# *Examination of key child and psychosocial predictors of Oppositional Defiant Disorder (ODD) in young people with Attention Deficit Hyperactivity Disorder (ADHD)*

**Jo-Ann Winther**

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Department of Paediatrics

Faculty of Medicine, Dentistry and Health Sciences

University of Melbourne

ORCID identifier: 0000-0002-5003-4108

# Abbreviations List

| | |
|---|---|
| AACAP: | The American Academy of Child and Adolescent Psychiatry |
| ADHD-C: | ADHD-Combined presentation |
| ADHD-HI: | ADHD-predominantly Hyperactive presentation |
| ADHD-I: | ADHD-predominantly Inattentive presentation |
| ADHD: | Attention Deficit Hyperactivity Disorder |
| A-DISC: | Anxiety Disorders Interview for Children |
| APA: | American Psychiatric Association |
| ANOVA: | Repeated Measures Analysis of Variance |
| AQ-Child: | The Autism Spectrum Quotient: Children's Version |
| AQ-Adol: | The Adolescent Autism Spectrum Quotient |
| ASD: | Autism Spectrum Disorder |
| ATP: | The Adolescent Transitions Program |
| BSE: | Between Search Errors total score (CANTAB) |
| CANTAB: | Cambridge Neuropsychological Test Automated Battery |
| CBCL: | Child Behaviour Checklist |
| CBT: | Cognitive Behavioural Therapy |
| CD: | Conduct Disorder |
| CON: | Waiting-list Control Group |
| CT: | Child Training |
| DAS: | Dyadic Adjustment Scale |
| DD: | Dysthymic Disorder |
| DESR: | Deficient Emotional Self-Regulation |
| DMDD: | Disruptive Mood Dysregulation Disorder |
| DSM: | Diagnostic and Statistical Manual of Mental Disorders |
| FAD: | Family Assessment Device |
| FDI: | Freedom from Distractibility Index Scores |
| FSIQ: | Full Scale Intelligence Quotient |
| FUI: | Family Unit Inventory |
| GAD: | Generalised Anxiety Disorder |

| | |
|---|---|
| GBG: | Good Behaviour Game |
| HSCL: | Hopkins Symptom Checklist |
| ICD: | International Classification of Diseases |
| IQ: | Intelligence Quotient |
| MANOVA: | Wilks Multivariate Test of Significance |
| MDD: | Major Depressive Disorder |
| ML: | Mastery Learning |
| MST: | Multisystemic Therapy |
| MTA: | Multimodal Treatment Study of Children |
| MTFC: | Multidimensional Treatment Foster Care |
| ODD: | Oppositional Defiant Disorder |
| PACS: | Parental Account of Childhood Symptoms |
| PDD: | Persistent Depressive Disorder |
| PRI: | Perceptual Reasoning Index (WISC) |
| PSI: | Processing Speed Index (WISC) |
| PT: | Parent Training |
| RSA: | Respiratory Sinus Arrhythmia |
| SAS: | Social Adversity Scale |
| SD: | Standard Deviation |
| SES: | Socio-Economic Status |
| SPSS: | Statistical Package for the Social Sciences |
| SS: | Spatial Span (CANTAB) |
| STEP: | School Transitional Environment Project |
| SWM: | Spatial Working Memory |
| TFCO: | Treatment Foster Care Oregon |
| TRF: | Teacher Report Form (CBCL) |
| VCI: | Verbal Comprehension Index (WISC) |
| VSWM: | Visual Spatial Working Memory |
| VWM: | Verbal Working Memory |
| WISC-4: | Wechsler Intelligence Scale for Children-Fourth Edition |
| WM: | Working Memory |
| WMI: | Working Memory Index (WISC) |
| WRAT-4: | Wide Range Achievement Test-Fourth Edition |
| YSR: | Youth Self Report (CBCL) |

# Abstract

*Introduction:* ADHD and ODD are two common developmental mental health conditions that can co-occur. There are associated functional impairments across clinical, biological, psychological, and social domains, which may be more severe when they co-occur. It is important to understand and recognise risk factors for young people with ADHD who are developing or maintaining ODD in order to target them early with appropriate and effective therapy interventions. To date, there are limited longitudinal datasets examining key risk factors associated with these comorbid conditions and more research is needed to investigate what places a young person with ADHD more at risk for ODD. This study examines the role of key child characteristics, psychosocial factors, ADHD subtypes and commodities.

*Methods*: A three-year blinded evaluation of children with ADHD, investigating those with and without follow-up ODD. At baseline, relevant demographic, functional and clinical information was collected from clinically referred participants with ADHD (N=419). All participants were followed up three years later. Twenty-five percent (N=104), were unable to be reassessed. 150 participants were recruited with ADHD and follow-up ODD (120 males; 30 females) and 60 were recruited with ADHD alone (43 males; 17 females). Groups were compared for clinical presentations (ADHD subtype, comorbid disorders), key child characteristics (gender, IQ, temperament, Visual Spatial Working Memory, academic ability, emotional regulation, social problems), and psychosocial factors (parental level of education, family functioning, parental relationship functioning, parenting skill, parental psychopathology).

*Results:* The two groups did not differ for age, gender, or SES at baseline. Young people with ADHD and follow-up ODD had increased temperament activity level, worse Verbal Comprehension, worse Visual Spatial Working Memory, increased aggression, and worse social problems in comparison with the ADHD alone group. In the psychosocial domain, lower parental level of education, overall higher parental psychopathology, and higher parental Obsessive Compulsive symptoms and Interpersonal Sensitivity significantly predicted ADHD and follow up ODD. ODD, CD

and ADHD-C significantly predicted ADHD and follow up ODD group membership. In contrast, GAD acted as a protective factor, predicting membership in the ADHD alone group. Backwards stepwise logistical regressions were completed for all significant factors, finding that increased temperament activity, parent reported increased aggression and teacher reported increased social problems were the strongest predictors of ADHD and ODD follow up compared to the ADHD alone group.

*Discussion*: The findings suggest that when working with young people with ADHD it is important to effectively manage their temperament activity levels, aggression and to teach skills that improve their social skills. Effectively treating ADHD-C symptoms, aiding Verbal Comprehension and Visual Spatial Working Memory difficulties, and supporting parents with furthering their education and mental health difficulties should be included in a comprehensive treatment plan. It is important for clinician's to be aware of the bi-directional relationship between ADHD and parent behaviour and functioning, which highlights the importance of systemic intervention. Further understanding how to optimally manage the helpful aspects of a young person's anxiety may assist in preventing behavioural problems. Study limitations, future clinical and research directions are discussed.

# Declaration

This is to certify that:

- The thesis comprises only my original work towards the PhD except where indicated in the Preface,
- Due acknowledgement has been made in the text to all other material used,
- The thesis is fewer than 100 000 words in length, exclusive of tables, maps, bibliographies, and appendices.

**11th May 2020**

_____ _____

Signature Date

# Preface

This PhD thesis contains the result of research undertaken at the Department of Paediatrics, University of Melbourne, and the Royal Children's Hospital (Academic Child Psychiatry Unit (ACPU) and the Developmental Neuropsychiatry Program (DNP)) between May 2014 and April 2020. The thesis and the work described is my own work. I collaborated with the ACPU and DNP teams in the recruitment and clinical assessments of the participants.

# Acknowledgements

I would like to dedicate this thesis to all my past and current clients. They inspire and motivate me to continually update my knowledge and skills and be the best clinician I can be.

There are people who I would like to acknowledge for their significant support and care throughout this doctoral journey:

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# CHAPTER 1: Introduction to Attention Deficit Hyperactivity Disorder (ADHD) – all Presentations (Hyperactivity/Impulsive (HI), Inattentive (I) & Combined (C) type)

## 1.1 INTRODUCTION TO ADHD – ALL PRESENTATIONS

This chapter reviews the development of ADHD (all presentations). It covers: the historical context; the epidemiology of ADHD including prevalence estimates, differences across gender and age; key clinical characteristics of ADHD; key child characteristic components, both biological and psychological (for example, IQ scores, Working Memory, academic achievement and emotional regulation); and key social components (for example, family functioning, parental relationship functioning and parenting skill, and parental psychopathology). In addition, key comorbid conditions (especially key clinical characteristics of ADHD and Conduct Disorders, ADHD and Anxiety, ADHD and Depressive Disorders, and ADHD and Autism Spectrum Disorder); developmental course (natural history) of ADHD; pharmacological (primarily stimulant and non-stimulant medications) and non-pharmacological (for example, behavioural, cognitive and neurocognitive interventions) treatment approaches; and future directions are explored.

## 1.2 HISTORICAL CONTEXT OF THE DEVELOPMENT OF ADHD (HI, I & C)

### 1.2.1 Introduction

The current iteration of the Diagnostic and Statistical Manual of Mental Disorders (DSM) (American Psychiatric Association, 2013) categorises ADHD within the neurodevelopmental disorders group. Other disorders included in this group are: Intellectual Developmental Disorders; Communication Disorders; ASD; Specific Learning Disorder; and Motor Coordination Disorder. These are a group of disorders in which the development of the central nervous system is disturbed, which can impact on a child's motor functioning, learning, language, non-verbal communication, and social relationships. ADHD is not a new diagnosis - it has been studied for over two hundred years and has been shaped by different theories and ways of defining its core symptoms.

