths, while primary psychopaths displayed normal conditioning ability (Schultz, Balderston, Baskin-Sommers, Larson, & Helmstetter,

2016). It is possible that fear conditioning deficits relate to an increased number of impulsive traits in those with CD. As a result of the inconsistencies above, it is important to also consider impulsive-irresponsible traits alongside CU in order to explore what might be the driving factor behind fear conditioning deficit in those with ADHD and CD, hence impulsive-irresponsible traits being included here and not in other chapters. Further, other studies have found that it is the severity of conduct problems and antisocial behaviour that is most strongly related to fear conditioning deficits (Fairchild et al., 2008; Syngelaki et al., 2013). Therefore, the secondary aim of the current study was to investigate the relationship between fear conditioning deficits and both CU and impulsive traits in those with ADHD and CD while also exploring the contributing effects of ADHD and CD severity. In order to measure impulsive traits we used the impulsive-irresponsibility subscale on the YPI which we were already using to measure CU traits.

A further neglected area in research of those with CD is extinction learning. Extinction learning is the phase of the fear conditioning paradigm where the presentation of the neutral stimulus is no longer paired with the aversive one in order for the associated fear response to dissipate upon presentation of the neutral stimulus. Typically, studies employing fear conditioning paradigms examine the overall differences between skin conductance responses to reinforced and non-reinforced slides over the entire extinction phase and find no differences between those displaying antisocial behaviour and those who do not (Fairchild et al., 2010, 2008; Northover, 2015; Syngelaki et al., 2013). However, there is reason to believe that the extinction phase in conditioning paradigms needs further exploration in regards to its role in antisocial behaviour. In a study by Syngelaki et al., (2013), deficits in fear conditioning in young offenders were more pronounced in the late

phases of the conditioning paradigm. The authors speculated that this effect may have been apparent due to young offenders extinguishing the conditioned response more rapidly than typical controls. Therefore, perhaps it is the case that adolescents displaying antisocial behaviour have a faster rate of extinction (they lose the association between a stimuli and punishment more quickly) than those who do not display antisocial behaviour. In addition, although abnormal amygdala functioning has primarily been discussed in its role in deficient *acquisition* of the conditioned response, the amygdala is also thought to be involved in the *extinction* of conditioned fear (Davis, 1992). Therefore, amygdala abnormalities found in those with CD (Fairchild et al., 2011) may also lead to abnormal extinction learning. Furthermore, evidence from individuals suffering from psychopathology at the other end of the spectrum in terms of anxiety may suggest abnormal extinction processes. In contrast to the fearlessness associated with fear conditioning deficits present in those displaying antisocial behaviour, individuals suffering from Obsessive-Compulsive Disorder (OCD) have very high levels of anxiety. Studies of fear conditioning in these individuals have found that they have deficits in extinction recall (Milad et al., 2013), i.e. after the conditioned response has been extinguished (a neutral stimuli is no longer associated with an aversive one) these individuals still respond to the neutral stimuli as if it is still associated with an aversive stimulus. Therefore, it may be the case that those at the other extreme of the continuum, who are characterised by fearlessness, lose the association between the stimuli more readily. Therefore as a tertiary aim, we sought to explore whether the rate of extinction to the conditioned response was faster in individuals with CD compared to those with ADHD alone.

In order to explore whether individuals with CD have a faster rate of

extinction one must employ a method of analysis that accounts for more than just an overall difference at the extinction phase as has been the case in previous studies.

One way to explore a change over time (and therefore rate of extinction) is using an Area under the Curve (AUC) analysis (see Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003) which has typically been used in endocrine research. Due to the differences that are also expected to be present at the acquisition phase, any analysis of AUC during the extinction phase will also need to control for these differences.

In order to explore these aims, adolescents with a clinical diagnosis of ADHD with or without comorbid CD underwent a fear conditioning task while their skin conductance was recorded. Based on the findings of van Goozen et al. (2016), our primary hypothesis was that individuals with ADHD and comorbid CD would show deficits in the acquisition of a conditioned fear response relative to those with ADHD alone. In addition, based on the findings of Syngelaki et al., (2013), we predicted that ADHD+CD participants would show a faster rate of extinction than individuals with ADHD alone. Finally, due to inconsistent findings of the relationship of CU traits and impulsivity with fear conditioning impairments, we held an open hypothesis; however, if a relationship was found we predicted it to be in the negative direction, such that increased number of traits would be associated with increased impairments in fear conditioning, while fear conditioning impairments would also be positively related to CD severity but unrelated to ADHD severity.

5.2. Method

5.2.1. Participants

Forty one adolescents (36 males; 5 females) between 11 and 18 years ($M = 14.4$, $SD = 1.9$) took part in the current study, 22 were in the ADHD alone group, while 19

were in the ADHD+CD group. For additional information on recruitment, exclusion criteria and ethical approval see section 2.2.1.

5.2.2. Materials and Measures

5.2.2.1. Clinical measures.

See section 2.2.2.1. The internal reliability of severity ($\alpha = .90$) CD severity ($\alpha = .77$) was good.

CU and Impulsive-Irresponsible traits were measured using the Youth Psychopathy Inventory (YPI; Andershed, Kerr, Stattin, & Levander, 2002). The internal reliability of the YPI overall ($\alpha = .95$) and subscales ($\alpha = .82$, and $\alpha = .80$, respectively) was good.

5.2.2.2. Socioeconomic Status (SES)

See section 2.2.2.2.

5.2.2.3. Intelligence Quotient (IQ)

See section 2.2.2.3.

5.2.2.4. Fear conditioning.

The fear conditioning experiment was a replication of that described by Bechara and Damasio, (2002). Participants viewed 48 coloured slides (red, blue, orange and green) presented on a computer screen. Ten of the 48 slides were paired with a loud (99 dB) aversive white noise lasting 1000msec, which was presented bi-aurally using headphones. The slides served as the visual conditioned stimuli (CS), the aversive loud noise was the unconditioned stimulus (US), and SCRs (mean SCR amplitude over the six second period following the slide presentation) to the CS were measured as the dependent variables during conditioning. The coloured slides were presented for three seconds, with a 10 second inter-stimulus interval. White noise was paired with the blue slide (CS+) two seconds after slide onset.

The fear conditioning protocol was divided into four phases: a habituation phase (HAB), a first acquisition phase (ACQ1), a second acquisition phase (ACQ2) and an extinction phase (EXT). The blue slides were reinforced with the US only during the acquisition phases. During the habituation phase, two unreinforced blue slides (CS+) and two red slides (CS-) were mixed with two green and two orange slides (8 trials overall). The combined acquisition phases consisted of eight unreinforced blue slides, 10 reinforced blue slides and 10 red slides (which were never reinforced), while two green and two orange slides were also mixed in (32 trials overall). The extinction phase consisted of six unreinforced blue (CS+) and three red (CS-) slides (the other colours did not appear during this phase; 9 trials overall). For each of the phases the coloured slides were presented in the same pseudo-random order for each participant. Each phase was scored by subtracting SCRs to CS- from SCRs to unreinforced CS+ to assess whether SCRs increased as a result of differential conditioning to the CS+.

After the experiment had finished participants were asked questions (as described in Fairchild et al., (2008)) about the paradigm to ensure they were paying attention. The participants were required to identify how many different colours they saw (0.5 for correct answer), to name the colours (0.5 for correct answer), to identify how many different colours were followed by the white noise (0.5 for correct answer), and to name the colour followed by the white noise (2.5 for a correct answer). Participants needed a total score of 1.5 to be included in the analysis.

5.2.2.5. Skin conductance recording.

Electrodermal activity was recorded using a skin conductance amplifier (PSYLAB Contact Precision Instruments, UK). Skin conductance paste (ABRALYT 2000, Chloride free abrasive electrolyte gel, supplied by Falk Minow Services DE-82211

Herrsching) was used to fill the 8mm diameter silver/silver chloride electrodes, which were placed on the distal phalanges of the index and middle fingers of the non-dominant hand; electrodermal activity was sampled at 500 Hz. A SCR typically takes 4 or 5 seconds to complete and so SCRs were measured in the six second period following presentation of the CS. A valid SCR was considered to exceed an amplitude of .05 μ Siemens (μ s) (see Fairchild et al., 2008; Syngelaki et al., 2013). The internal consistencies (Cronbach's alphas) for the SCRs to the US and CS+ were 0.93 and 0.76 respectively.

5.2.3. Data Analysis

Data from two participants could not be included because of technical problems (both ADHD alone).

The IQ of 8 participants was estimated to be less than 70 so were not included (3 ADHD and 5 ADHD+CD). Two of the remaining participants were unable to answer which slide was paired with the noise but as they scored above 1.5 to the attention questions so they were included (these participants did not differ from other included participants in any demographic, clinical or experimental performance measures ($ps > .05$)).

Mixed model ANOVAs were used to assess baseline SCR and US responding between groups. One-way ANOVAs were used to compare conditioning ability between groups by calculating SCR difference scores between CS types for each of the conditioning phases. It was hypothesised that there would be no difference between groups in how participants would respond to the different CS types for the habituation and extinction phase (overall). However, we predicted that there would be a significant difference between groups in how they responded to the CS types during the acquisition phases. Degrees of freedom were corrected using

Greenhouse-Geisser estimates of sphericity, where assumptions of sphericity were violated. Effect sizes were reported as partial eta squared (η^2 ; small $\geq .01$, medium $\geq .06$, large $\geq .14$; Cohen, 1988).

We also wanted to compare rate of extinction between the groups. As mentioned above, to do this, we calculated AUC of SCRs to the CS+ during the extinction phase. The AUC can be used when conducting repeated measures over multiple time points, it can simplify, whilst increasing the power of an analysis without sacrificing the information contained in multiple measurements (Pruessner et al., 2003). We chose the AUC with respect to ground (AUCg) analysis as it is sensitive to changes over time and can accept both increases and decreases in response (which can be the case between trials of the extinction phase; see Pruessner et al., 2003). The calculation of AUCg for each participant is summarised by equation 1.

Equation 1. The calculation of Area Under the Curve with respect to ground.

$$AUCg = \frac{m_2 + m_1}{2} + \frac{m_3 + m_2}{2} + \frac{m_4 + m_3}{2} + \frac{m_5 + m_4}{2} + \frac{m_6 + m_5}{2} + \frac{m_7 + m_6}{2} + \frac{m_8 + m_7}{2}$$

Where m refers to the SCR to the CS for each blue trial presented after the final pairing of the noise, excluding the first unpaired trial after the last noise. As highlighted by (Lonsdorf et al., 2017) it is important to control for differences at preceding phases of conditioning paradigm, therefore we employed analysis of covariance (ANCOVA) with AUCg as the dependent variable, group (ADHD vs. ADHD+CD) and in independent variable, while controlling for differences between groups in conditioning ability at the acquisition phase.

Pearson's r correlations (and Spearman's rho where necessary) and multiple regression examined the effect of clinical characteristics on conditioning ability.

Analysis were carried out using SPSS 20.0 (SPSS inc., Chicago, IL).

5.3. Results

Table 5.1 presents demographic and clinical characteristics of included participants of the sample. As expected, groups differed in CD severity ($p < .01$), where ADHD+CD participants scored higher in CD severity than those with ADHD alone. They also differed in Impulsive-Irresponsible traits ($p = .02$), but not in CU traits, age, IQ, SES or ADHD severity (all $ps > .05$). Due to evidence of an association between age and fear conditioning ability found in a large longitudinal study (Gao et al., 2010a) it was decided to run our analyses relating to differential conditioning of the CSs and differences in rate of extinction additionally controlling for age.

Table 5.1. Demographic and clinical characteristics of the sample

	ADHD+CD (n = 14)	ADHD (n = 17)	P value
Age	13.6(1.9)	14.7(2.0)	.14
IQ _{Wasi}	84.7(12.1)	91.2(11.7)	.14
% female	7.1	5.9	.89
SES			.38
% Low	53.8	29.4	
% Medium	30.8	52.9	
% High	15.4	17.6	
CD _{Dawba}	6.3(2.6)	1.2(2.1)	<.001
ADHD _{Dawba}	12.8(4.2)	11.2(6.1)	.47
CU _{YPI}	34.9(7.3)	31.4(7.7)	.22
ImIrr _{YPI}	46.4(9.0)	39.3(5.5)	.02

Note: Means are presented with standard deviations in brackets (except where indicated otherwise). Both ADHD_{Dawba} and CD_{Dawba} represent number of symptoms and are restricted to the ADHD groups. IQ is also restricted to ADHD groups. CU_{YPI} is the CU subscale for the YPI. *Key:* ADHD = Attention Deficit Hyperactivity Disorder. CD = Conduct Disorder. CU = Callous Unemotional. ImIrr = Impulsive-Irresponsible DAWBA = Development and wellbeing assessment. IQ = Intelligence quotient (2 subtest WASI). SES = Socioeconomic Status. YPI = Youth Psychopathy Inventory.