### 1.2.2 ADHD Before the Contemporary DSM Classifications

A German physician, Weikard, in 1775, provided the first clinical descriptions of inattentiveness, hyperactivity and impulsivity (Barkley & Peters, 2012). Weikard (as cited in Barkley and Peters (2012)) classified these difficulties as an "illness" that occurred more commonly in young people than adults, with males having a higher rate of attention problems than females. In 1798, Crichton (as cited in Palmer & Finger, 2001) described patients with "extreme mental restlessness". He believed that "derangement in some functions of the brain were responsible for inhibiting activities", which resulted from "undue brain reactiveness to mental and emotional stimuli", that lead to "simple hyper-excitability" (Weikard, as cited in Sandberg, 1996). Thus, linking inattention, motor overactivity and emotional problems which led to further research looking at underlying brain pathology and inherited vulnerability as risk factors for behavioural problems in children.

At the start of the twentieth century, Still (1902) classified three subtypes of a "defect of moral control" in children with disturbed behaviour: 1. "a defect of cognition in relation to the environment"; 2. "a defect of moral consciousness"; and 3. "a defect of inhibitory volition". He proposed three possible causes: a "gross lesion of brain"; "no physical disease or injury affecting the brain, but a more general arrest or delay in its development"; and "no known cause" (as cited in Vance, 1997 ). Tredgold (1908) used the term "Neuropathic Diathesis" to describe "an aetiological model based on an inherited brain defect". He demonstrated a longitudinal correlation between "minor brain damage" in childhood and subsequent behaviour problems and/or learning difficulties. This thinking informed the work in Europe and the United States of America during the Encephalitis epidemic of 1917-1918. When studying children's behaviour after Encephalitis, they found a significant correlation between brain damage and behaviour disorder in children, leading to psychological and social risk factors in cognitive and behavioural disorders being rejected (Sandberg, 1996).

During the 1930's, brain pathology was focused on as the causation of hyperactivity in children. Kahn and Cohen (1934) (as cited in Sandberg, 1996) believed that an abnormal organisation of the brain stem, possibly related to acquired brain injury, prenatal encephalopathy, birth injury or a congenital defect in the brain stem-based activity-modulating stem caused the difficulties. In 1932, the term "Hyperkinetic

Disease" was used by Kramer and Pollnow (as cited in Lange, Reichl, Lange, Tucha and Tucha, 2010). They described "a hyperkinetic disease of infancy" characterised by significant motor restlessness in children along with "a conspicuous lack of purposefulness". The key symptoms for children were that they could not stay still (for example, running up and down the room, climbing on furniture). They also reported that Hyperkinetic disease was often associated with emotional dysregulation (aggression and anxiety) and mood instability (increased excitability, frequent fits of rage, and a tendency to become aggressive or burst into tears for minimal reasons) (Lange et al., 2010). In 1937, Bradley found amphetamine medication (Benzedrine) effective for treating the hyperactivity symptoms in children, by stimulating the higher inhibitory centres in the brain to increase voluntary control by compensating for the defective brain stem activity-modulating system (Sandberg, 1996).

By mid-twentieth century researchers and clinicians started to challenge brain damage as being the only cause for hyperactive behavioural symptoms in children. Childers, in 1935, reported that brain damage is not necessary for hyperactivity in children, which led to the term "Minimal Brain Damage" being used (Vance, 1997). Liliefeld, Pasamanick and Rodgers (1955) and Pasamanick, Rogers and Lilienford (1996) found an association between maternal and foetal factors and subsequent hyperactive behaviour problems (as cited in Sandberg, 1996) recommending the term of "Minimal Brain Dysfunction" be used instead of "Minimum Brain Damage". By the 1950's behavioural diagnostic descriptions were proposed: "Hyperkinetic Behaviour Syndrome" and "Hyperkinetic Impulse Disorder" (Laufer & Denhoff, 1957).

### 1.2.3 The DSMs: Contemporary Classifications of ADHD (DSM-II, III, III-R, IV, IV-TR and 5, 1963-2013)

Attention-Deficit/Hyperactivity Disorder (ADHD) first appeared in the second edition of the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (APA, 1968). In the first four editions of The Diagnostic and Statistical Manual of Mental Disorders (DSM) there was a shift from focusing on hyperactivity as a dominant symptom for the disorder (DSM-II, Hyperkinetic Reaction of Children), to attention difficulties becoming a core defining criteria (DSM-III, Attention Deficit Disorder (ADD) with or without hyperactivity), to then a single diagnosis covering the

core inattention, impulsivity, and hyperactivity symptom dimensions (DSM-III-R, Attention Deficit Hyperactivity Disorder (ADHD)).

The DSM-IV (APA, 1994) was influenced by behavioural and molecular genetics research, using structural and functional neuroimaging, to confirm the importance of the biological underpinning of the disorder (Lange et al., 2010). The DSM-IV (APA, 1994) included three subtypes of ADHD: a predominantly inattentive type; a predominantly hyperactive-impulsive type; and a combined type with symptoms of both inattention and hyperactivity-impulsivity dimensions.

More recent research has demonstrated the chronic and persistent nature of the disorder and that adults were able to be validly and reliably diagnosed with ADHD (Lange et al., 2010). So, the DSM-5 (APA, 2013) added further descriptive examples of ADHD in adolescents and adults and the age of onset increased to before 12 years (previously before age 7). It was recognised that symptoms change over time in an individual with ADHD, so the term "subtype" was replaced with "presentation" and a severity rating, to express the degree of functional impairment in everyday life (mild, moderate or severe), was added (APA, 2013). There was also a reduction from six to five minimum number of symptoms in the symptom domain required for older adolescents and adults.

### 1.2.4 Summary

ADHD has been conceptualized differently over time. An early focus on brain pathology / brain damage as the cause for the symptom presentation has been challenged by subsequent research. At present, ADHD is viewed as a behavioural diagnostic description of a syndrome/disorder. The focus on the core symptom of hyperactivity-impulsivity shifted to an increased focused on inattention, seeing a name change from Hyperkinetic Reaction of Children to Attention Deficit Hyperactivity Disorder. Diagnostic revisions from DSM-IV (1994) to DSM-5 (2013) did not modify the core symptom domains or revise the 18 core symptoms, suggesting that the definition of ADHD has largely withstood the test of time (Epstein & Loren, 2013). At present, ADHD is categorised within the neurodevelopmental disorders group in the DSM-5, with a diagnosis being able to be made either with a presentation of a predominantly inattentive type, a predominantly hyperactive-impulsive type and a combined type with pervasive symptoms of both inattention and hyperactivity-impulsivity dimensions. In addition, the

chronic and persistent nature of the disorder has become clear with adults now able to be validly and reliably diagnosed with ADHD.

## 1.3 EPIDEMIOLOGY OF ADHD

### 1.3.1 Introduction

ADHD is one of the most common diagnoses for children and adolescents, although there is considerable variability in ADHD point prevalence estimation worldwide. Polanczyk, de Lima, Horta, Biederman, & Rohde (2007) estimated the global prevalence of ADHD as 5.29%. Methodological factors have been considered when looking at why differences have occurred, including: source of information; evolving diagnostic criteria and different classification systems; age and gender of participants sampled: ratings of impairment; and geographical location / cultural differences (Camilleri & Makhoul, 2013; Matthews, Nigg, & Fair, 2014; Guilherme Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007). However, there have been no obvious differences in prevalence rates observed between study years, suggesting that the community prevalence of ADHD has remained stable over the past three decades (Guilherme Polanczyk et al., 2007; Kapil Sayal, Prasad, Daley, Ford, & Coghill, 2018).

### 1.3.2 Age and ADHD

Participant age is a crucial variable affecting prevalence estimation, as the point prevalence of ADHD decreases from childhood through adolescence and into adulthood (Faraone, Biederman, & Mick, 2006; Hill & Schoener, 1996; Vance, 1997). The literature suggests that 40-60% of affected children continue having the disorder during adulthood, depending on the criteria for persistence applied (Rohde, Verin, & Polanczyk, 2012).

There is also a strong and growing interest now in the diagnosis of ADHD in adults and it is being recognized and diagnosed more frequently (APA, 2013; Estévez et al., 2014; Matte et al., 2015). The World Health Organisation World Mental Health survey looked at ten countries to produce a global adult ADHD prevalence rate of 3.4%, the range varied between 1.2% (Spain) to 7.3% (France) (Fayyad et al., 2007). Using a similar approach to the one developed by Polanczyk et al. (2007), in children and adolescence. Simon, Czobor, Bálint, Mészáros, and Bitter (2009) reviewed the literature

looking at adults with ADHD, finding six studies to include in their meta-analysis. They found a pooled ADHD prevalence rate of 2.5%.

### 1.3.3 Gender and ADHD

Research consistently suggests that the ADHD prevalence is higher in boys than in girls. In the systematic review completed by Polanczyk et al. (2007) the pooled ADHD prevalence for boys was 2.45 times higher than that for girls (only non-referred samples were included). Other studies have reported male-to-female ratios ranging from 4:1 to 9:1, depending on the setting, for example, community versus clinical samples (American Academy of Pediatrics, 2000; Verma & Kushwaha, 2014). Polanczyk et al. (2007) suggest the prevalence among girls seems to be higher in community samples than in clinical samples as there appears to be a difference for diagnosis and treatment referrals for females.

### 1.3.4 Ethnicity and ADHD

The impact of ethnicity on the prevalence rates of ADHD is not well understood. Some studies suggest that ethnicity has no effect on prevalence rates of ADHD (Angold et al., 2002; Froehlich et al., 2007), while others suggest a higher prevalence of ADHD symptoms in African American children (Miller, Nigg, & Miller, 2009). Thomas, Sanders, Doust, Beller, and Glasziou (2015) reported that ADHD prevalence estimation is greater in the Middle East compared to North America. However, they believed that it was due to "language restriction" and when adjusted for, lower pooled estimation of ADHD and less discrepancy of ADHD prevalence estimation in different regions were found. This area requires more research to fully understand if ethnicity plays a role in prevalence rates of ADHD.

### 1.3.5 Socio-Economic Status (SES) and ADHD

Like ethnicity, the role of Socio-Economic Status (SES) in prevalence estimates of ADHD is not clear. Positive and negative associations between SES and ADHD appear in the literature. Some studies have found higher rates of ADHD in low SES environments (Döpfner, Breuer, Wille, Erhart, & Ravens-Sieberer, 2008; Graetz, Sawyer, Hazell,

Arney, & Baghurst, 2001; Pineda et al., 1999; Russell, Ford, Rosenberg, & Kelly, 2014), while others have reported no significant differences between SES levels in ADHD prevalence studies (Canino et al., 2004; Zwirs et al., 2007). Some studies from the USA have suggested a greater prevalence of childhood ADHD among families with a higher income (Getahun et al., 2013; Visser, Lesesne, & Perou, 2007). However, this association requires caution, as it is not clear what role the availability of health insurance plays (Getahun et al., 2013; Sayal & Goodman, 2009).

It is challenging to fully understand the impact of SES on the prevalence rates of ADHD as it is difficult to separate other interleaved risk factors such as parental ADHD diagnosis, lack of financial independence, lower educational attainment levels, low level of parental support networks, and availability of health insurance (Getahun et al., 2013; Polanczyk et al., 2007; Russell et al., 2014; Sayal & Goodman, 2009).

### 1.3.6 Key Methodological Factors for ADHD

Numerous studies have been conducted globally to determine the prevalence of ADHD in the community for both children and adults. Studies have found rates ranging from as low as 0.9% to as high as 20% (Bird, 2002; Faraone, Sergeant, Gillberg, & Biederman, 2003). Polanczyk et al. (2007) conducted a comprehensive systematic review and meta-analyses to better understand the reasons for this variability in the prevalence rates of ADHD being reported. They looked at one hundred and two investigations with non-referred samples of children and adolescents from all continents and documented a worldwide pooled prevalence of ADHD of 5.29% for children and 2.5% for adults (APA, 2013; Polanczyk & Rohde, 2007).

Polanczyk et al. (2007) reported three main reasons for the variability in the prevalence rates: the information source used, especially questionnaire versus interview and informant; presence or not of a definition of impairment; and diagnostic system utilised. Geographic location did show some significant variability between countries, however not all, suggesting that geographic location plays a limited role in the reasons for the large variability in the ADHD prevalence rates worldwide. Instead the variability seems to be explained primarily by the methodological characteristics of studies (Polanczyk et al., 2007).

### 1.3.7 Summary

The point prevalence of ADHD varies considerably across the world (from 1% to 20%). This variability appears to occur due to: the information source used, especially questionnaire versus interview and informant; presence or not of a definition of impairment; diagnostic system utilised; age and gender of the participant (Polanczyk et al., 2007). A comprehensive systematic review and meta-analyses looked at one hundred and two investigations with non-referred samples of children and adolescents from all continents and documented a worldwide pooled prevalence of ADHD of 5.29% for children and 2.5% for adults (Polanczyk and Rohde, 2007; APA, 2013). Further research into how ethnicity and SES influence the variability of ADHD prevalence is required.

## 1.4 KEY CLINICAL CHARACTERISTICS OF ADHD IN ALL PRESENTATIONS: PREDOMINANTLY HYPERACTIVITY-IMPULSIVE PRESENTATION (ADHD-HI); PREDOMINANTLY INATTENTIVE (ADHD-I); AND COMBINED PRESENTATION (ADHD -C)

### 1.4.1 Introduction

In DSM-5, ADHD is categorised in the neurodevelopmental disorders group. Neurodevelopmental disorders are defined through their interference with the functioning of developmental processes in the brain and central nervous system. They are characterized by developmental deficits that produce impairments of personal, social, academic, or occupational functioning. These disorders typically manifest early in development, often before the child enters school.

ADHD is defined by impairing levels of inattention, disorganization, and/or hyperactivity-impulsivity that interfere with functioning or development (APA, 2013). To diagnose ADHD six or more symptoms (five symptoms for those over 17 years of age) must be present before age 12 years and be present over the preceding six-month period or more. Several inattentive or hyperactive-impulsive symptoms must also be present in two or more settings (for example home, school/work, with friends or relatives, in other activities). The symptoms do not occur exclusively during the course of schizophrenia or other psychotic disorders and are not better explained by another mental

health condition (Mood Disorder, Anxiety Disorder, Dissociative Disorder, Personality Disorder, substance intoxication or withdrawal). ADHD is characterised by three different sets of presentations: the diagnosis should specify whether it is a "combined presentation", "predominantly inattentive presentation" or a "predominantly hyperactive-impulsive presentation". Its severity should be specified (mild, moderate, and severe) and it should be noted if it is "in partial remission" (APA, 2013).

### 1.4.2 Diagnostic Process for ADHD

There are no diagnostic laboratory tests or diagnostic biological markers specific for ADHD. As a group, compared with peers, children with ADHD display increased slow wave electroencephalograms, reduced total brain volume on magnetic resonance imaging, and possibly a delay in posterior to anterior cortical maturation, but these findings are not diagnostic (APA, 2013).

ADHD is commonly diagnosed after a comprehensive assessment of gathering information, from multiple informants, over one to three sessions with a qualified professional (Paediatrician, Psychiatrist, Psychologist or Mental Health Clinician). Information is often collected using a combination of: a clinical interview with history taking and screening for associated comorbidities; observation; examination; and a variety of assessment tools (checklists, behaviour questionnaires, and/or rating scales). During a clinical interview it is important to collect information from both parents and teachers. Information should include: details of the mother's pregnancy and birth; the child's developmental milestones, speech and language function, early temperament; daily activities; school performance including behavioural problems and peer relationships; medical history; possible emotional stressors on the child and any co-morbidities.

Most children with ADHD have no abnormalities on physical examination, but a thorough examination should be performed to rule out neurological disorders, dysmorphic features, visual and hearing impairments, and other disorders that may mimic ADHD (Leung & Lemay, 2002). Other evaluations, such as tests of intelligence (for example, Wechsler Intelligence Scale) and educational achievement (for example, Woodcock-Johnson Psychoeducational Battery, Revised; Wide Range Achievement Test

– 4) are also often used to help rule out intellectual deficits and learning disabilities (Fletcher, Francis, Morris, & Lyon, 2005).

### 1.4.3 Diagnostic System Utilised for ADHD

The main two classification systems used for diagnosing ADHD are the International Classification of Diseases (ICD) and the Diagnostic and Statistical Manual of Mental Disorders (DSM). The latest versions of these manuals are the DSM-5 (APA, 2013) and the ICD-10 (World Health Organization, 1992). In the DSM-5, ADHD sits under the Neurodevelopmental Disorders section, codes 314.00 – 314.01, whereas for the ICD-10, ADHD sits under the Mental, Behavioural and Neurodevelopmental Disorders - Behavioural and emotional disorders with onset usually occurring in childhood and adolescence, codes F90-F90.9.

Although the list of symptoms and the evaluated constructs are very similar between the DSM-5 and ICD-10 they construct the diagnosis differently (Polanczyk et al., 2007). ICD-10 requires a minimum number of symptoms in all three dimensions (inattention, overactivity, and impulsivity), whereas DSM-5 allows for a minimum number of symptoms in just the attention or the hyperactive/impulsivity dimension. Another difference between DSM-5 and ICD-10 is in being able to diagnose ADHD if there are comorbidities present: ICD-10 has Mood, Anxiety and Developmental Disorders as exclusion diagnoses; DSM-5 (and early versions) allows an ADHD diagnosis in the presence of these disorders. Polanczyk et al. (2007) found there were large differences in reported prevalence estimates in studies using DSM-IV and ICD-10, with fewer cases being diagnosed using ICD-10.

Both manuals describe the core features of ADHD in a similar way, identify the disorder originating in childhood, and often persisting into adulthood with symptoms interfering with functioning in at least two settings. One key difference is the DSM-5 outlines that the symptoms must be present prior to age 12 years, whereas ICD-10 criteria state before the age of 7 years. With the extended age-of-onset criterion for the diagnosis of ADHD in DSM-5 (APA, 2013) there was an expectation that this may lead to higher point prevalence estimates across developmental stages compared to previous DSM versions (Faraone et al., 2015; Vande Voort, He, Jameson, & Merikangas, 2014). Thomas et al. (2015) undertook a meta-analysis with one of their aims to examine whether

estimates have increased with the publication of DSM-5. Looking at one hundred and seventy-five studies of ADHD prevalence estimates, with an overall pooled estimated of 7.2%, no statistically significant difference was found between DSM editions.