5.3.1. Probes

A mixed model ANOVA was conducted on skin conductance responses to the red and blue slides during the habituation phase to make sure there was no difference in baseline SCR between groups before the US had been administered. We found no effect of CS type ($F(1, 25) = 3.31, p = .06, \eta^2 = .12$) meaning there was no difference in SCRs to the different colours during baseline. There was also no effect of group ($F(1,25) = .13, p = .97, \eta^2 = .01$) nor an interaction between CS and group ($F(1,25) = .23, p = .78, \eta^2 = .01$).

When examining box and whisker plots for SCRs to the US a single outlier was revealed, this participant (who was in the ADHD alone group) was therefore excluded from the following analyses. A mixed model ANOVA was used to determine if there were any differences in SCRs to the 10 probes (US) across the groups. A main effect of time (time referring to the differing SCRs over the different US presentations) was found ($F(9, 252) = 24.6, p < .001, \eta^2 = .47$) reflecting a significant habituation effect across the US presentation. No main effect of group ($F(1,28) = 3.1, p = .06, \eta^2 = .13$) nor interaction between group and time were found ($F(9, 252) = .84, p = .47, \eta^2 = .03$) indicating that both groups found the US equally aversive.

Due to the overall difference between groups in SCR to the US overall approaching significance, this difference was explored further by examining the SCR to each US using independent samples t-tests. As is clear from Figure 5.1, differences were only present during the second acquisition phase and not the first. Mean SCR to the US was therefore calculated for the first and second acquisition phase separately. Following this, one-way ANOVAs revealed that these means were not significantly different between groups in acquisition 1 ($F(1, 28) = 1.3, p = .27$,

$\eta^2 = .04$) but were significantly different in acquisition 2 ($F(1, 28) = 7.9, p < .01, \eta^2 = .22$). Due to differences in SCR to the US at acquisition 2, differences in conditioning at acquisition 2 were run with and without controlling for SCR to the US at acquisition 2.

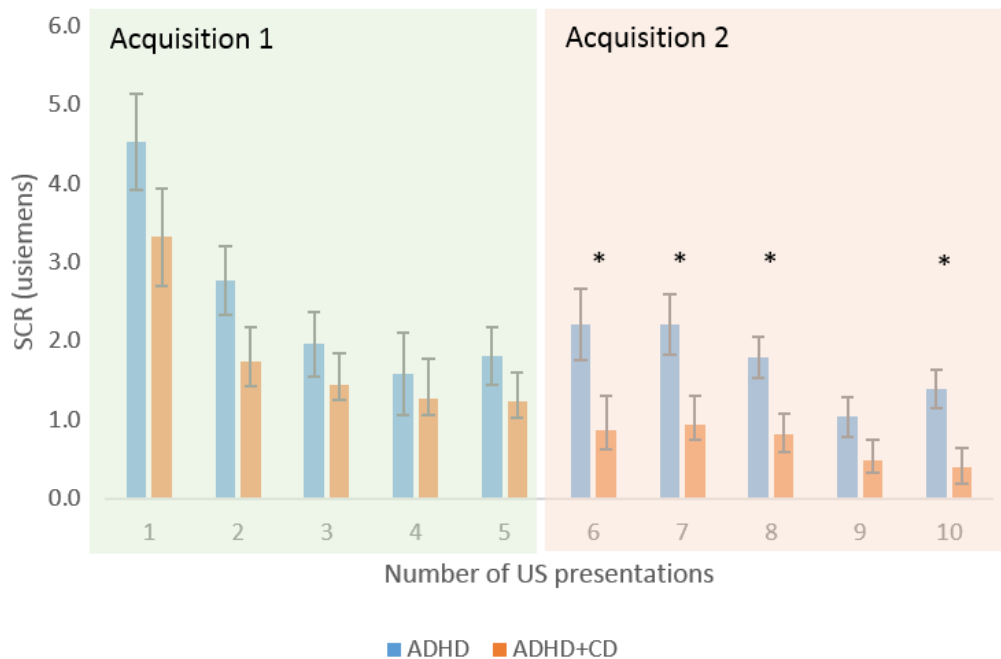


Figure 5.1. Skin conductance responses to the Unconditional Stimulus (noise). Error bars represent ± 1 Standard error. * $p < .05$

5.3.2. Acquisition

Difference scores between the CS+ and CS- were calculated to compare conditioning ability between groups. There was no significant effect of group at either the habituation ($F(1,28) = .04, p = .84, \eta^2 = .001$) or extinction ($F(1, 28) = 2.8, p = .11, \eta^2 = .10$) phase. There was also no significant difference between groups at these phases when additionally controlling for age ($ps > .05$). There was a significant difference between groups at the first acquisition phase ($F(1, 28) = 5.3, p = .03, \eta^2 = .16$) where the ADHD+CD group showed a smaller difference in SCR between the two CS types (see Figure 5.2) than the ADHD group, with the effect becoming stronger when controlling for age ($F(1,27) = 6.5, p = .02, \eta^2 = .19$). The difference

between groups at acquisition phase two was not significant ($F(1,28) = 1.1, p = .32, \eta p^2 = .04$), or when controlling for mean SCR to US at acquisition 2 ($F(1, 27) = 0.28, p = .60, \eta p^2 = .01$), or after additionally controlling for age ($F(1,26) = .04, p = .85, \eta p^2 = .01$).

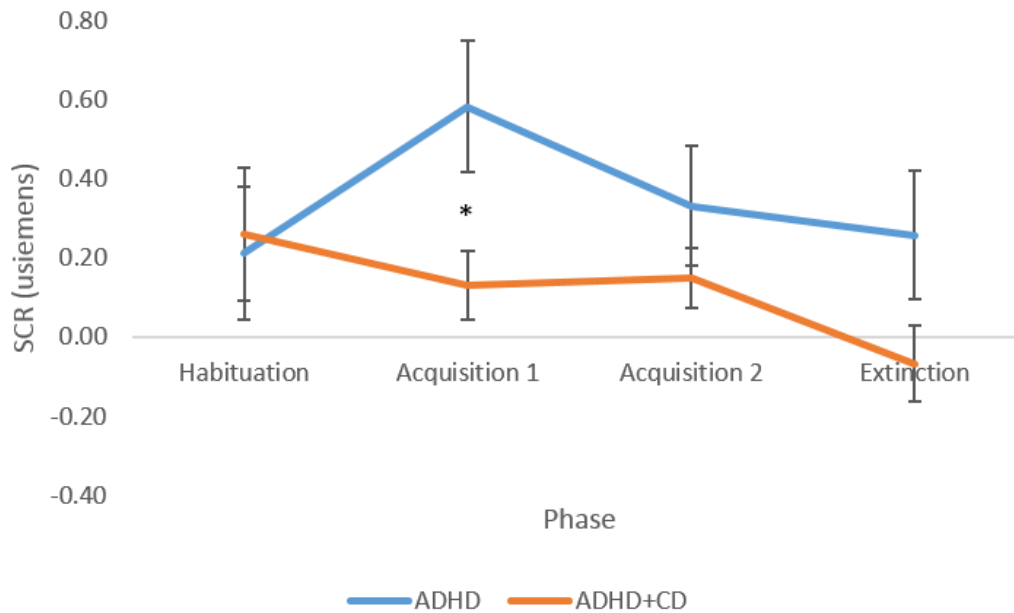


Figure 5.2. Differential SCR responding to CS+ and CS- between groups. Error bars represent +/-1 Standard error. * $p < .05$

Despite the above results showing that there was a larger difference between the groups at acquisition phase one, it is clear from Figure 5.3 (panel B) that the ADHD+CD group showed some differential conditioning to the CS+ during the first acquisition phase, while the difference between CS+ and CS- during the second acquisition phase was minimal. To test this statistically we ran paired sample t-tests for acquisition phase 1 and 2 for each group separately. For the ADHD group, SCRs to the CS+ were significantly higher than the CS- for both the first and second acquisition phase ($t(15) = 4.2, p = .001$ and $t(15) = 3.1, p = .02$, respectively). For the

ADHD+CD group the difference between CS+ and CS- was only significant at acquisition phase 1 ($t(13) = 2.5, p = .03$) and not phase 2 ($t(13) = 2.0, p = .07$).

Chapter 5: Fear Conditioning and extinction

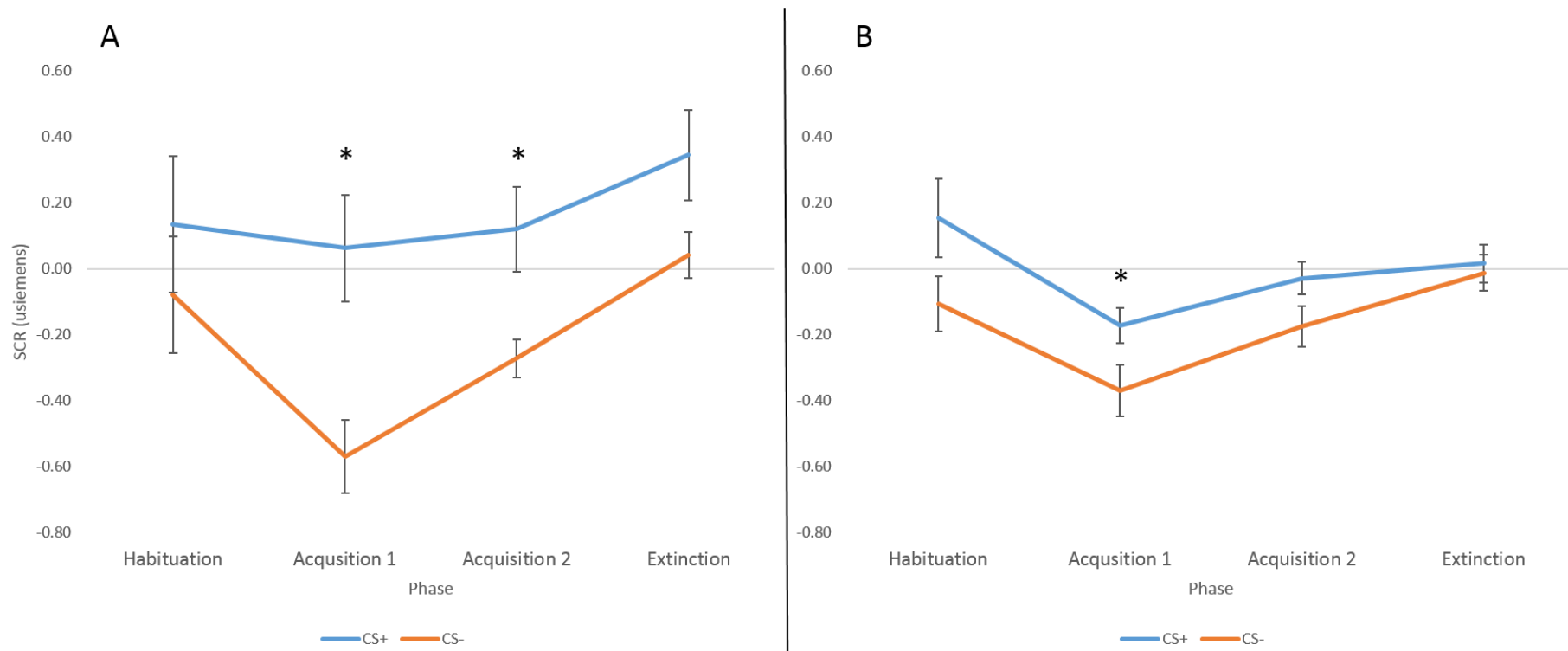


Figure 5.3. Mean SCR to the CS+ and CS-. ADHD alone are shown in Panel A. ADHD+CD are shown in Panel B. Error bars represent +/-1 Standard Error. * $p < .05$

5.3.3. Rate of Extinction

After calculating AUC_g and examining box and whisker plots, two further outliers were revealed, and we subsequently excluded these from the subsequent rate of extinction analysis (both were ADHD alone participants). Figure 5.4 presents mean SCRs to each CS+ presentation during the extinction phase. Participants in the ADHD group had a significantly larger AUC_g ($M = 1.4$, $SE = .50$) than ADHD+CD participants ($M = -0.11$, $SE = .27$; $F(1,26) = 4.9$, $p = .04$, $\eta p^2 = .16$). Crucially there was also a significant difference between groups while additionally controlling for differences between groups in conditioning ability during the acquisition phase overall ($F(1, 25) = 4.5$, $p = .04$, $\eta p^2 = .15$), while the same results held when controlling for acquisition 1 and 2 separately ($F_s(1,25) > 4.7$, $p_s < .04$, $\eta p^2_s > .17$). Additionally controlling for age reduced the effect to a trend towards significance ($F(1,24) = 3.7$, $p = .066$, $\eta p^2 = .13$).