In Australia it is common for the DSM classification criteria to be used within the Mental Health Setting. So, the DSM-5 criteria will be used throughout this manuscript and for this research.

### 1.4.4 Three Presentations of ADHD

#### *1.4.4.1 ADHD Predominantly Hyperactive/Impulsive Presentation (ADHD-HI)*

For a diagnosis of ADHD Predominantly Hyperactive/Impulsive (ADHD-HI) presentation, six (or more) of the following symptoms must have persisted for at least six months to a degree that is inconsistent with the persons developmental level and that negatively impacts directly on social and academic/occupational activities: often fidgets with or taps hands or feet or squirms in seat; often leaves seat in situations when remaining seated is expected; often runs about or climbs in situations where it is inappropriate; often unable to play or engage in leisure activities quietly; is often "on the go," acting as if "driven by a motor"; often talks excessively; often blurts out an answer before a question has been completed; often has difficulty waiting his or her turn; and often interrupts or intrudes on others.

Less than 15 percent of individuals with ADHD manifest a pure HI presentation (Egger, Kondo, & Angold, 2006; Ramsay & Rostain, 2014). ADHD-HI is more often diagnosed in very young children and is the most common presentation in pre-school and school age children with ADHD (Posner et al., 2007). One explanation for this is that the inattention criteria are developmentally inappropriate for very young children (Dulcan & Lake, 2012).

Wilens, Biederman, and Spencer (2002) found children with ADHD-HI performed relatively well in school compared with people with inattentive symptoms but had serious difficulties at home and/or situations with fewer social/interpersonal structures. Children with ADHD-HI reportedly manifested more difficulties in engaging with unsupervised activities and controlling outbursts of anger when playing with other children (Wilens et al., 2002). They also experience more accidents, unintentional

injuries, visits to Emergency Departments and being suspended more from day care (Posner et al., 2007).

Although ADHD-HI is less frequent in young adults, it is significantly associated with behavioural inhibition deficits (Shaw, Stringaris, Nigg, & Leibenluft, 2014) and emotional problems (Satterfield et al., 2007) in comparison with young adults with ADHD predominantly inattentive type (ADHD-I). This is mainly associated with an elevated risk for substance abuse, cigarette smoking, driving problems and oppositional/antisocial behaviours leading to interpersonal difficulties and legal problems (Ramsay & Rostain, 2014).

#### *1.4.4.2 ADHD Predominantly Inattentive Presentation (ADHD-I)*

For a diagnosis of ADHD predominantly Inattentive (ADHD-I) presentation six (or more) of the following symptoms must have persisted for at least six months to a degree that is inconsistent with the individuals developmental level and that negatively impacts directly on social and academic/occupational activities: often fails to give close attention to details or makes careless mistakes; often has difficulty sustaining attention in tasks or play activities; often does not seem to listen when spoken to directly; often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace; often has difficulty organizing tasks and activities; often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort; often loses things necessary for tasks or activities is often easily distracted by extraneous stimuli; and is often forgetful in daily activities.

Inattentive difficulties are expressed by difficulties in maintaining attention and high levels of distractibility (APA, 1994). They manifest with individuals having problems completing homework/assignments, difficulties prioritising tasks to complete, having poor organisation and time management skills (Ramsay & Rostain, 2014). They are often described by parents and teachers as “daydreamers” (Dulcan & Lake, 2012). They may forget instructions of a school task in the middle of doing it or lose important items needed to complete a task (Wilmshurst, 2005). They often prefer activities that give them immediate reinforcement and have a low-level tolerance for delayed satisfaction (Ramsay & Rostain, 2014). Diminished academic performance, increased

early school failure and consequently low-self-esteem and increased mood problems are commonly associated problems for individuals with ADHD-I (Barkley, 1998).

Individuals with ADHD-I usually have fewer difficulties with peers and family members compared with other ADHD presentations (Wilmshurst, 2005) and significantly lower rates of conduct disorder compared to the other ADHD presentations (Capdevila-Brophy et al., 2014).

About 20 % - 30 % of the population with ADHD present predominantly inattentive symptoms (Wilens et al., 2002). However, there is an increased prevalence rate for ADHD-I compared with ADHD-HI across later developmental stages (adolescence and adulthood), with 90%-95% of adolescents and adults with ADHD having inattention symptoms as a major component of their disorder (Wilens & Spencer, 2010). Age is significantly associated with declining hyperactive/impulsive symptoms (Biederman et al., 2010), while the inattentive symptoms persist for a much longer time in individuals with ADHD from childhood to adulthood (Cherkasova, Sulla, Dalena, Pondé, & Hechtman, 2013; Wilens et al., 2009). People with ADHD-I are often underdiagnosed compared to those with ADHD-HI and ADHD-C (Wilmshurst, 2005).

#### *1.4.4.3 ADHD Combined Presentation (ADHD-C)*

For a diagnosis of ADHD Combined presentation (ADHD-C) presentation both the criteria for Inattentive presentation and Hyperactivity/Impulsivity must be met for the past six months. ADHD-C is the most common presentation in clinically referred samples of young people (Connolly & Bernstein, 2007), which typically has more severe symptoms and is relatively more stable across developmental stages compared with the other presentations (Lacramioara & Eugene, 2007; Wilens et al., 2002; Wilens & Spencer, 2010).

These individuals have difficulty not only with fidgeting, talkativeness, restlessness, and impulsivity, but also getting distracted off task, lack persistence, have difficulty sustaining focus and being organised. These symptoms can have a significant impact on their relationships with others, ability to undertake study, ability to make important decisions considering the long-term consequences, holding down jobs and places them at greater risk of potential harm (due to their impulsivity, and/or hasty actions that occur in the moment without forethought).

Biederman, Faraone, Milberger, Jetton, et al. (1996) conducted a prospective four-year follow-up study of children with ADHD-C and found significant differences between children with ADHD-C and healthy control participants in rates of behavioural, mood and anxiety disorders, increasing markedly from baseline to follow-up assessments. In addition, children with ADHD had significantly more impaired cognitive, family, school and psychosocial functioning than did the healthy control group. Most young people with ADHD-C have one or more comorbid conditions (Polanczyk et al., 2007; Wilens & Spencer, 2010) which leads to individuals with ADHD-C often experiencing higher levels of functional impairments (Cahill et al., 2012; Wilens et al., 2002).

### 1.4.5 ADHD Diagnostic Challenges

Several issues affecting the diagnosis of ADHD have been identified. ADHD is a complex neurodevelopmental condition with vast differences in clinical presentations (Willcutt et al., 2012). However, not only are there differences between individuals with the diagnosis, there is also instability of ADHD in the ADHD presentation across key developmental stages for a given individual with ADHD (Willcutt et al., 2012).

The requirement of some impairment from the symptoms in two or more settings is intended to avoid including situational-specific problems (for example, only at home). However, there is often low agreement between parent and teacher reports (Sayal & Goodman, 2009), therefore situational variation in ADHD presentation across different environments (home and school) is common, making it difficult to assess how significant the difficulties are across different settings (Willcutt et al., 2012) and therefore meeting this criterion.

Other conditions that mimic core ADHD symptoms, including hearing/vision problems, neurological and/or endocrine disorders, sleep disorders, and family dysfunction/social disadvantage/cultural marginalization (Buttross, 2009) can complicate the diagnostic process and must be assessed.

Individuals often suffer from psychiatric comorbidities, which affect the presentation of ADHD, can confound the diagnosis (Steinhoff, 2008) and contribute to the overall functional impairments. ADHD symptoms can also present in a similar way to other diagnoses. Take for example the diagnostic criteria for Generalized Anxiety Disorder which includes symptoms of restless or feeling keyed up or on edge and

difficulty concentrating or mind going blank (APA, 2013); Bipolar Disorder, Manic or Hypomanic Episodes which includes being more talkative than usual, flight of ideas, distractibility, excessive involvement in activities; or Acute Stress Disorder which includes problems with concentration (APA, 2013). Also, it may be easy to mistake trauma symptoms for ADHD symptoms: hyper-vigilance and dissociation could be mistaken for inattention; and/or impulsivity might be brought on by a stress response (Glod & Teicher, 1996). Therefore, a comprehensive and multidimensional assessment process to diagnose ADHD is always recommended (Souza, Serra-Pinheiro, Fortes, & Pinna, 2007).