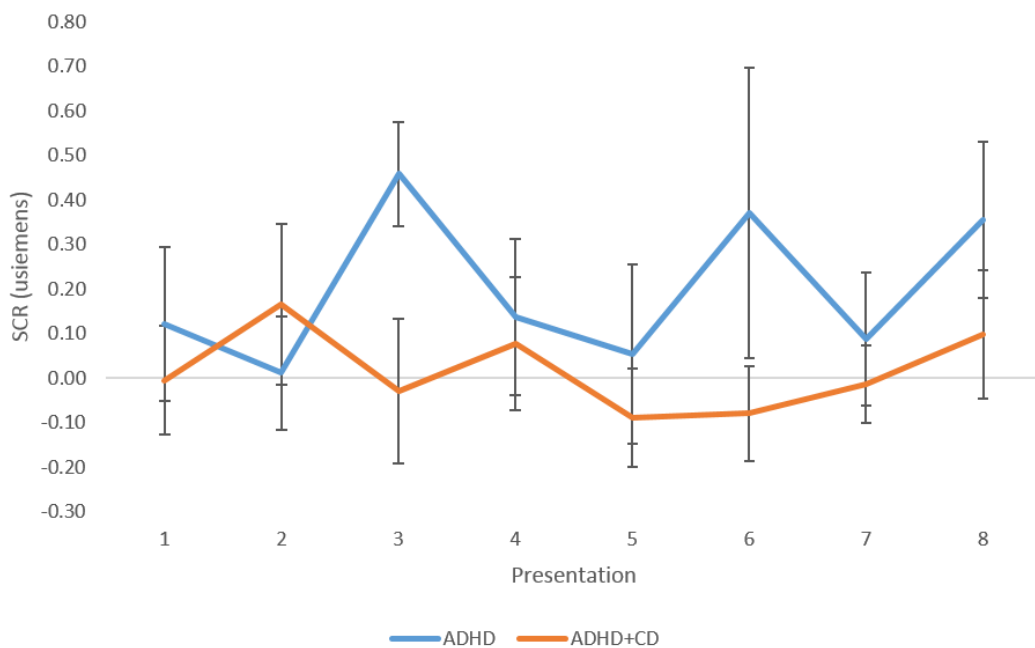


Figure 5.3. Mean SCRs to each CS+ presentation during the extinction phase. Error bars represent +/-1 Standard error

5.3.4. The Relationship between Clinical Characteristics and Conditioning Ability

Table 5.2 displays the relationships between clinical characteristics and performance on the fear conditioning task. CD severity, CU traits and Impulsive-Irresponsible traits were separately negatively correlated with differential conditioning during the first acquisition phase. We entered CD severity, CU traits and Impulsive-Irresponsible traits into a regression model and found that while the model overall was not significant, Impulsive-Traits were the only factor that significantly predicted differential fear conditioning ($\beta = -.56, p = .04$), while the other characteristics were not significantly related ($ps > .05$). Interestingly, when adding all factors into the model together, the previously negative correlation between CU traits and differential fear conditioning became positive ($\beta = .35, p = .21$), albeit non-significantly.

Table 5.2. Correlation matrix of clinical and conditioning variables

	1	2	3	4	5	6	7	8	9
1. CD _{Dawba}	-								
2. ADHD _{Dawba}	.25*	-							
3. CU _{YPI}	.41**	.23	-						
4. ImIrr _{YPI}	.46**	.15	.55***	-					
5. Hab	.16	-.22	.19	.24	-				
6. Acquisition1	-.30*	-.15	-.33*	-.36*	.11	-			
7. Acquisition2	-.22	.09	-.10	0.23	-.21	.51**	-		
8. Acquisition all	-.31*	-.05	-.26	-.34*	-.04	.89***	.85***	-	
9. Extinction	-.02	.07	-.04	.07	-.06	.23	.28	.28	-
10. RoE _{AUCg}	-.32	.03	.03	-.17	-.16	.04	.11	.09	.36

Note: * $p < .05$; ** $p < .01$; *** $p < .001$; CU_{YPI} = Callous Unemotional; GM_{YPI} = Grandiose/Manipulative; ImIrr_{YPI} = Impulsive Irresponsible; Hab = Habituation; CD_{Dawba} = Conduct Disorder severity from DAWBA; ADHD_{Dawba} = ADHD score from DAWBA; RoE_{AUCg} = Rate of Extinction as determined by Area under the Curve with respect to ground.

5.4. Discussion

In the present study we investigated differences in fear conditioning between adolescents with ADHD and comorbid CD and those with ADHD alone. As a secondary aim, we sought to determine if any observed deficits related to any particular psychopathic trait or were a function of CD severity. This study was also the first to explore differences in rate of extinction between those with ADHD and CD, and those with ADHD alone. In relation to the first aim, and partially consistent with predictions, we found that adolescents with ADHD and CD displayed significantly reduced differential SCR responding to the CS types compared to the ADHD alone group during the first acquisition phase. These findings are consistent with data from adolescent (Fairchild et al., 2010, Fairchild et al., 2008; Gao et al., 2010a; Gao et al., 2010b; Syngelaki et al., 2013; van Goozen et al., 2016) and adult studies (Birbaumer et al., 2005; Flor et al., 2002) of those with antisocial behaviour problems. Together, with the above findings, they suggest that impairments in fear conditioning results in increased risk of antisocial behaviour across age groups and across diagnostic categories. In addition, as fear conditioning is often taken as a proxy for amygdala function, our findings are consistent with the idea that the amygdala is impaired in individuals with CD (Fairchild et al., 2011).

Our findings support the theory that impairments in fear conditioning predisposes the individual to the development of antisocial behaviour. A lack of fear conditioning means that for those with CD, situations that would ordinarily be associated with punishment and therefore evoke anxiety are less likely to be avoided. Our findings of deficient fear conditioning in those with additional CD are consistent with fearlessness accounts of antisocial behaviour (Raine, 2002; van Goozen et al., 2007).

In terms ADHD, as was the case in van Goozen et al., (2016) and other studies of autonomic and amygdala responses comparing those with ADHD and comorbid CD against ADHD alone (Herpertz et al., 2008; Pliska, Hatch, Borcharding, & Rogeness, 1993), our findings suggest that emotional learning impairments are specifically associated with those with additional CD. This may help explain why this subgroup of ADHD children develops antisocial behaviour and are clinically more severe in both prognosis and persistence of problems (Thapar, Langley, O'Donovan, & Owen, 2006).

Consistent with other studies of fear conditioning in adolescents with CD, we found no relationship between impairments in fear conditioning and number of CU traits (Fairchild et al., 2008; Northover, 2015). Interestingly, the majority of studies of fear conditioning in adult psychopaths find that impairments are associated with primary psychopathy, or the affective dimension of psychopathy (Birbaumer et al., 2005; Lykken, 1957; Veit et al., 2013), of which CU is the childhood equivalent. Instead of CU traits, we found that such impairments were negatively related Impulsive-Irresponsible traits. These traits are also a dimension of psychopathy, but instead of the lack of empathy and remorselessness represented by CU traits, Impulsive-Irresponsible traits characterise those who are impulsive and thrill-seeking (Andershed et al., 2002). As far as we are aware, this is the first study to find such a relationship in adolescents with ADHD and CD, and therefore the results will require replication. However, our findings are consistent with a recent study in the adult psychopathy literature (Schultz et al., 2016). Shultz and colleagues found that the ability to learn the association between a neutral and an aversive stimulus was intact in primary psychopaths, but not in secondary psychopaths. It is these secondary psychopaths that are characterised by impulsiveness. Interestingly, in the current

study, when both Impulsive-Irresponsible traits and CU traits were entered into a model together, Impulsive-Irresponsible traits were negatively related to differential fear conditioning, why the remaining unique variance associated with CU traits (albeit non-significantly) was positively related to differential fear conditioning.

It is difficult to reconcile the differences in findings between the present study and those which find fear conditioning deficits are related to the affective-interpersonal dimension in the adult psychopathy literature. However, most studies on fear conditioning in adult psychopathy are conducted on unsuccessful (incarcerated) adult psychopaths. There is evidence that unsuccessful psychopaths are more impulsive and irresponsible than successful psychopaths (Yang et al., 2005). Thus, it is possible that fear conditioning impairments are more typical of unsuccessful psychopaths. There is evidence that, relative to unsuccessful psychopaths, successful psychopaths do not show structural and functional impairments in the amygdala (Yang, Raine, Colletti, Toga, & Narr, 2010), the brain region heavily involved in intact fear conditioning. If indeed true, it would follow that successful psychopaths would not show such impairments.

In addition, we found that ADHD+CD participants' SCR to the US during the later acquisition phase was significantly lower than that of ADHD alone, this could suggest that either ADHD+CD are faster to habituate to US, or the ADHD alone group demonstrate a failure to habituate to the US, however without a typically developing control group one cannot determine which would be a more typical response during this paradigm. Previous evidence has found a similar pattern of results when comparing CD and control participants (Fairchild et al., 2010). Other studies have also found that reduced startle as measured by eye blink magnitude was related to severity of CD symptoms (Syngelaki et al., 2013) which suggests the

current findings are indicative of an atypical increased attenuation to the US in CD participants.

In relation to our third aim, and in line with our hypothesis, we found that adolescents with ADHD and CD displayed a faster rate of extinction in their SCR than those with ADHD alone, and importantly, that this relationship also held after controlling for differences between groups during the acquisition phase of conditioning. This finding is in line with the suggestion by Syngelaki et al., (2013) that the reduced differential responding to the CS by the late acquisition phase in their young offender sample may have been the result of expedited rate of extinction. Interestingly, in our study, although we only found a difference between groups at the first acquisition phase in differential conditioning, the ADHD with CD group did show significant differential responding to the CSs in the first acquisition phase, but this differential responding had disappeared in acquisition phase two. This suggests that there is some US-CS pairing learning in those with ADHD+CD (albeit significantly less than in those with ADHD alone) in the early acquisition phase, but they already start to lose this association by the later acquisition phase. This is in line with some evidence that those at genetic risk of antisocial behaviour have lower fear memory consolidation (Lonsdorf & Kalisch, 2011).

The finding of an expedited rate of extinction in those with ADHD and CD raises questions about the functioning of the brain regions that are involved in fear extinction in those with CD. Along with the amygdala, which is also involved in fear conditioning and has already been shown to be dysfunctional in those with CD, the hippocampus, paralimbic and infralimbic cortices also play a role in fear extinction (Graham & Milad, 2011). Activation in the paralimbic cortex stimulates the amygdala, leading the fear response. In those with anxiety disorders, extinction

failure is associated with the persistence of the paralimbic response after extinction training (Burgos-Robles, Vidal-Gonzalez, & Quirk, 2009), therefore it would follow, that those with CD who are characterised by low anxiety, would show reduced activity in the paralimbic area leading to a faster rate of extinction. Indeed, there is evidence that activity in the dorsal anterior cingulate cortex (dACC), an area functionally analogous to the paralimbic cortex (Graham & Milad, 2011), is significantly underactive in those with CD (Stadler et al., 2007). Since activation in the paralimbic cortex stimulates the amygdala leading to a fear response (Corcoran & Quirk, 2007), this could explain why the rate of extinction was faster in those with CD, even after controlling for acquisition differences. Another area involved in extinction learning is the infralimbic cortex. Activation of the infralimbic cortex inhibits the amygdala, preventing a conditioned response. Therefore, our finding of a faster rate of extinction in those with CD could suggest over-activity in this region, which would lead to faster inhibition of the amygdala. As yet, no study has examined activity in the infralimbic cortex in those with CD. Interestingly, the infralimbic cortex has previously been implicated in aggression. In a rodent study, Halász, Tóth, Kalló, Liposits, and Haller (2006) found that violent acts committed by rats towards an intruder were accompanied by an increase in activity in the infralimbic cortex. Of course, as with other findings from the present study, this is the first study which has found a faster rate of extinction in those with ADHD and CD, therefore replication is required. In addition, it is clear that the current study was not designed to answer questions about functioning in either the paralimbic or infralimbic cortex during fear conditioning or extinction, therefore future studies should explore this.