### 1.4.6 Summary

ADHD is a neurodevelopmental disorder that is defined by impairing levels of inattention, disorganization, and/or hyperactivity-impulsivity that interferes with functioning or development (APA, 2013). It is commonly diagnosed after a comprehensive assessment of gathering information, from multiple informants, over one to three sessions with a qualified professional. Information is often collected using a combination of: a clinical interview with history taking and screening for associated comorbidities; observation; examination; and a variety of assessment tools (checklists, behaviour questionnaires, and/or rating scales). Two diagnostic classification systems are used for ADHD: DSM; and ICD. Although the list of symptoms and the evaluated constructs are very similar, they construct the diagnosis differently. In Australia, it is common for the DSM classification criteria to be used within Mental Health settings, so DSM-5 criteria will be used for this research. The present DSM-5 diagnostic criteria for ADHD recognise three different presentations: predominantly Hyperactive-Impulsive (ADHD-HI), predominantly Inattentive (ADHD-I), and the Combined (ADHD-C) presentation. Individuals with ADHD-HI often present with difficulty sitting still, restlessness, tapping hands or feet, interrupting others, or making important decisions without consideration of long-term consequences. Individuals with ADHD-I often present with concentration difficulties, their mind wandering off task, lacking persistence, having difficulty sustaining focus and being disorganized. Individuals who meet criteria for both inattentive and hyperactive-impulsive presentations are diagnosed with ADHD-C, which is the most common presentation in clinics. ADHD-C typically has the most severe symptoms and a greater than chance association with comorbid disorders.

Challenges to the diagnostic process have been identified: the vast differences in clinical presentation between individuals with ADHD; the variable persistence of ADHD presentations across key developmental stages; situational variability of ADHD symptoms; and frequent comorbid conditions.

## 1.5 KEY AETIOLOGICAL RISK FACTORS FOR ADHD

### 1.5.1 Introduction

Historically, research has argued for the biological nature of ADHD, with genetic factors affecting the development of structural and functional neural circuits that play a crucial role in psychological and behavioural functions (Miranda, Marco, & Grau, 2007). After all, it is estimated that first order relatives in families with one ADHD member are seven times more likely to have ADHD (Pennington, 2002) and twin studies estimate ADHD heritability to be about 70% (Faraone, Perlis, et al., 2005). However, with epigenetics emerging in the 1990's, research has not just focused on genetics, but also the environmental factors that might modify epigenetic patterns. Some studies specifically have looked at the role that epigenetics play in the pathogenesis of psychiatric disorders (Abdolmaleky, Zhou, & Thiagalingam, 2015; Peedicayil, 2007, 2015, 2017). Such factors include: social isolation (Cole et al., 2007); childhood neglect/abuse (McGowan et al., 2009); childhood sex abuse (Beach, Brody, Todorov, Gunter, & Philibert, 2010); Post Traumatic Stress Disorder (Smith et al., 2011); and total life stress (Smith et al., 2011).

Therefore, to fully understand the aetiology and developmental course of ADHD, it is important to take an interactive biological and psychosocial approach. Bronfenbrenner and Ceci (1994) argued that the environment mediates the child's biological predisposition to develop ADHD by determining how the hyperactivity, impulsivity and inattention are understood and managed by the child's social system. The relation of genes-to-environment is transactional in nature, with ADHD being determined by genetic underpinnings, but the developmental course of the disorder being influenced by the interaction between biological and psychosocial risk factors (Bronfenbrenner & Ceci, 1994). Pennington (2002) notes that environmental and genetic factors can act as protective or risk factors in the development and the course of disorders such as ADHD.

For ADHD, several biological and environmental factors will be explored including individual child characteristics as well as psychosocial characteristics. However, this will not be an exhaustive review of the literature. For example, child biological factors such as molecular genetic associations, underlying neurobiological, underlying neurophysiological and neuroendocrinological factors will not be covered as they are not relevant for the aims and hypotheses of this thesis. Child functioning factors including temperament, intelligence, Working Memory, academic achievement, emotional regulation and social competency will be covered as they can potentially alter the developmental course of ADHD and are investigated in this thesis.

## 1.6 INDIVIDUAL CHILD CHARACTERISTICS FOR ADHD

### 1.6.1 Temperament

#### *1.6.1.1 Introduction*

Temperament broadly refers to consistent individual differences in behaviour that are biologically based and are relatively independent of learning, system of values and attitudes. Individual differences in temperament appear early in life and remain relatively stable over the lifespan (Chess & Thomas, 1999).

Temperament and psychopathology correlate consistently across childhood (De Pauw & Mervielde, 2010; Nigg, 2006). Four explanations for these associations have been proposed: *predisposition* (or vulnerability), that negative extremes in certain temperamental traits cause specific types of pathology; *complication*, that psychopathology influences temperament; *common cause*, that third variables are causing both temperament and psychopathology thereby producing correlations, or that psychopathologies are extreme versions of common temperamental traits (continuity explanation); and *pathoplasty/exacerbation*, that temperamental traits alter the course of psychopathology (De Bolle, Beyers, De Clercq, & De Fruyt, 2012; Nigg, 2006; Tackett, 2006). Temperamental dimensions can have a dual role that consists of predictive factors for the onset of psychopathology and as the basis for a better understanding of future developmental trajectories (Côté et al., 2009; Dougherty et al., 2011; Stringaris, Maughan, & Goodman, 2010).

Although the broad definition is generally agreed on, many classification schemes for temperament have been developed, and there is no consensus (Trofimova, 2016), causing significant implications for research findings in this area to be compared (Burke, Loeber, & Birmaher, 2002).

#### *1.6.1.2 Temperament and ADHD*

Certain temperament traits, in particular the anger component of negative emotionality (broad temperamental factor including fear, anxiety and sadness, as well as a tendency to become emotionally upset in the face of frustration), as well as effortful control (the focusing and shifting of attention, inhibitory control, perceptual sensitivity, and a low threshold for pleasure) are associated with ADHD (De Pauw & Mervielde, 2011; Eisenberg et al., 2005; Foley, McClowry, & Castellanos, 2008; Martel & Nigg, 2006). Wiersema and Roeyers (2009) demonstrated an association between lower ratings of effortful control, worse performance on an inhibitory control task, and higher ratings of ADHD symptoms. Rabinovitz, O'Neill, Rajendran, and Halperin (2016) also found that anger/frustration at ages 3-4 years, as well as executive control at age 6, predicted ADHD symptom severity at age 7.

Martel and Nigg (2006) assessed a sample of 179 children (92 with ADHD and 52 controls) they found reactive control was related to hyperactivity-impulsivity as rated by both parents and teachers. Effortful control was related to inattention in parent but not teacher data. They found that children with regulation deficits and a reactive motivational style were at risk for the development of ADHD.

Two distinct pathways to psychopathology have been observed, with fear predicting internalizing disorders and anger predicting externalizing disorders, including ADHD (Auerbach et al., 2008; Rothbart & Bates, 2007). Effortful control has also been linked to behavioural difficulties in children (Posne & Rothbart, 2000; Rothbart, 2007).

Some researchers suggest there could be a moderating effect of effortful control on the relationship between reactive temperament and ADHD (Eisenberg et al., 2005; Martel & Nigg, 2006) although most research is limited by a cross-sectional approach (Rabinovitz et al., 2016). Further longitudinal study in this area is needed.

### 1.6.2 Intelligence Quotient (IQ) and ADHD

#### *1.6.2.1 Introduction*

Human intelligence consists of the abilities to learn from experience, adapt to new situations, understand and handle abstract concepts and use knowledge to manipulate one's environment (Sternberg, 1977). An intelligence quotient (IQ) is a total score derived from a standardized test designed to assess human intelligence. IQ is a biological characteristic that could differentiate young people with ADHD as a group from the general population. Research has indicated in clinical samples that ADHD and lower IQ covary, while in epidemiological samples individuals with ADHD show the same spread of scores across the spectrum as the general population: from the intellectually disabled range through to the superior range, with the greatest number of individuals in the average range. Several studies have reported an association between impaired IQ and ADHD in children, while others have not, suggesting further systemic research in this area is required.

#### *1.6.2.2 IQ Test Measurement*

IQ tests have moved considerably from the days when scientists Paul Brocca (1824-1880) and Sir Francis Galton (1822-1911) thought they could determine intelligence by measuring the size of the human skull (assuming the larger the skull, the smarter the person) or Wundt (1932-1920) assessing introspection (the human ability to reflect on their own thoughts). However, defining Intelligence is the subject of ongoing controversy in developmental psychology. There have been a vast range of intelligence definitions and assessment approaches, from psychometric measurement through to the qualitative description of the multiple forms of intelligence (Goldstein, 2013). The main criticism is that standardised psychometric tests are only able to capture a limited number of individual intellectual abilities, thereby ignoring multiple different "intelligences", as well as the significant effects of culture, the environment and differences in developmental processes (Neisser et al., 1996).

Nevertheless, the current most universally accepted approach to measuring IQ involves psychometric measurement (Binet, 1907 and Wechsler, 1985 as cited in Goldstein, 2013 ). The first modern intelligence test was developed in 1904, by Alfred Binet and Theodore Simon, the Simon-Binet IQ test. This test consisted of several

components such as logical reasoning, finding rhyming words and naming objects. Since then several editions of psychometric assessments have been developed that provide a comprehensive evaluation of both verbal and visuospatial intelligence. The two prominent IQ tests currently used for children and adolescents are: the revised version of the Wechsler Intelligence Scale for Children (Wechsler, 2016); and the Stanford Binet (Roid & Barram, 2004). Other assessments used include: the McCarthy Scales of Children's Abilities (MacCarthy, 1972); Cognitive Assessment System (1997, as cited in McGill, 2015); Kaufman Assessment Battery for Children (Kaufman & Kaufman, 1983, 2004); and Reynolds Intellectual Assessment Scales (Reynolds & Kamphaus, 2003). Some measures have been developed to measure specific skills, for example: the Peabody Picture Vocabulary Test (Dunn, Dunn, Bulheller, & Häcker, 2007) which measures verbal intelligence skills; and the Raven's Progressive Matrices (De Lemos & Raven, 1989) which measures non-verbal visuospatial skills.

Both the Wechsler tests and the Stanford-Binet test have similar subtests that measure Verbal skills and Performance (non-verbal) skills. The Wechsler test consists of five Primary Index Scores that make up the Full Scale IQ score (designed to measure overall intelligence): Verbal Comprehension (the ability to verbally reason); Visual Spatial (the ability to understand visual details in relationships in order to solve puzzles and construct geometric designs); Fluid Reasoning (the ability to detect relationships among visual objects); *Working Memory* (the ability to register, maintain, and manipulate visual and auditory information); and Processing Speed (the speed at which a child can accurately make decisions). The Stanford-Binet test also measures five factors of cognitive ability and gives a combined score, the intelligence quotient: Fluid Reasoning (the ability to solve problems in which no prior knowledge is required); Knowledge (ones accumulated stock of general information that has been committed to long-term memory); Quantitative Reasoning (measures a person's numeracy); Visual-spatial Processing (the recognition of both patterns and spatial relationships and the ability to recognize the whole from its constituent parts); and Working Memory (the multiple processes that capture, sort and transforms information in a person's short-term memory).

The Full-Scale IQ (FSIQ) score is determined by a formula that sums the Verbal (VCI) and Performance (PRI) IQ scores. The median raw score of the norming sample is defined as FSIQ 100 and scores each standard deviation (SD) up or down are defined as 15 IQ points greater or less. Approximately two-thirds of the population scores are

between FSIQ 85-115. About 2.5 percent of the population scores above 130, and 2.5 percent below 70. Scores above 130 are reported in the superior or "gifted" range, scores 120-129 are reported as the "very high" range, scores 110-119 are reported in the "high average" range, scores 80-90 are reported in the "average to low average". A score between FSIQ 70-79 signals "borderline" mental functioning and lower than FSIQ 69 signals an "intellectual disability".

Patterns of correlation tend to be consistent for a given individual over time. Male and female FSIQ scores do not differ in the general population, although there are gender differences in some components of the IQ tests, females tend to perform better in Verbal IQ components (Chung & Auger, 2013; Neisser et al., 1996), while males generally perform better in Performance (non-verbal) tasks (Neisser et al., 1996). However, these differences could result from different sociocultural expectations and experiences for males and females in our community (Neisser et al., 1996).

Wechsler (2016) states that IQ only forms part of a comprehensive assessment of an individual's strengths and weaknesses and should be interpreted in the context of their life and current circumstances. Therefore, it should never be used in isolation.

#### *1.6.2.3 IQ Scores in Children with ADHD*

People with ADHD have typically been found to span the same FSIQ range as the general population (low to high in the FSIQ range) (Biederman, Fried, Petty, Mahoney, & Faraone, 2012; Kaplan, Crawford, Dewey, & Fisher, 2000; Loge, Staton, & Beatty, 1990), with most individuals with ADHD falling in the average range (Weyandt & Gudmundsdottir, 2015). Investigation of monozygotic twins with and without ADHD found no significant differences in FSIQ scores (Sharp et al., 2003).

When investigators have looked at different subtests for children with ADHD, compared to children without ADHD, they have produced variable and inconsistent findings. Many report there are no significant differences between young people with ADHD and without ADHD (Castellanos, Sonuga-Barke, Milham, & Tannock, 2006; Huang-Pollock, Nigg, & Carr, 2005; Jonsdottir, Bouma, Sergeant, & Scherder, 2006; van Mourik, Oosterlaan, & Sergeant, 2005). However, several studies have reported an association between ADHD and: discrepant Verbal and Performance IQ scores (Frazier, Demaree, & Youngstrom, 2004; Max et al., 2003; Toplak, Rucklidge, Hetherington, John,

& Tannock, 2003; Vance, Maruff, & Barnett, 2003); lower Working Memory abilities (Brocki, Nyberg, Thorell, & Bohlin, 2007; Friedman et al., 2007; Nigg, 1999; Schuck & Crinella, 2005; Slaats-Willemse, Swaab-Barneveld, de Sonneville, van der Meulen, & Buitelaar, 2003; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005); and deficits in Processing Speed (Calhoun & Mayes, 2005; Schuck & Crinella, 2005).

Calhoun and Mayes (2005) found children with Neurodevelopmental Disorders (ADHD, Autism, Bipolar Disorders, and Learning Disorders) had lower Processing Speed Index scores (PSI) and Freedom from Distractibility Index scores (FDI) suggesting they have more impairments in attention, learning, processing speed and writing skills.

### 1.6.3 Executive Functioning and Working Memory

#### *1.6.3.1 Introduction*

Executive Functioning has been suggested to be important to assess in children and young people with ADHD (Klingberg et al., 2005). Executive Function is a term for several inter-related cognitive processes needed for purposeful, goal-orientated behaviour (Anderson, 2001; Lerner & Lonigan, 2014). Executive Functioning is responsible for paying attention, organizing, and planning, initiating tasks and staying focused on them, regulating emotions and self-monitoring (keeping track of what you're doing). Working Memory is one component of Executive Functioning, which is important for learning and academic performance. Working Memory is defined as a limited capacity system that temporarily stores and manipulates information while performing complex tasks (Baddeley, 2010). Typically, four components of Working Memory have been studied: verbal storage; verbal central executive; visuo-spatial storage; and visuo-spatial central executive. These components come from the Working Memory theory developed by cognitive psychologist Aaron Baddeley (Baddeley, 1986). The verbal and visuo-spatial storage components are responsible for the short-term storage and rehearsal of visual and visuo-spatial information respectively (Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005). The Central Executive component is thought to be an attentional controller responsible for oversite and coordination of the two storage systems (Martinussen et al., 2005). The functions of the Central Executive component are believed to include focusing attention, dividing attention among concurrent tasks, and providing

an interface between Working Memory and long-term Memory (Martinussen et al., 2005).

#### *1.6.3.2 Executive Function, Working Memory and ADHD*

Children and adolescents with ADHD have persistent trouble staying on task, difficulties in avoiding distractions, and acting impulsively without planning. Research on ADHD has shown impairments in inhibitory control (Barkley, 1997; Nigg, 2000) and Working Memory (Castellanos & Tannock, 2002; Martinussen et al., 2005). (Pennington & Ozonoff, 1996) found that in 83% of the papers they reviewed looking at Executive Function processes (attention, planning, goal directed behaviour and Working Memory) significant differences in Executive Function were apparent between the typically developing control and ADHD groups.

Working Memory has been proposed as a core deficit in young people with ADHD (Kasper, Alderson, & Hudec, 2012; Rapport, Chung, Shore, & Isaacs, 2001). Research indicates that Working Memory is important for learning (Alloway & Alloway, 2010; Gathercole, Pickering, Ambridge, & Wearing, 2004), and Working Memory deficits have been linked to learning problems and poor school performance in children with ADHD (Alloway & Alloway, 2010; Alloway, Elliott, & Place, 2010; Schreiber, Possin, Girard, & Rey-Casserly, 2014). Schreiber et al. (2014) found that Working Memory ability was significantly worse in children with ADHD compared with an age and gender matched healthy control group. Kempton et al. (1999) found children with ADHD were significantly more impaired on tasks assessing Spatial Short-Term Memory, Spatial Working Memory, set-shifting and planning than carefully age-, gender- and IQ-matched typically developing control children.

Martinussen et al. (2005) conducted a meta-analysis of research on Working Memory impairments in children with ADHD compared to Working Memory performance in typically developing children, looking at twenty-six studies. In all but two studies, children on medication had discontinued it for at least twenty-four hours prior to testing. They looked at the four separate Working Memory components (verbal storage; verbal central executive; visuo-spatial storage; and visuo-spatial central executive). The results indicated that Working Memory in children with ADHD was impaired relative to comparison children on all four components. Differences in the verbal domain were moderate in size while those in the visuo-spatial domain were large

(Martinussen et al., 2005). Group differences in Working Memory were larger in those studies that controlled for reading disability or language impairments (Martinussen et al., 2005). From these findings it is unclear if academic problems in children with ADHD result from Working Memory deficits rather than being a direct consequence of inattention and/or hyperactivity-impulsivity. Therefore, interventions assisting with Working Memory deficits maybe important for reducing academic impairments in children with ADHD. Further systemic research in this area is required.

### 1.6.4 Academic Achievement in Children with ADHD

#### *1.6.4.1 Introduction*

Academic achievement represents performance outcomes that indicate the extent to which a person has achieved specific goals that were the focus of activities in educational institutions (school, college, and university). It can be measured by looking at goals met across multiple subject areas (for example, critical thinking) or the acquisition of knowledge in specific domains (for example, typically literacy and numeracy for primary and secondary school students). Academic achievement is an important predictor of SES later in life, and wellbeing and health in adulthood (Huisman, Kunst, & Mackenbach, 2005). Hence it is an important factor to examine in individuals with ADHD. It is known that the severity of ADHD symptoms are correlated with a reduced level of academic attainment and increased rates of school rejection. Children with ADHD have a greater than chance association with spelling, reading and mathematics learning difficulties. However, academic achievement has a complex and dynamic interrelationship with cognition, behavioural, emotional dysfunction, and environmental factors (for example, parenting skills) which all need to be considered when looking at academic achievement outcomes for individuals with ADHD.