Our study was the first to explore rate of extinction as opposed to overall

difference in SCR during the extinction phase between groups. Studies that look at mean differences in SCR over the extinction phase as a whole, typically find no differences between groups (e.g. Fairchild et al., 2010, 2008; Syngelaki et al., 2013); therefore, future studies should also employ methods to explore rate of extinction to test if our findings can be replicated.

The findings of the current study have important clinical implications. In line with van Goozen et al., (2016), we found further evidence that emotion processing impairments in those with ADHD are specifically present in those with additional CD. Although requiring replication, evidence that extent of fear conditioning impairments are associated with Impulsive-Irresponsible traits underlies the need to take into account individual differences among those displaying antisocial behaviour when establishing programmes to tackle behavioural problems. As has been emphasised in the past (van Goozen & Fairchild, 2008), the fact that those displaying antisocial behaviour fail to learn from aversive outcomes implies punishment is clearly not an effective strategy to bring about behaviour change in such individuals. Nevertheless, the fact that we found that those with ADHD and CD do show some (but reduced) fear conditioning ability, along with studies showing that intact fear conditioning is a protective factor against developing antisocial behaviour (Raine, 1996), implies that future experimental studies may want to explore the conditions under which successful fear conditioning can be evoked in those with CD. There is evidence to suggest that fear (conditioning) deficits very early in development precede aggression and criminal behaviour (Baker, Shelton, Baibazarova, Hay, & van Goozen, 2013; Gao et al., 2010; Gao et al., 2010). Future research in young children at risk of later antisocial or aggressive behaviour should seek to develop

ethical and age appropriate fear conditioning paradigms to assess fear learning ability at a younger age.

A strength of our study was that all participants had a clinical diagnosis of ADHD and were grouped into those who did or did not have additional CD, which allowed us to determine in which domains those with CD have additional impairments. ADHD is typically associated with impulsivity, however we found no relationship between ADHD severity and fear conditioning impairments, nor had those with ADHD and CD significantly more severe ADHD symptoms, highlighting that the relationship with Impulsive-Irresponsible traits was not a confound of ADHD severity. Nevertheless, our study could have been improved further with the addition of a typically developing control group and a group of adolescents with CD without comorbid ADHD. This would allow us to determine if those with ADHD without CD show any impairments in fear conditioning, while having a CD alone group would allow us to determine if those with combined CD and ADHD display more or less impairments relative CD alone.

Despite our study using a well replicated fear conditioning paradigm, future studies should explore the use of paradigms that make use of aversive stimuli that are ecologically more similar to the types of natural stimuli involved in the socialisation of children and adolescents. Tada et al., (2015) recently presented a promising paradigm whereby the aversive US was a social/verbal punishment. Not only is this more ecologically relevant to the type of aversive stimulus that would follow an antisocial act, but it was also shown to successfully bring about a conditioned response. However, as this is the only study to use this paradigm to date, future studies should explore the replicability of these findings and the paradigms ability to evoke a UR in those displaying antisocial behaviour.

In addition, using a small sample we were able to replicate findings that those with ADHD and CD have deficits in the acquisition of the fear response (van Goozen et al., 2016), further providing support for this finding. However, because of our small sample, replication with larger sample sizes in relation of our novel findings of Impulsive-Irresponsible traits negatively predicting fear acquisition and ADHD and CD showing an expedited rate of extinction will be required to gain more confidence in the generalisability of these.

In conclusion, we found further evidence that those with ADHD and comorbid CD compared to those with ADHD alone have impairments in fear conditioning. The impairments found in the ADHD+CD group in this chapter are in line with the emotional processing impairments observed in this group in the other experimental chapters. In addition, we found that fear conditioning impairments were significantly and positively associated with Impulsive-Irresponsible traits and not with CU traits. Finally, whilst controlling for acquisition differences, examination of the SCR responses during the extinction phase indicated that those with ADHD and CD displayed an increased rate of extinction. This suggests that participants with CD retain punishment information in response to a behaviour for a shorter time. However, due to the novelty of these findings, replication is encouraged.

6. Chapter 6: General Discussion

6.1. General Discussion

Chapter 1 of this thesis highlighted the overlap between two costly disorders found in childhood and adolescence, outlining how emotion processing impairments have been found in both groups, and the uncertainty, because of the high comorbidity, as to whether impairments were driven by either of the disorders, or were a problem common to both. It also highlighted how current research still did not understand what was driving such impairments, and highlighted attentional impairments as one potential candidate. As a result, the central aim of the current thesis was to explore various components of emotion processing in adolescents with a diagnosis of ADHD and divided into groups based on whether or not they met additional criteria for a diagnosis of CD. It was particularly interested in impairments in the ability to process distress related stimuli, such as fear, and to explore the role of attentional mechanisms in explaining such differences. Due to the heterogeneous nature of ADHD, it also sought to reveal which clinical characteristics were driving impairments in emotion processing. The overarching aim of highlighting in which groups impairments in emotion processing are apparent and what is driving them was to inform future intervention in those affected; to reduce conduct problems in those already displaying such behaviours, and risk for those who show emotion impairments but are not yet showing associated problem behaviours.

The thesis did this by investigating performance on a range of emotion processing tasks. Chapter 2 explored facial emotion recognition, while Chapter 3 investigated both cognitive and affective empathy in response to emotion inducing film clips. In both these chapters, participants' eye gaze was measured with an eye-tracker to determine where participants focused their attention while being engaged in emotion processing tasks. Chapter 4 presented and discussed the development of a

novel pictorial emotional Stroop task (CEPS), which measured emotion interference by irrelevant emotional distractors. In Chapter 5 participants' ability to learn the association between an aversive and neutral stimuli was assessed while also exploring the speed at which they lost such an association. Additionally, in all four experimental chapters the thesis explored the contribution of both CD and ADHD symptom severity and CU traits to performance in emotion processing.

The current chapter will describe and discuss the key findings of the research and how they relate to previous work. It will then highlight the strengths and weaknesses of the current study and offer some suggestions as to the direction research should take in light of the current findings.

6.1.1. Emotion Recognition

Impairments in emotion recognition have been found in a variety of populations that display problems with antisocial behaviour (Blair et al., 2005; Bowen et al., 2014; Dadds et al., 2006), including those with CD (Fairchild et al., 2010, 2009). However, a problem with the past research into recognition impairments, particularly in adolescence, was their failure to control for comorbid ADHD, which commonly co-occurs in CD (Biederman, 2005) and young offenders more generally (Young et al., 2015). It is important to control for ADHD because research has demonstrated that individuals with ADHD also show deficits in emotion recognition (Pelc et al., 2006; Singh et al., 1998). It was unclear whether deficits observed in those with ADHD are a result of their ADHD symptomology or their co-morbid CD (e.g. Schwenck et al., 2013). Therefore it was crucial to measure emotion recognition deficits in a group of ADHD participants, divided into whether they did or did not have additional CD, while also comparing performance to a group of typically developing controls, in order to determine in which groups these impairments are apparent. A further issue

was to ascertain whether emotion recognition impairments are general and exist across emotions (e.g. Dawel, O’Kearney, McKone, & Palermo, 2012), are specific to negative emotions (Bowen et al., 2014; Marsh & Blair, 2008), or are even more specifically only found for fear faces (Adolphs, 2002). Additionally, it is unclear if such deficits are driven by the severity of CD (Fairchild et al., 2009) or ADHD, or related to the existence of CU traits (e.g. Woodworth & Waschbusch, 2008). We explored the role of attentional gaze in these groups, and its contribution to emotion recognition performance.

The above issues were addressed in Chapter 2 of the current thesis, and to the best of our knowledge, this was the first study to compare the above three groups in emotion recognition performance whilst also measuring eye gaze. Partially in line with predictions, it was found that participants with ADHD and comorbid CD were specifically impaired in the recognition of both fear and neutral faces compared to ADHD alone and typically developing controls, while participants with ADHD alone performed similarly to controls. On the basis of the hostile attribution bias (Dodge & Newman, 1981) we also predicted that the ADHD and CD group would be impaired in neutral recognition because they would interpret these faces as angry, but the current study found no evidence of this. However, we did find evidence that this group was more likely to misinterpret fear faces as depicting anger.

When examining the eye gaze patterns of the three groups, we found that although both ADHD groups spent less time focusing on the eye-region of the face compared to healthy controls, the ADHD and CD group spent no less time focusing on the eye region than the ADHD alone group. This suggests that impairments in focusing on the eye region is a problem for ADHD in general. Further, the finding that percentage of time spent focusing on the eyes was positively correlated with

emotion recognition accuracy, along with the lack of difference in percentage of time spent looking at the eye region between the ADHD groups, suggests that although looking at the eyes is important for successful recognition, other mechanisms must be involved in the recognition impairments observed in those with ADHD and CD. Together these results suggest that impairments in the recognition of fear is specific to additional CD, while impairments in looking towards the eyes is more general to ADHD. This suggestion was further supported by the results of the dimensional analysis of symptom severity where it was found that emotion recognition was negatively related to CD severity and (less strongly to) CU traits but not to ADHD severity, while time spent looking at the eyes was negatively related to ADHD severity and not to CD severity or CU traits.

6.1.2. Empathy

The uncertainty surrounding emotion recognition impairments in those with ADHD alone and/or comorbid ADHD and CD is also apparent for the domain of empathy. Some studies found empathy impairments in ADHD generally (Braaten & Rosen, 2000; Dyck et al., 2001), while others found they were related specifically to CD (Anastassiou-Hadjicharalambous & Warden, 2008; Cohen & Strayer, 1996; van Goozen et al., 2016). It is also unclear if impairments are specific to cognitive empathy, or affective empathy or to both. Crucially, a recent study found that impairments in affective empathy only, were predictive of the conduct problems shown in those with ADHD (van Goozen et al., 2016). But again, as was the case with emotion recognition, it is unclear how much these impairments can be explained by a lack of attention to the eye region of the face, while a consensus is still far from being reached as to the relative contributions of CD and ADHD symptom severity and CU traits to these impairments.

These issues were addressed in Chapter 3 of this thesis, by examining cognitive and affective empathy in those with ADHD alone and ADHD with comorbid CD (this chapter had no control group) in response to emotion inducing film clips. As with Chapter 2, participants' eye gaze was also measured while they viewed the clips. In line with the recent findings of van Goozen et al., (2016) it was found that ADHD and comorbid CD participants had a specific impairment in affective empathy in comparison to ADHD alone participants. Against predictions, we found that this impairment was a general one across positive (happy) and negative (sad and fear) valenced clips. Interestingly, although we predicted that such an impairment would be most pronounced for fear, the difference between groups was smallest for this emotion. In addition, it was found that ADHD and comorbid CD participants had less of a preference to look at the eyes than ADHD alone participants across clips.

When the above differences between groups in empathy and attention were explored further in relation to clinical characteristics, some interesting relationships were uncovered. Firstly, the lower affective empathy, specifically for negative emotions (sad and fear), in the ADHD and CD group was found to be mediated by CU traits, such that the differences between the groups were explained by the increased number of CU traits in the ADHD and CD group. However, this was not the case for the reduced experience of affective empathy for happiness observed in this group. Instead, this was found to be negatively related to severity of CD symptoms (and not CU traits) such that the more severe the individual's conduct symptoms the less they experienced affective empathy during the happy clips. A novel interaction also emerged when exploring this further. It was found that severity of ADHD symptoms moderated the relationship between CD severity and affective

empathy for happiness; there was only a significant negative relationship in participants with low to median levels of ADHD severity, whereas there was no relationship in those with high ADHD severity. One can tentatively speculate that the reasons for those with CD presenting with antisocial behaviours may differ between those with low and high levels of ADHD (although it must be kept in mind that all participants had at some point been diagnosed with ADHD). The chapter also explored the clinical characteristics that explained the impairment in looking towards the eyes in the ADHD and CD group, and found that this was explained by severity of ADHD as opposed to CD severity or CU traits. In addition, preference to look towards the eyes was not related to affective empathy, but there was some evidence to suggest it predicted cognitive empathy for happy film clips. For sad emotion clips specifically, regression analyses indicated that CU traits negatively predicted affective empathy over and above both eye-preference and CD and ADHD severity, highlighting that while attentional deficits may be related to ADHD symptoms severity, they do not explain empathy impairments.