#### *1.6.4.2 Academic Achievement and ADHD*

Frazier, Youngstrom, Glutting, and Watkins (2007) completed a review and meta-analysis of individuals with ADHD and achievement. Based on calculated effect sizes in all relevant studies published between 1990 and 2006 (72 studies) individuals with ADHD had a significant lower academic achievement than non-ADHD individuals. Polderman, Boomsma, Bartels, Verhulst, and Huizink (2010) completed a prospective

study to address the predictive validity of ADHD. They found evidence for a negative prospective relationship between ADHD and academic achievement, with the strongest relationship present for the inattentive symptoms of ADHD, consistent with other researchers (Baumgaertel, Wolraich, & Dietrich, 1995; Carlson & Mann, 2000; Loe & Feldman, 2007; Milich, Balentine, & Lynam, 2001). One explanation might be that symptoms of hyperactivity decline with increasing age, while inattentive symptoms show a relatively constant pattern overtime (Biederman, Mick, & Faraone, 2000; Larsson, Lichtenstein, & Larsson, 2006). One four-year-follow-up study by Biederman, Faraone, Milberger, Curtis, et al. (1996) did find a significant relationship between academic performance and hyperactivity while controlling for IQ and SES. Overall, DuPaul and Langberg (2015) report academic underachievement point prevalence rates for young people with ADHD of 50% to 80%.

It is reported that ADHD symptoms interfere with academic achievement via problems sustaining attention, difficulties focusing on task instructions, having disorganised notes and written reports, higher rates of off-task physical activity (motor overactivity of hands and feet) that all result in poor test performance and suboptimal development of academic skills (DuPaul & Stoner, 2014). Young people with ADHD have a significantly reduced level of academic attainment linked to higher rates of suspension (Bauermeister et al., 2007; Daley & Birchwood, 2010), early school leaving and increased rates of placement in special classes with additional tutoring (APA, 2013; Barkley, DuPaul, & McMurray, 1990; Loe & Feldman, 2007; Rennie, Beebe-Frankenberger, & Swanson, 2014; Semrud-Clikeman, Pliszka, & Liotti, 2008).

DuPaul and Langberg (2015) report that many children with ADHD who enter kindergarten are already significantly behind their same age peers in basic maths and pre-reading skills. Then when they enter primary school they manifest significant delays in reading, maths and spelling, difficulties in written expression, difficulties completing homework, completing tests and producing work due to their high rates of off-task behaviour (DuPaul & Langberg, 2015). Similar results are found for young people in secondary school with ADHD showing impairments in reading, maths and spelling (Frazier et al., 2007). Latimer et al. (2003) and Breslau et al. (2009) argue that academic underachievement among children diagnosed with ADHD may be explained by a failure to develop basic skills during the early grades that are necessary for the development of higher cognitive skills later in school.

The risk factors for academic underachievement in young people with ADHD remain only partially understood, as the core characteristics of ADHD are associated with complex interrelated cognitive, behavioural, and emotional dysfunctions that affect academic and social performance at school. Academic achievement is strongly positively correlated with IQ performance (Lassiter & Bardos, 1995; Neisser et al., 1996), with intelligence the single most powerful predictor of academic performance (von Stumm, Hell, & Chamorro-Premuzic, 2011). Impairments in Executive Functioning, especially Working Memory deficits have also been linked to low levels of self-control, poor emotional regulation and other cognitive deficits such as planning and organising abilities that are firmly related to academic underachievement (Bryce, Whitebread, & Szűcs, 2015; Castellanos & Tannock, 2002; Ferrin & Vance, 2014). Working Memory is strongly positively correlated with performance in Mathematics and English (St Clair-Thompson & Gathercole, 2006).

There are many other characteristics that influence academic achievement such as persistence in school, willingness to study (Neisser et al., 1996) and motivation (Daley & Birchwood, 2010). Emotional regulation difficulties can affect academic performance, specifically poor self-regulation involving attention, Working Memory, and/or inhibition difficulties (Becker, McClelland, Loprinzi, & Trost, 2014). Behavioural problems such as aggressiveness, oppositional behaviour, conduct problems, and hyperactivity can also predict academic underachievement (Metsäpelto et al., 2015). The importance of a capable teacher is emphasized (Polderman et al., 2010), as a good teacher achieves an environment in which children are engaged and motivated for learning, leading to positive school adjustments (Greenberg et al., 2003). Latimer et al. (2003) found that there was a developmental path between childhood ADHD and negative academic and behavioural outcomes in adolescence, which was mediated by measures of children's self-esteem and behavioural adjustment in middle school. Poor school performance can lead to an increased negative self-perception of their ability with increased frustration, and antagonism toward school with or without aggressive behaviour (Miles & Stipek, 2006).

Latimer et al. (2003) also report that poor maternal adjustment and poor parenting skills can contributed to negative outcomes in adolescence. Furthermore, Flouri (2007) outlines that impoverished environments, in terms of resources (for example, low income) or in terms of cognitive stimulation (for example, parental Depression or low parental education) also predict lower academic attainment in children with ADHD. Taken

together, these findings suggest that academic outcome is not only affected by the child's academic aptitude, but parent factors also influence children's success as they progress through school. Parenting skill and parental psychopathology will be discussed further later in this chapter; however, further research is needed in the area of academic success and family variables (for example, the extent to which families encourage and motivate their children to do well academically, or the amount of support and structure that is provided at home with respect to homework). Narrowing in on the types of parenting skills that contribute to children's academic success could help define specific goals in parent intervention programs for young people with ADHD (Deault, 2010).

### 1.6.5 Emotional Regulation and ADHD

#### *1.6.5.1 Introduction*

Emotional regulation is an important characteristic for human beings to develop, as it is crucial for long-term wellbeing, good personal relationships, and successful performance at work. It is generally a skill we develop from infancy through to adulthood. Individuals with ADHD can find it difficult to modify their emotions to accomplish their goals and can worsen their externalising and internalising problems. There is a strong association between ADHD and emotion dysregulation.

#### *1.6.5.2 Emotional Development and Emotional Regulation Abilities in Children*

Emotion regulation is the ability to respond to the ongoing demands of experience with the range of emotions in a manner that is socially tolerable and sufficiently flexible to permit spontaneous reactions, as well as the ability to delay spontaneous reactions as needed (Cole, Michel, & Teti, 1994). It is seen as an individual's ability to modify an emotional state to promote adaptive, goal-oriented behaviours (Thompson, 1994). It encompasses the processes that allow an individual flexibility to select, attend to, and appraise emotionally arousing stimuli. Emotions are complex involving interconnected physiological correlates (for example, changing heart rate, blood pressure, galvanic skin response and brain wave activity), behavioural reactions and cognitions (Tremblay, Brun, & Nadel, 2005). Emotional regulation generally develops from infancy through to adulthood, during early social and effective encounters (Tremblay et al., 2005) from

caregivers, teachers and peers (Shaffer, 2009). By the time a young person goes to school they are expected to have developed some self-soothing and self-settling skills. As children's speech and language abilities increase, so does their frustration tolerance and delayed gratification skills, and more sophisticated and effective cognitive and behavioural strategies for emotional regulation are developed (Shaffer, 2009). Children with good emotional regulation skills are better able to focus attention and control impulses, which in turn enables better academic and social skills to develop (Shaffer, 2009).

Emotion dysregulation arises when these adaptive processes are impaired, leading to behaviour that defeats the individual's interest. Shaw et al. (2014) define emotional dysregulation as encompassing: emotional expressions and experiences that are excessive in relation to social norms and context-inappropriate; rapid, poorly controlled shifts in emotion ("lability"); and anomalous allocation of attention to emotional stimuli. Children with lower emotional regulation skills appear more prone to developing externalising (for example, aggression) and internalising (for example, Anxiety, Depression and social withdrawal) problems (Shaffer, 2009). Emotional dysregulation is a symptom reported for many DSM-5 disorders such as: ADHD; Oppositional Defiant Disorder; Disruptive Mood Dysregulation Disorder; Depression; and Persistent Depressive Disorder (APA, 2013).

#### *1.6.5.3 Emotional Regulation Impairment and ADHD*

Emotional dysregulation is common in individuals with neurodevelopmental disorders, including ADHD (APA, 2013). In the early diagnostic criteria of ADHD, emotion dysregulation was placed above inattention among its symptoms (Clements, 1966), but it was not until in DSM-III it became an 'associated feature'. Epidemiological research has found a strong association between ADHD and emotion dysregulation (Becker et al., 2006; Sjöwall, Roth, Lindqvist, & Thorell, 2013; Stringaris & Goodman, 2009). Stringaris and Goodman (2009) in a population study found mood lability in 38% of children with ADHD, a ten-fold increase over population rates. Clinic-based studies in youth with ADHD report similar prevalence estimates of emotion dysregulation of 24% to 50% (Shaw et al., 2014).