6.1.3. Emotion Regulation

In Chapter 4, we investigated emotion regulation in ADHD alone and those with ADHD and CD. First, it was highlighted how the emotion regulation literature is currently overly dependent on self-report measures and lacks a reliable and valid objective measure. The chapter therefore highlighted a component of emotion regulation, namely attention, where an objective measure could be appropriately developed. Following reviewing the flaws of previous measures, a new measure was developed and it was tested in experiment 1 on undergraduate students. The aim was to create a paradigm in which responding to a target task was equally difficult for each condition, while also creating an emotional congruency effect akin to a true

Stroop effect, such that participants were faster to respond to congruent emotional target/distractor pairs than they were to incongruent emotional target/distractor pairs. On this count experiment 1 was successful. Participants were faster to respond when the target and distractor matched in terms of their valence compared to when they did not match, suggesting that the paradigm produced automatic processing of the irrelevant emotional distractors, and that the two target conditions were effectively depicting positive and negative valence. Further, the general interference effects in the presence of emotional distractor conditions compared to object distractor conditions was explored. It was found that emotional distractors significantly facilitated responses compared to the presence of object distractors (in other words they were faster to respond in the presence of an emotional distractor compared to that of an object), and follow-up tests revealed that this pattern of results was driven by fearful faces.

Following the successful application of this paradigm in undergraduates, the second experiment of Chapter 4 applied this task to a small group of adolescents with ADHD alone and ADHD with CD. It was found that across congruency and emotional conditions, participants found responding to the target aspect of the task equally difficult. When examining the effect of congruency, interestingly the performance of ADHD alone participants was in the same direction as that of the undergraduates, in that they responded faster to congruent than to incongruent trials. ADHD and CD participants, on the other hand, showed the opposite pattern of results, failing to show a facilitation effect in the presence of congruent trials. When exploring the specific trial conditions which brought about this difference between groups further, it was found that this was mainly driven by trials in which the target appeared on the eye location of the irrelevant distractor. When exploring the effect of

emotional vs. object distractors, a difference between groups was found specifically when targets were presented over the eyes of a fear face, such that ADHD alone participants were facilitated, while ADHD with CD participants showed interference. These findings suggest that attentional mechanisms involved in the automatic processing of emotions of others, which contributes to successful emotion regulation (Gross, 1998; Shaw et al., 2014), may be impaired in those with ADHD and CD. When exploring the effect of the clinical characteristics, strong relationships were found between CD severity and interference scores when targets were presented over fearful eyes, as indicated by the large effect sizes; however, due to the small sample size not all correlations reached the required significance level.

6.1.4. Fear Conditioning

The final experimental chapter of the thesis examined differences between ADHD alone and ADHD with CD adolescents in the acquisition of an association between an aversive and neutral stimulus and the speed at which this association was lost again, as measured by SCRs. Given the inconsistency about the role of CU (the interpersonal affective dimension of psychopathy) traits in these deficits in the adult and adolescent fear conditioning literature, we also explored how conditioning related to CU and impulsive-irresponsible traits, as well as CD and ADHD severity.

The chapter first demonstrated, consistent with van Goozen et al., (2016), that participants with ADHD and CD showed reduced differential fear conditioning compared to ADHD alone (specifically in the early acquisition phases). Second, for the first time it was shown that ADHD and CD participants showed a faster rate of extinction of the conditioned response, even when controlling for the differential conditioning apparent during the acquisition phases. Interestingly, this pattern of results is the opposite to that found in those with anxiety disorders (Milad et al.,

2008, 2013) who fail to extinguish the relationship between the neutral and aversive stimulus when these are no longer paired. This finding was further supported by the fact that ADHD and CD participants demonstrated some (although reduced in comparison to ADHD alone) differential fear conditioning in the early acquisition phase but no difference in the later phase. Together these findings suggest that fear conditioning and extinction abnormalities are not present in ADHD alone, but are specific to additional CD. In line with other studies on antisocial adolescents, no relationship between CU traits and fear conditioning deficits was observed (Fairchild et al., 2010, 2008), while we found that impulsive-irresponsible traits were associated with reduced fear conditioning ability over and above CD severity. Consistent with a recent study in the adult psychopathy literature, this set of results suggests that fear conditioning impairments are specific to those who are highly impulsive (secondary psychopathy; Schultz et al., 2016) as opposed to those who have interpersonal affective problems characteristic of primary psychopaths. Interestingly, when impulsive-irresponsible traits and CU traits were entered into a model together, the direction of relationship between CU traits and fear conditioning reversed such that higher CU traits was associated with better fear conditioning. Having outlined the key findings of the thesis, the next section will attempt to integrate the findings of the chapters by drawing on key themes and their relation to integral theories in the literature.

6.1.5. Is Emotion Processing Impaired in Combined ADHD and CD Compared to ADHD Alone?

A key aim of the current thesis was to explore whether impairments in emotion processing are general to ADHD or specific to those with additional conduct problems. While emotion recognition (Fairchild et al., 2009; Marsh & Blair, 2008),

empathy (Anastassiou-Hadjicharalambous & Warden, 2008; Cohen & Strayer, 1996; Miller & Eisenberg, 1988), fear conditioning (Fairchild et al., 2010, 2008; Syngelaki et al., 2013) and emotion regulation (Calins & Demon; Deborde et al 2015; McLaughlin et al 2011) impairments have been observed in those with CD, such impairments have also been found in those with ADHD (emotion recognition; Pelc et al., 2006; Singh et al., 1998, empathy; Braaten & Rosen 2000; Dyck et al 2001).

In all 4 chapters of the current thesis, we found evidence that those with ADHD and CD were more impaired than ADHD alone in the different dimensions of emotion processing measured. Evidence was particularly strong in Chapter 2 with the inclusions of a typically developing SES matched control group highlighting how ADHD and CD were specifically worse in fear and neutral recognition than both controls and ADHD alone groups. As a result, our findings are consistent with those that have found impairments in participants with CD (Fairchild et al., 2010, 2009) and other adolescents who display antisocial behaviour (Bowen et al., 2014), while they suggest that the results of studies that had previously found recognition impairments in those with ADHD (Pelc, Kornreich, Foisy, & Dan, 2006; Singh et al., 1998) may have been driven by comorbid CD which was unaccounted for. The current thesis' results highlight the importance of identifying and appropriately controlling for comorbid CD when conducting research on emotion processing in those with ADHD. The results of Schwenck et al., (2013), further highlight this point. In this study, despite differences between ADHD and controls in recognition initially being apparent, after the authors excluded those with comorbid CD and reanalysed the data, differences in emotion recognition were no longer observed. Similarly, strong evidence for emotion processing deficits being specific to those with comorbid CD came from Chapter 4, which showed that performance in the

ADHD and CD group on an emotional Stroop task was atypical compared to ADHD alone participants. Our results were also in line with other recent studies that had compared ADHD and ADHD with CD on other measures of emotion regulation (e.g. Melnick & Hinshaw, 2000; Northover, Thapar, Langley, & van Goozen, 2015a). Given these previous results and the nature of our new paradigm, it seems safe to conclude that those with ADHD and comorbid CD have a specific problem in automatic processing of the emotions of others, which is important for the adaptive regulation of emotion. Chapters 3 and 5 explored empathy impairments and fear conditioning and extinction impairments in both groups. The absence of a typically developing control group in these studies restricts the conclusions being drawn about the specificity of problems to one group, however both studies again found that individuals with ADHD and CD showed impairments in emotion processing relative to ADHD alone. Chapter 3 demonstrated a general deficit in affective empathy in ADHD and CD participants and was hence consistent with the recent findings of van Goozen et al., (2016) who found specific affective empathy deficits in ADHD and CD over and above ADHD alone. While our findings further suggest that those studies that found empathy impairments in those with ADHD (Braaten & Rosen, 2000; Dyck et al., 2001) resulted from the failure to account for comorbid CD. Marton, Wiener, Rogers, Moore, & Tannock (2009) found that while ADHD children were rated as less empathic than controls by their mothers, this was accounted for by comorbid CD. The findings of this thesis have gone beyond mere parent ratings and found a similar result using empathy inducing film clips. ADHD alone and ADHD with CD groups were not found to differ in cognitive empathy, but one cannot conclude that both groups showed intact performance without the availability of a typically developing control group for comparison. This was

similarly the case in Chapter 5, which examined fear conditioning and extinction. Again, consistent with van Goozen et al., (2016), ADHD and CD participants showed reduced differential fear conditioning compared to ADHD alone, while they also showed an expedited rate of extinction, highlighting that they lost the association between the neutral and aversive stimulus at a faster rate than ADHD alone even after accounting for the differences in initial conditioning between the groups. These results are in line with those that have found fear conditioning impairments in those with CD (Fairchild et al., 2010, 2008) and those that found intact fear conditioning in those with ADHD (Coffin, Baroody, Schneider, & O'Neill, 2005; Pliska et al., 1993).

Despite the different paradigms used, the varying sample sizes and availability of control groups, it is important to highlight that in all four chapters the performance of the ADHD and CD group were impaired in comparison to the ADHD alone group in emotion processing tasks. This pattern of results highlights even further how those with a combination of both disorders have a more severe prognosis (Thapar et al., 2001), with emotion processing impairments across a range of modalities being apparent. In addition, and despite not measuring executive functions in the current thesis, our findings, showing emotion processing impairments across a variety of tasks, are in line with findings of van Goozen and colleagues (2016) who found that the problems in those with ADHD and at genetic risk for conduct problems, were specifically mediated by emotion processing impairments (specifically for fear) rather than executive functioning impairments.

6.1.6. What is the Evidence that Emotion Processing Impairments are Specific to Fear Stimuli?

Another central aim of the current thesis was to determine if emotion impairments in those with additional CD are generally present and exist across emotions, or are

limited to negative emotions, and/or fear specifically. Previous research has presented a heterogeneous set of findings in relation to the issue of the specificity of emotion processing impairments in those displaying antisocial behaviour. Some theories have highlighted the important role of fear deficits in explaining such behaviour (low fear hypothesis; Lykken, 1957; Raine, 2002), while others suggest that impairments in the processing of distress cues (fear, sad, pain) are responsible for antisocial behaviours (VIM; IES; Blair, 1995; 2005). Inconsistencies in theory have been echoed by inconsistencies in the resulting original investigations exploring these deficits. Meta-analyses have found evidence that fear and sadness present a specific problem for these individuals (Marsh & Blair, 2008), while others suggest there are impairments in the processing of all emotions (Dawel et al., 2012), while still other recent research supports the idea that fear impairments are driving the antisocial behaviour of these individuals (van Goozen et al., 2016). The findings of the current thesis varied in terms of the specificity of the emotion impairments depending on the measure of emotion processing used. The measures from the current thesis can be divided into a number of types of emotion processing; (1) emotion recognition and cognitive empathy, (2) automatic processing of emotional stimuli, (3) affective empathy and (4) fear conditioning; these will be explored in turn. (1) Both emotion recognition (Chapter 2) and cognitive empathy (Chapter 3) can be considered to be measuring the conscious recognition of the emotions of others. From Chapter 2 it is clear that those with ADHD and CD show a clear deficit in the recognition of fear; however, the cognitive empathy results in Chapter 3 suggest that ADHD and CD participants were no worse than those with ADHD alone at identifying the emotions depicted by the characters in the emotional film clips, across all three emotions (happy, fear, sad). In comparison with the clear deficits

presented in Chapter 2, it is possible that the extra contextual and auditory cues present in the empathy film clips aided participants in identifying the emotion the main character was experiencing, so they could be less reliant on the facial features of the character. Considering this as an explanation for the discrepancy between the above two chapters, the results of Chapter 2 alone provide strong evidence of a fear impairment in those with ADHD and CD. Previous studies have used a larger number of emotions (e.g. Bowen et al., 2014; Fairchild et al., 2010b), which included emotions that shared some ambiguity (e.g. surprise/fear, anger/disgust). In the current study we used 5 very distinct emotions, which were less ambiguous and shared few features in terms of facial configuration. As a result, the fact that ADHD and CD participants still significantly confused fear faces with other emotions (an error made particularly often was identifying fear as anger), shows they had clear impairments in the ability to recognise static images of fear faces.

(2) As opposed to the conscious recognition of others' emotions as measured by both the emotion recognition task (Chapter 2) and cognitive empathy (Chapter 3), the emotional Stroop task (Chapter 4) explored the more automatic processing of emotional stimuli. In this study both the reduced congruency effect and the reduced facilitation by emotional stimuli in the ADHD and CD group suggests that this group had impairments in automatically processing emotional stimuli. Given that follow-up tests revealed such effects were driven by differences between groups in the processing of fear stimuli (and not happy), this suggests that this group has deficits in the automatic processing of fear stimuli. However, it must be kept in mind that only happy and fearful faces were employed in this study, and it is therefore not clear if these participants have additional impairments in the automatic processing of other negative emotions.

(3) Affective empathy, the ability to share in the emotions of another, depends on the correct identification of emotion in others in order to be able to affectively empathise with the appropriate emotion. Due to the absence of differences between groups in cognitive empathy, but the presence of differences across emotions in affective empathy, we can infer that the groups did not differ in the ability to recognise the emotions depicted by the main characters, but that the ADHD and CD participants were less able to share in these emotions. This impairment was general across sad, happy and fear film clips. When one also considers the results from the emotion recognition chapter, it would appear that ADHD and CD participants have specific impairments in the ability to recognise fear faces in others, and are impaired in the ability to share in the emotions of others more generally (in all the emotions assessed in Chapter 3 at least).

A final type of emotion processing explored was fear conditioning (4); this form of associative learning assesses the ability of an individual to produce a fear response to an aversive stimulus, and to associate it with a neutral stimulus. As is clear from the name of the task, this task only assesses fear learning specifically, and it is clear from previous research that those with CD have impairments on this task (Fairchild et al., 2010, 2008). Importantly, we found evidence of reduced differential fear conditioning and increased rates of extinction in those with ADHD and CD, while exploration of the SCRs to the aversive noises during the later acquisition phase also revealed that these participants were less responsive in this regard.

Taken together, the above patterns of results suggest that ADHD and CD participants have *specific* impairments in the conscious recognition of fearful emotional expressions, in the automatic processing of fearful expressions, and in the ability to produce a fear response and associate this response with a neutral stimulus.

However, they have a more general impairment in their ability to share in the emotions of others across both positive and negative emotions.

6.1.7. Differences in Attentional Gaze between Groups and its Contribution to Emotion Processing Impairments

Three experimental chapters explored attentional mechanisms in explaining emotion processing impairments by comparing differences between groups and examining the relationships between attention and performance on the emotion processing tasks on a continuous scale. The differences and similarities between the results of the chapters will now be examined. In Chapter 2, it was found that both ADHD groups focused less on the eye region than control groups, while there was no difference between ADHD with and without CD. Interestingly, emotion recognition was found to be positively related to the proportion of time spent looking at the eyes, but given that ADHD groups differed in emotion recognition accuracy and not overall attention to the eyes, it suggests that other mechanisms must be at play in terms of explaining recognition impairments. On the other hand, in Chapter 3 it was demonstrated that ADHD with CD participants had less of a preference to look towards the eyes than ADHD alone, while a preference to look towards the eyes was not found to be predictive of affective empathy. Despite these findings appearing discrepant, a common relationship across these chapters is that a lack of attention to the eye region was specifically related to ADHD severity. The ADHD and CD group in both chapters had significantly higher ADHD severity therefore it would appear that attentional deficits to the eye region are not the result of additional CD problems, but instead are driven by the severity of the ADHD symptoms. However, the findings from Chapter 4 (emotional Stroop task) seem inconsistent with this reasoning. In this chapter it was found that the differences between the groups in facilitation by emotional distractors were most pronounced when targets were

presented over the eye region of the face; in these instances, ADHD alone participants were facilitated and ADHD with CD showed interference when responding. This suggests that ADHD alone participants are more focused on the eye region than ADHD and CD participants – a result in line with that of undergraduates. ADHD severity can, in this case, not explain the differences between groups; the groups in this (albeit very small) sample did not differ in ADHD severity, nor was there a relation between ADHD severity and eye-interference. There must therefore be some additional deficit in eye region focusing in those with CD. Interestingly, when examining time to first fixation on the eye region of the face in Chapter 2 (emotion recognition), the ADHD with CD group were found to be significantly slower to first fixate on the eyes for fear faces compared to typically developing controls (they were also slower - but not significantly so - than ADHD alone). Due to the time-dependent nature of the Stroop task (it was a reaction time task), it may be that this delay in focusing on the eye region in ADHD with CD has contributed to the lack of facilitation in responses compared to ADHD alone. Together, the above pattern of results suggests that while general attentional impairments in looking towards the eyes are shared by both groups with ADHD those with ADHD and CD may have a specific problem in the speed with which they first direct their attention to the eye region of the face. In Chapter 2 (emotion recognition), there was no time restriction on responding to the emotions, nor was there instruction to try to respond quickly, as a result of this, despite ADHD and CD taking longer to first fixate on the eyes, this did not influence their ability to recognise the emotions. Had there been a limited time in which to process the emotions and respond in this task, even larger differences between groups may have emerged.

6.1.8. Interrelations between Symptom Severity, CU traits and Emotion Processing Impairments

As described in the above section, ADHD severity was responsible for the lack of attention to the eye region observed both in Chapter 2 and Chapter 3, while it was unrelated to the emotion processing deficits across the experimental chapters. In all chapters (with exception of the emotion regulation [Chapter 4], which had a very small sample size) ADHD severity was positively related to CD severity, which suggests that those with a combination of ADHD and CD, as well as having emotional impairments, they are also burdened with more severe ADHD symptoms. Difficulties in executive functioning are inherent in ADHD (Rubia, 2011), and these may interfere with the ability of adolescent with ADHD to inhibit their (negative) emotional reactions. This may explain why they have a particularly poor prognosis (Thapar et al., 2001).

CD severity and CU traits were strongly related in the sample (*Rs* of between .44 and .55 in the chapters), which suggests that both measures may be measuring similar constructs (although they were by no means perfectly related). The contribution of these constructs to emotion processing impairments varied depending on the specific aspect of emotional functioning measured. CD severity was a stronger predictor of emotion recognition, emotion regulation, and happy affective empathy impairments (and fear conditioning to a lesser extent), while CU traits was found to mediate the difference in ADHD groups for affective empathy for negative emotions. The particularly strong relationship between CU traits and impairments in empathy makes logical sense since one of the key descriptive characteristics of CU traits is lack of empathy. However, this result could also be an artefact of the way CU traits were measured and the order in which tasks were completed during the testing session. The YPI was completed before the empathy task, and specific

questions in the YPI query as to how emotional the participant gets while watching characters on TV or in movies. A participant who answers that they do not feel emotional when watching characters in a film (and answers similarly on related questions) will score highly on CU traits. Further, when it came to the participant completing the empathy task, and being asked how strongly they felt certain emotions while watching the clip, they may have felt some pressure to answer in a way consistent with the earlier answers given, resulting in a high correlation between CU traits and low affective empathy.

When examining simple correlations in the experimental chapters, CD severity and CU traits were both significantly related to a number of emotion processing deficits. However when subsequently entered into a regression model together, the effect of CU dropped off (with the exception of affective empathy for negative emotions), suggesting that CU traits and CD severity are perhaps measuring similar constructs. However, a number of studies researching limbic system functioning in those with CD have found unique associations of both CU traits and CD severity in predicting functioning (Lockwood et al., 2013; Sebastian et al., 2012). Therefore, more research is needed to ascertain the overlap between these two constructs. Further, if both constructs do indeed offer unique predictions as to functioning of those with ADHD and CD, given the high overlap between CU and CD severity, future research should also explore if there is a causal relationship between the two, and the direction of such a relationship. It is possible that the presence of CU traits results in a more severe presentation of those with CD, but equally it is possible that those with high severity CD, which the current thesis has evidenced as having strong emotional impairments, subsequently leads to such individuals scoring in a higher range on measures of CU traits.

An additional novel finding, as previously discussed, was the relationship between impulsive-irresponsible traits and impairments in fear conditioning, over and above CD severity and CU traits. It may be that in those with ADHD and CD, because of problems in fear conditioning, are prone to having a particularly impulsive disposition. In a healthy individual with intact fear conditioning, when an antisocial behaviour is appropriately punished (or results in an aversive consequence) the two things will become associated, so when in the future this individual considers responding in this way, the associated punishment will be evoked and therefore inhibit that behaviour. In those with ADHD and CD (as evidenced by Chapter 5) this relationship is not formed (and therefore it may not be formed for many other potential behaviour-consequence pairings). They therefore will not benefit from the inhibition and therefore this may lead to a generally impulsive, unregulated disposition.

6.1.9. How do the Current Findings Relate to Theories of Antisocial Behaviour?

Broadly, our findings are consistent with the integrated emotion systems theory (IES; and preceding VIM; Blair, 1995, 2005), low fear theories (Lykken, 1957; Raine, 2002b) and neurobiological models (van Goozen et al., 2007) of antisocial behaviour in that we found specific impairments in emotion processing in adolescents with ADHD and additional CD only. According to both the IES and VIM models, it is distress-related cues, such as fear, which activate the violence inhibition mechanism and subsequently initiates a withdrawal response. This stops an individual committing an antisocial act (Blair, 2005a). If this is indeed the case, then participants with ADHD and CD, as evidenced by the current thesis, are less able to process these cues and consequently fail to produce a withdrawal response. Blair (1995) argued that the VIM is automatic, and in line with this, in Chapter 4

evidence was provided that individuals with ADHD and CD have impaired automatic processing of fear stimuli to the extent that they were not facilitated by the presence of fear on congruent trials. Further, Chapter 2 demonstrated that not only were those with ADHD and CD impaired in their ability to recognise fear, they were also more likely to confuse it with anger. This is important because anger activates the opposite action tendency to fear, approach rather than withdrawal, because the expression of anger signals that the expresser is feeling hostile and may act unpleasantly which has to be counter-acted (Shaver, Schwartz, Kirson, & O'Connor, 1987). Indeed, research has shown that when processing angry expressions in others, participants are faster to initiate approach movements toward the angry expression than avoidance movements (Wilkowski & Meier, 2010). In addition, research on aggressive boys (a symptom of CD) has also found that they act in aggressive ways to those who display anger compared to controls (Klaczynski & Cummings, 1989). Therefore, the findings of the current thesis suggest that if an individual with ADHD and CD is behaving violently towards another and that other person displays a fearful response, an individual with CD may continue with or even increase aggressive acts because they misperceive the threat signal as provocative and hostile.

A further aspect of the VIM is the stipulation that the activation of the VIM and the behaviour which was being performed at the time of its activation become associated. Therefore, the thought of committing an act that has previously been associated with the VIM would lead to its activation and therefore inhibition of said act and over time this leads to appropriate socialisation (Blair, 1995). In Chapter 5, we found that participants with ADHD and CD have impairments in their ability to form associations between a neutral and aversive stimulus. Therefore, along with the impairments in the processing of distress cues demonstrated in both Chapter 2 and 4,

those with ADHD and CD, even if they were able to process distress cues effectively, would also not come to associate their behaviour with the activation of the VIM. This means that the likelihood of these behaviours happening in the future would not be reduced, because they are less likely to acquire the proactive withdrawal response when such a behaviour is initiated.

As stated previously, the amygdala is heavily involved in fear conditioning, so much so that fear conditioning paradigms are now thought of as a proxy for amygdala dysfunction (Fairchild et al., 2011). In Chapter 5, we demonstrated that participants with ADHD and CD have impairments in fear conditioning which suggests that they may also have impairments in amygdala functioning, and that amygdala dysfunction is present in those with CD and not ADHD alone. Recent studies such as Yu et al., (2016) found amygdala abnormalities in those with ADHD; however, it is likely that this pattern of results was found because they did not exclude participants with CD from the ADHD group (although strangely they did exclude them from the control group). Similarly, Tajima-Pozo et al., (2016) found reduced amygdala volumes in those with ADHD, but again did not exclude or account for comorbid CD. Both the findings of the current thesis and those of Fairchild et al., (2011) suggest that amygdala dysfunction is driven by CD, and highlight the importance of considering CD symptomology in studies that examine amygdala functioning in those with ADHD.

The findings of the current thesis also have relevance for other uncertainties on the role of the amygdala in antisocial behaviour. In outlining the IES model, Blair (2005) extended the VIM model to describe the amygdala as central to the emotional deficits in those displaying such behaviours (Blair, 2005). Research has differed in its findings and interpretation of the role of the amygdala in emotion processing.

Some have argued that it is specifically activated by distress cues only (Blair et al., 1999; Adolps et al., 2005), while others suggest it is important for processing negative emotions in general (Fitzgerald, Angstadt, Jelsone, Nathan, & Phan, 2006). Others still have suggested that it is important in directing attention to emotionally salient cues in the environment such as the eyes (Dadds et al., 2008; Spezio et al., 2007). The findings from other chapters have relevance to this debate. We found in both Chapter 2 and 4 that adolescents with CD have impairments in the processing of fear stimuli. However, if the amygdala has a role in directing attention to emotionally salient stimuli, our findings are ambiguous. On one hand, we found that ADHD and CD participants did not differ in their time spent looking at the eyes (Chapter 2), but on the other hand, when examining the time to first fixation on the eye-region results (Chapter 2), we found that participants with ADHD and CD were slower to first fixate on the eye region. This, along with the importance of the eyes demonstrated in Chapter 4, suggests that there may be a delay in focusing on the eye region in those with ADHD and CD, supporting the idea that the functioning of the amygdala is problematic in those with CD.

In addition, in Chapter 5 we found that participants with comorbid ADHD and CD showed impairments in retaining an association between a neutral and aversive stimulus, as evidenced by the faster rate of extinction learning. Such a pattern of results is consistent with studies that have suggested that the amygdala is involved in emotional memory (e.g. Phelps, 2004). However, it must be highlighted that there was no direct measure of amygdala function or volume in the current study therefore the above reasoning should be taken with caution.

Findings from Chapter 5 are consistent with the low fear hypothesis (Lykken, 1957; Raine, 2002b). This theory suggests that individuals who display antisocial

behaviour have a lower propensity to experience fear. The lack of association between an antisocial act with punishment, as shown by the results of Chapter 5 (fear conditioning), will mean that those with ADHD and CD may be less likely to fear the consequences of their actions and therefore more likely to behave antisocially. In addition, we found evidence in Chapter 5 that participants with CD were less responsive to the aversive stimuli (i.e., noise) itself. Therefore, in addition to failing to form associations, adolescents with ADHD and CD would also experience fear evoking stimuli as less aversive, and these types of stimuli are therefore less likely to shape their future behaviour. This pattern of results is consistent with the somatic marker hypothesis (Bechara & Damasio, 2005). If adolescents do not find a stimulus aversive, a somatic marker is not produced; therefore, when participants act in a way that should be associated with a somatic marker, and this does not happen, this would not influence their decision making. Together, these findings in those with ADHD and CD provide further support for the suggestion that punishment may be a less effective intervention for those who behave antisocially (van Goozen et al., 2007).

The above theories mainly centre on the processing of negative and aversive stimuli, and indeed the current thesis found strong evidence that those with ADHD and CD have impairments in the processing of these cues. However, in addition to problems with negative emotions, we also found that those with ADHD and CD, although being able to recognise happy faces, were unable to share in the happiness of others (Chapter 3). The processing and ability to feel happy for others is not something that is included in any of the models above. However, the ability to empathise with others' happiness can be important in the shaping of social behaviour. One could tentatively speculate that an inability to share in the happiness

of others deprives one of an opportunity for reinforcement of prosocial behaviour; if one fails to empathise with someone who is happy, one is less likely to be motivated to behave in ways which would produce happiness in others. Given previous research highlighting an increased reward sensitivity in those with CD (Morgan, Bowen, Moore, & van Goozen, 2014), improving the ability to empathise with happiness (a positive reward), along with an improved ability to process and empathise with negative emotions, which is now starting to take place (Hubble et al., 2015), could be a fruitful approach for those who display conduct problems.

6.1.10. Strengths, Limitations, and Implications

6.1.10.1. Strengths.

The current thesis held a number of strengths over previous research. First, by recruiting a sample of participants who all had a diagnosis of ADHD and by separating them into those with and without comorbid CD, the current thesis was able to determine in which emotion processes groups differed. Chapter 2 was particularly strong because it also included a typically developing control group, which enabled the thesis to establish which impairments were specific to additional CD and which were general to both groups with ADHD. On a related note, we were able to use a clinical and well-characterised sample of adolescent psychiatric patients for the assessment of ADHD and CD symptomology, and this also enabled us to explore the contribution of clinical problems dimensionally to the emotion processing impairments. Next, we explored the role of CU traits in these impairments, as previous findings regarding the importance of CU traits seem to vary depending on the type of sample used, with stronger findings in community samples. The current clinical sample demonstrated that with the exception of affective empathy (but bearing in mind the previously mentioned caveats) CD

symptom severity was more strongly related to emotion impairments than CU traits were. In addition, our questionnaire measures assessing symptom severity and CU traits demonstrated high internal reliability suggesting that these measures were able and justified to explore deficits on a dimensional scale.

A further strength of the current research was its use of a wide variety of tasks tapping into emotion processing highlighting the variety of ways emotion processing impairments present themselves in those with ADHD and CD. In addition, this was the first study that applied eye-tracking while assessing emotion recognition and empathy in a sample of adolescents with ADHD, and divided into those with and without comorbid CD. The inclusion of this methodology helped us to demonstrate that while ADHD was associated with attentional impairments to the eyes, and attention to the eye is important to effective processing of emotions, these deficits alone did not explain the emotional impairments in those with ADHD and CD.

The thesis also makes a potentially significant contribution to the literature with its development of a novel pictorial emotional Stroop task (CEPS). This Stroop task successfully produced a true Stroop congruency effect, something which previous pictorial Stroops in the literature have failed to do (Ashwin et al., 2006). The paradigm is elegant in its simplicity in that the target task itself involves responding to only two categories of stimuli, while reaction times are collected by recording vocal responses, both of which save time in getting participants used to the response options. It is our belief that this means that the paradigm will lend itself for use in younger and/or more problematic participant groups.

6.1.10.2. Limitations.

The results of the thesis also need to be interpreted in light of its limitations. Despite recruiting participants with ADHD and subsequently dividing them into those with and without comorbid CD, the studies that were carried out would have been strengthened by the inclusion of a typically developing control group. This would have been particularly useful in the study on empathy (Chapter 3). Although we were able to show specific empathy deficits in those with ADHD and CD, it is unclear whether the cognitive empathy performance of both ADHD groups was similar to that of typically developing adolescents, or if the observed performance was impaired in comparison to controls. In addition, the inclusion of a CD alone group would have helped us to understand to what extent the observed emotion impairments are specific to those with combined diagnosis of ADHD and CD or whether these similarly exist in those with CD alone, and whether the mechanisms driving these impairments differ between these CD sub-groups. As a result, future research should seek to include both typically developing controls and those with a diagnosis of CD without associated ADHD.

It is also important to highlight the cross-sectional nature of the study's findings. Although we have found that emotional impairments are specific to ADHD and CD, the study cannot comment on the causal relationship between the impairments and conduct disorder with mere reference to its own results. For example, we cannot infer from the current findings whether those with CD and who are behaving in antisocial ways have reduced their amount of social contact with others, which has negatively impacted on their socialisation. This may be particularly important for the empathy chapter. Although we found that a preference to look towards the eyes did not play a role in affective empathy performance, it is not clear if such impairments played a role in the development of empathy earlier in

life. Indeed, previous studies have emphasised the importance of caregiver and child eye contact for the development of social communication early in life (Bedford et al., 2015). Future longitudinal studies exploring social attention are needed to determine if impairments in eye looking in early infancy lead to later conduct problems.

Further, although we intended to recruit enough female participants to make meaningful gender comparisons, the final sample size of females was not large enough to allow us to do this and both males and females were grouped into a single sample. We are aware of research that hypothesises that different mechanisms play a role in the development of conduct problems in males and females (Leve, Kim, & Pears, 2005; Meier, Slutske, Heath, & Martin, 2009; Pajer et al., 2008), but there is also evidence that the same emotion processing (Fairchild et al., 2010) and brain structure abnormalities (Fairchild et al., 2013) exist in females and males. It is because of the latter studies that we included females in the analyses.

Other methodological limitations relate to the eye-tracking paradigms. First, in Chapter 2 (emotion recognition), when participants were choosing the emotion of each face, the emotional options (i.e., words) remained on the screen. These emotional word options on the screen accounted for a lot of variance in eye-gaze in each trial and this reduced the amount of data available when participants were looking at the face. Future use of this paradigm should adjust for this and present the face on its own first, during which time eye gaze can be measured, to be followed by the presentation of the word options. In addition, in relation to Chapter 3 (empathy), the analysis window for eye tracking was restricted to a 4 second segment in each clip, which was the segment judged to consist of the highest emotional intensity. By restricting our analyses to this 4sec window, other potentially valuable information is lost. For example, it is possible that looking towards the eyes is more important

earlier on in clips but this is now unknown. Future studies should strive to identify more than one segment for analysis to determine reliability and consistency of the data within clips and the relative importance of the eyes earlier and later in the clip.

6.1.10.3. Implications.

6.1.10.3.1. *Diagnosis.* Evidence provided by the current thesis that emotion processing impairments are an additional problem for those with ADHD and CD compared to those with ADHD alone highlights the importance of assessing comorbid conduct problems in those with ADHD. The findings of the thesis also have relevance to the extent that problems in emotion regulation are involved in ADHD. In a recent review, Shaw et al., (2014) outlined three potential models which may account for the high prevalence of emotion dysregulation in those with ADHD, these were: (1) ADHD and emotion dysregulation are correlated but distinct dimensions, (2) emotion dysregulation is a core diagnostic feature of ADHD, and (3) emotion dysregulation and ADHD combined, constitutes an entity distinct from both ADHD and emotion dysregulation alone (Shaw et al., 2014). Given that Shaw's conceptualisation of emotion regulation "encompasses processes that allow the individual to select, attend to, and appraise emotionally arousing stimuli" (Shaw et al., 2014, p. 276) the findings from the current thesis have implications for Shaw's three models. In regards to the first model, that ADHD and emotion dysregulation are correlated but distinct dimensions, the current thesis finds evidence that is against this, in that we found that problems in both recognition of emotions and ability to automatically attend to such emotions was strongly correlated with CD severity and not ADHD severity. Shaw's second model proposed that emotion dysregulation is central to the hyperactivity, impulsivity and inattention of those with ADHD and suggested that emotion dysregulation is seen as an expression of the same

neurocognitive deficits that underpin other symptoms of ADHD; in other words it is a core diagnostic feature (Shaw et al., 2014). The current thesis found evidence against this proposal in that Chapter 2 demonstrated that emotion recognition impairments were specific to those with ADHD and CD, suggesting it is not a core diagnostic feature but only specific to those with additional CD. The results of the current thesis, along with research reviewed in Chapter 1 that cold cognitive deficits were a feature of ADHD (e.g. Rubia, 2011) while hot, emotional deficits were specific to those with additional CD suggests that emotion dysregulation is not a core feature of ADHD. Together, the results of the current thesis are most in line with the third model proposed by Shaw et al., (2014), that the combination of ADHD and emotion dysregulation represents a distinct entity. Our results would suggest that emotion dysregulation in those with ADHD is driven by comorbid CD because the current thesis found that emotional deficits were specific to those with additional CD. Indeed, a large majority of the studies that Shaw et al., (2014) includes to highlight the problems of emotion dysregulation in those with ADHD do not account for comorbid CD in their samples. To give a couple of examples, Greene et al., (1996) conceptualised that those with ADHD have a social disability as a result of their impairments in emotion processing compared to the control group, however, the ADHD group had a significantly higher number of individuals with CD and they made no attempt to control for or exclude these individuals. Further, Zalecki and Hinshaw (2004), found higher relational aggression in those with ADHD compared to a control group, but again, the ADHD group had a higher prevalence of CD than the control group but this was not acknowledged or controlled for. These are two examples, but Shaw et al's., (2014) review consisted of many more instances of this in areas such as emotion recognition (Boakes, Chapman, Houghton, & West, 2007;

Corbett & Glidden, 2000; Da Fonseca, Segui, Santos, Poinso, & Deruelle, 2009; Rapport, Friedman, Tzelepis, & Van Voorhis, 2002; Williams et al., 2008; Yuill & Lyon, 2007), reactive aggression (Matthys, Cuperus, & Van Engeland, 1999; Waschbusch et al., 2002), classroom rule breaking (Abikoff et al., 2002), amygdala structure (Plessen et al., 2006) and emotional control as assessed via event related potential (ERP) studies (Du et al., 2006), which were all used as examples of instances of emotion dysregulation in those with ADHD. Together the above suggests that comorbid conduct disorder should be routinely assessed in those with ADHD because the associated impairments and therefore prognosis can be largely different between those with ADHD alone and those with ADHD and comorbid CD.

Our findings also have relevance for the inclusion of ‘with limited prosocial emotions’ as a specifier in the diagnosis of CD (APA, 2013). We found high correlations between CD severity and CU traits (synonymous for limited prosocial emotions) which suggests that severity of CD may be measuring a similar concept as CU traits. In addition, the majority of the time we found that CD severity was more strongly predictive of emotional impairments than were CU traits. Importantly, the diagnostic criteria also includes a specifier as to the severity of the presentation of the conduct problems. The results of the current thesis suggests this may be more important than CU traits, especially when considering the emotion processing impairments of those with CD.

6.1.10.3.2. Intervention. The current findings highlight the importance of tailoring interventions to the specific impairments present in each individual. As a result of previous suggestions that those high in CU traits are characterised by a lack of attention to the eye region, a number of interventions were employed which taught participants to redirect their attention towards the eyes (Dadds et al., 2012; Hubble et

al., 2015). However, the findings of the current study suggest that impairments in looking towards the eye region of the face appear to be a characteristic of ADHD in general, so these interventions may only be helpful in those cases. Interestingly, in the study by Hubble et al., (2015), as well as training YO's to redirect their attention to important regions of the face to improve their emotion recognition performance, the training also helped them to interpret the features of the emotional faces by offering guidance as to which facial movements depict which emotions. Given the results of the current study and particularly those of Chapter 2, it is likely that the improvement in emotion recognition and the subsequent reduction in severity of offences committed resulted from this aspect of the intervention rather than the mere redirection of attention.

Interventions improving emotion recognition are also important because successful facial emotion recognition is thought to be involved in empathy (Blair, 2005), while empathy for negative emotions is involved in shaping socialisation and deterring individuals from engaging in antisocial acts. We found evidence that those with CD are impaired in fear recognition which suggests that interventions improving the recognition of fear could be useful as there may be carry over effects to affective empathy in general. Importantly, we did not find evidence of impairments in cognitive empathy in those with ADHD and CD but did find impairments in affective empathy, which suggests intact cognitive empathy is not fundamental to successful affective empathy, but as previously suggested, the additional vocal and contextual cues in the current study may have facilitated accuracy in cognitive empathy. Importantly, Blair (2005) suggests that motor empathy, the automatic mimicking of the facial expressions of others, is also prerequisite for emotion recognition which is required for appropriate affective

empathy. To date there is scarcity of research on motor empathy of those with CD (Bons et al., 2013) so future research should determine if those with CD are impaired in this and therefore whether interventions aimed at improving motor empathy would be helpful.

In addition, as well as finding impairments in affective empathy for negative emotions in those with ADHD and CD, we also found them for happiness. Empathising with the happiness of others produces positive reinforcement, as a result, any behaviour that is followed by positive reinforcement increases the likelihood in that behaviour happening again. Therefore, in typically developing individuals, prosocial behaviours towards others, will likely result in their happiness, which such individual can then empathise with and consequently receive positive reinforcement. However, because those with CD do not affectively empathise with happiness, if they were to engage in prosocial behaviour, such inability to process the happiness of others would mean prosocial actions are not followed by a positive reinforcement and therefore the chance of that behaviour happening again would not be increased. Interventions that improve the ability of those with CD to empathise with happiness may be a potentially useful avenue. We know that those with CD are highly sensitive to rewards (Matthys, van Goozen, Snoek, & Van Engeland, 2004), therefore if the ability of those with CD to empathise with happiness could be improved there may be a more natural source of reward that could help increase the occurrence of prosocial behaviours in these individuals. However, it must also be borne in mind that individuals with CD likely come from backgrounds where the happiness of others may not be available in abundance. For example, high rates of maternal depression have been found in those with CD (Tully, Iacono, & McGue, 2008) while the low SES environments that those with CD come from (Piotrowska,

Stride, Croft, & Rowe, 2015) may suggest there would be fewer sources of reinforcement available.

As with previous studies, we found evidence that those with ADHD and CD have problems with fear conditioning, while for the first time we also found increased rate of extinction learning of the conditioned response in these individuals. This further underlines the ineffectiveness of punishment as an intervention strategy for those with ADHD and CD. Current criminal justice policies generally focus on deterrence and punishment; the findings from the current study and previous literature suggest that antisocial individuals would be less responsive to these strategies. Given that such studies have indicated that impairments in both responding to and conditioning to aversive stimuli are present at a very early age (Baker et al., 2013; Gao et al., 2010b) and predict later aggression and antisocial behaviour, it highlights the importance of assessing these impairments early and offering appropriate interventions. Raine et al., (2001) examined the effects of an early educational and health enrichment programme on later psychophysiological arousal. They found that children who were assigned to a 2 year enriched nursery school intervention (compared to a typical nursery experience) showed increased physiological orientating and arousal (EEG and skin conductance) at 11 years of age. Importantly, research has indicated that heightened physiological arousal and intact fear conditioning can act as a protective factor against the development of antisocial behaviour (Brennan et al., 1997; van Bokhoven et al., 2005; van de Wiel, van Goozen, Matthys, Snoek, & van Engeland, 2004). As already stated, emotion recognition interventions are also being shown to be effective in reducing severity of antisocial behaviour in YOs, (Hubble et al., 2015), studies are now needed to assess if similar interventions work in reducing the conduct problems shown in those with

ADHD and CD. To date one such study has used emotion recognition interventions to improve emotion processing impairments in those with CD (Dadds et al., 2012), however degree of improvement in those in the treatment group was generally small, and those with CD still remained in the clinical range in terms of severity of problems following the intervention. As noted above, a number of these emotion processing impairments have been found to have a very early onset, therefore future studies should explore whether applying the training to younger age groups may provide a greater benefit.

6.1.11. Summary

The current thesis provides clear evidence that emotion processing is impaired in those with comorbid ADHD and CD compared to those with ADHD alone, and that these impairments are mainly driven by CD severity rather than CU traits. The specificity of the impairments in terms of the category of emotion in which impairments were present was found to vary depending on the particular type of emotion processing being assessed. Clear impairments in fear and neutral face processing were observed for the recognition of facial expressions in those with comorbid CD, while general impairments in affective empathy for negative and positive emotions were found for the same subgroup. In addition, only those with comorbid CD were found to have impairments in the automatic processing of fearful facial expressions. The current thesis also provided further support to research that has suggested that those with CD do not learn effectively from punishment, in that not only did the thesis find evidence of impairments in the acquisition of a fear response, but also that any association that was formed was subsequently lost at a significantly faster rate than those with ADHD alone.

Contrary to previous suggestions, deficits in processing of the eye regions of

emotional faces was not a characteristic of those with additional CD but instead was found to be a general problem in those with ADHD, and this problem increased with more severe presentations of ADHD.

Taken together the results highlight the importance of considering the heterogeneous nature of ADHD and the importance of considering the co-occurrence of CD. They emphasise the importance of assessing emotion processing impairments in those with ADHD early. For example, an emotion processing screening procedure for those who are referred to psychiatric services for symptoms of ADHD could be applied and tailored interventions guided by where specific impairments are apparent could be offered.

7. References

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8. Appendices

8.1. Appendix A: Description of Emotion Eliciting Videos Used in Chapter 3

The six clips selected were: sadness(1) – a young boy is shown crying over the body of a dead boxer who he had a strong relationship with; sadness(2) a man recalls the death of his close friend during the 9/11 terrorist attacks; happiness(1) – a young girl wins a horserace whilst riding a zebra and is shown celebrating with her father; happiness(2) – a man is shown celebrating after winning Olympic gold; fear(1) – a young girl watches a friend being attacked by a shark and is left on her own in a boat in the middle of the sea after the shark kills her friend; fear(2) – a woman is on a rope over a gorge when her harness breaks and is seen screaming for help.

8.2. Appendix B: Cognitive and Affective Scoring Criteria

The Cardiff empathy scoring system for cognitive and affective empathy
Cognitive Empathy
Target emotion
<p>0 = Target emotion not identified in main character</p> <p>1 = Target emotion identified at a low intensity</p> <p>2 = Target emotion identified at a high intensity Similar</p>
Similar relevant emotion 0
<p>0 = Similar relevant emotion not identified in main character</p> <p>1 = Similar relevant emotion identified at a low intensity</p> <p>2 = Similar relevant emotion identified at a high intensity Explanation</p>
Explanation of emotion
<p>0 = Incorrect or irrelevant explanation of emotion e.g., “The girl was fearful because she was scared”.</p> <p>1 = Explanation provides one factual reason for emotion e.g., “The girl was fearful because there was a shark”.</p> <p>2 = Explanation provides more than one factual reason for emotion e.g., “there was a shark and the boat got knocked” OR provides one consequence of the event e.g., “she thought she might die”.</p> <p>3 = Explanation provided one piece of factual information AND took into consideration the consequence of the event for the main character e.g., “There was a shark in the water and she thought it might kill her”.</p> <p>4 = Explanation provided more than one piece of factual information AND took into consideration the consequence of the event for the main character e.g., “there was a shark, her boyfriend fell in the water and she thought she might die”.</p> <p>5 = Explanation provided a thorough account of the main character situation providing multiple factual reasons for their emotions and elaborating on the possible consequences of the situation e.g., “There was a shark in the water and it had already killed her boyfriend. She was on her own and would be worried that it might come back for her and kill her as well”.</p>
Affective Empathy
Target emotion
0 = Target emotion not identified in self

<p>1 = Target emotion identified at a low intensity</p> <p>2 = Target emotion identified at a high intensity</p>
<p>Similar relevant emotion</p>
<p>0 = Similar relevant emotion not identified in self</p> <p>1 = Similar relevant emotion identified at a low intensity</p> <p>2 = Similar relevant emotion identified at a high intensity</p>
<p>Explanation of emotion</p>
<p>0 = Incorrect or irrelevant explanation of emotion e.g., “I felt happy because I like seeing people die”.</p> <p>1 = Explanation based on factual information e.g., “I felt sad because the man died”.</p> <p>2 = Explanation based on main characters emotions or involved participant transposing themselves into main characters situation e.g., “I felt sad because I thought about that happening to me”.</p>

8.3. Appendix C. Comparison of Congruency Effect between ADHD Groups and Undergraduate Controls

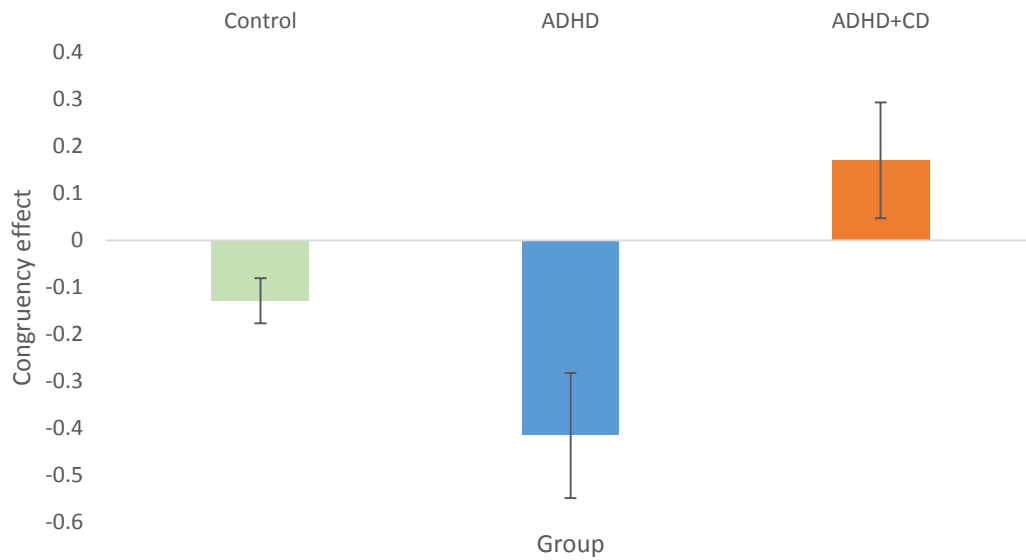


Figure S4. Mean congruency effect for pictorial emotional Stroop (Chapter 4) of controls, ADHD and ADHD+CD groups.