Problem-oriented emotional regulation strategies differ in young people with and without ADHD (Schmitt, Gold, & Rauch, 2012). Many young people with ADHD demonstrate problems with aggression, resistance to correction from authority figures and 'inhibitory volition' linked to emotional regulation difficulties (Barkley, 2015). They also have problems with inhibition of frustration, impatience, anger and hostility that are highly associated with emotional regulation problems (Barkley, 2015). Bunford, Evans, and Langberg (2018) found young people with ADHD and emotional regulation problems experienced a low threshold for emotional excitability/impatience, behavioural dyscontrol in the face of strong emotions and inflexibility/slow return to baseline. No difference in difficulties were found as a function of ADHD subtype, gender or comorbid Oppositional Defiant Disorder (ODD) (Bunford et al., 2018).

Individuals with a combination of ADHD and emotional regulation difficulties have been found to be significantly more impaired in peer relationships, family life, occupational attainment and academic performance than those with ADHD alone (Wehmeier, Schacht, & Barkley, 2010) even after controlling for comorbid disorders (such as ODD) (Biederman, Petty, Dolan, et al., 2008). Long term prognosis of people with ADHD and emotional regulation difficulties is poor. Biederman, Spencer, et al. (2012) found that emotional dysregulation in children with ADHD was associated four years later with more psychiatric comorbidities, greater social impairment, and ADHD persistence, compared to individuals with ADHD without emotion dysregulation. A population-based study by Althoff, Verhulst, Rettew, Hudziak, and van der Ende (2010) of 2076 children found children with emotional dysregulation had increased rates of Anxiety Disorders and Disruptive Behaviour Disorders in adulthood compared to non-dysregulated children.

### 1.6.6 Social Competency and ADHD

#### *1.6.6.1 Introduction*

Social competence consists of social, emotional, cognitive and behavioural skills needed for successful social adaptation. It requires the ability to take another's perspective, learn from past experiences and apply that learning to social interactions. Young people with neurodevelopmental disorders, such as ADHD, have a greater risk for social competence deficits compared to typically developing children. Deficits in social

competence lead to negative social interactions that can impact on relationships, lead to early school leaving and vocational problems in adulthood.

#### *1.6.6.2 Social Competence*

During the 1980's, social competence became a major focus of research. Gresham (1987) identified three sub-domains of social competence: adaptive behaviour; social skills; and peer acceptance (which is often used to assess social competence). He proposed that when children have poor social competence it is usually due to one of four deficits: skill deficits (in which children did not have the knowledge or cognitive abilities to carry out a certain behaviour); performance deficits; self-control skill deficits; and self-control performance deficits (in which children had excessive anxiety or impulsivity that prohibited proper execution of the behaviours or skills they had). The definitions of the 1980's have been expanded to now acknowledge the age, situation and skill specificity implicit in social competence (Pellegrini & Blatchford, 2000).

Current approaches to social competence include: peer regard/status (how popular one is with their peers) (Rose-Krasnor, 1997); social skill competence (Stump, Ratliff, Wu, & Hawley, 2009); relationship (the quality of one's relationships and the ability to form relationships, where competence depends on the skills of both members of the relationship) (Rose-Krasnor, 1997); and functional approaches (context-specific that depend on the identified social goals and tasks; this approach focuses on the outcomes of social behaviour and the processes leading to those outcomes) (Rose-Krasnor, 1997). Therefore, the term 'social competence' covers a broader domain than the term 'social skills' (Vahedi, Farrokhi, & Farajian, 2012). Social competence involves not only the behaviour a person needs to display to participate successfully in a variety of social settings, but also an awareness of how this behaviour affects their surroundings and their sensitivity to the needs of others (Vahedi et al., 2012).

Better social competence is related to peer acceptance, emotional health, school readiness, interpersonal relationships, and social adjustment. Children with greater social competence are likely to do well in social and academic areas and demonstrate a higher psychological resilience (Vahedi et al., 2012). Children with deficits in social competence have been found to have reduced empathy for others, are less cooperative, have poorer conflict resolution skills, and display negative behaviour and problems in

their social interaction skills (Chen & Jiang, 2002; Gouley, Brotman, Huang, & Shrout, 2008).

#### *1.6.6.3 Social Competence in Children with ADHD*

Young people with ADHD have a greater risk for social competence deficits compared to typically developing children (Barkley, DuPaul, et al., 1990; Biederman, Fried, et al., 2012; Hoza & McQuade, 2015). This is not surprising given the core symptoms of ADHD (inattention and hyperactivity) would be expected to make effective functioning with peers difficult. Problems of inattention limit the opportunities to acquire social skills through observational learning (Cunningham, Siegel, & Offord, 1985) and attend to social cues necessary for effective social communication (Landau & Milich, 1988). Hyperactive and impulsive behaviours contribute to generally unrestrained and overbearing social behaviour that makes children with ADHD undesirable to peers (Whalen & Henker, 1992).

Peer status/relations has been the area most focused on in the ADHD literature. Peer relationships are where children learn cooperation, negotiation and conflict resolution – skills that are critical for effective social functioning throughout life (Hoza, 2007). Research has shown that the view of one's peers are more predictive of later psychological functioning than other variables typically used in mental health research, such as teacher ratings, grade, achievement scores, IQ or absenteeism (Cowen, Pederson, Babigian, Izzo, & Trost, 1973). Peer functioning is a heterogeneous construct, and generally separated into two dimensions: peer group acceptance; and friendship success (Parker & Asher, 1993). However, broad level social skills/competence is also important to successful peer functioning (Ladd, 2005). Typically, studies have found that young people with ADHD experience difficulties across all peer domains of social skills competence, peer status and friendship (Landau, Milich, & Diener, 1998; McQuade & Hoza, 2008).

Andrade and Tannock (2014) found that elevated inattention and hyperactivity/impulsivity symptomatology in children who ranged in age from six to ten years, predicted greater peer problems. Hoza et al. (2005) examined peer status in clinically diagnosed 7-9-year-old children with ADHD and found that 52% fell in the rejected category and less than 1% were of popular status. These figures were consistent with Pelham and Bender (1982) where they found that 82% of children with ADHD have

peer rejection scores one standard deviation or more above the mean, with 60% two standard deviations above the mean. Peer rejection however was not the only problem, compared to their classmates, children with ADHD were lower on social preference, higher on social impact, less well-liked, had fewer dyadic friendships and they were disliked by children of higher status within the peer group (suggesting a process of exclusion by more popular peers) (Hoza et al., 2005).

Research has looked at the differences in social competence difficulties between the three ADHD presentations. In a study completed by Mikami, Huang-Pollock, Pfiffner, McBurnett, and Hangai (2007) children with ADHD-C exhibited off-topic and hostile responses, while those with ADHD-I also engaged in off-topic conversations and displayed more memory related difficulties, exhibited a pattern of social withdrawal and lower hostility. Ohan and Johnston (2011) and Ronk, Hund, and Landau (2011) found ADHD-C and ADHD-HI, specifically with disruptive aggressive behaviour were associated with more social competence problems and peer rejection. Hodgens, Cole, and Boldizar (2000) noted that children with ADHD-C were nominated by their peers as argumentative and combative. They found that ADHD-C and ADHD-HI received lower social preference scores from peers in the classroom but not in the playground. Children with ADHD-I were more likely to be identified as very shy and socially withdrawn, particularly in the playground setting (Hodgens et al., 2000). Hinshaw (2002) found that girls with ADHD-I report greater social isolation, but less peer rejection experiences compared to those with ADHD-C. This research emphasises the importance of separating the different ADHD presentations when assessing peer rejection and social competence difficulties as they were found to be experienced differently by children with ADHD-I and ADHD-C.

### 1.6.7 Summary

To fully understand the aetiology and developmental course of ADHD, it is important to take an interactive biological and psychosocial approach. For ADHD, several biological and environmental factors, including individual child characteristics, as well as psychosocial characteristics are important, however this thesis does not present an exhaustive review of the literature. Only the factors that are relevant for the aims and hypotheses of this thesis will be covered: temperament; intelligence (IQ); Working Memory; academic achievement; emotional regulation; and social competence. Certain temperament traits, in particular the anger component of negative emotionality and

effortful control, have been found to be associated with ADHD. However, temperament research has been limited to cross-sectional studies and requires further longitudinal investigation. The IQ score is potentially one of a range of significant characteristics that could differentiate young people with ADHD, as a group, from the general population. In epidemiological samples, ADHD is associated with FSIQ scores across the spectrum from the intellectually disabled range through to superior range, as in the general population. However, examination of the different subtests of IQ scores in the ADHD population has produced variable and inconsistent findings: some researchers finding no significant differences; while others reporting significant differences in Verbal and Performance IQ, Working Memory, and Processing Speed. Young people with ADHD and Working Memory difficulties have been found to have more difficulties staying on task, becoming distracted and more impulsive leading to greater learning problems and poor school performance. Although no significant ADHD group differences in FSIQ are reported, many children with ADHD are underachievers academically, and some evidence suggests that inattention may be a major factor in the low academic performance of these children. Poor emotional regulation skills are common in individuals with ADHD, with research finding a strong association between ADHD and emotion dysregulation. Emotional dysregulation places these young people more at risk for impaired peer relationships, dysfunctional family life, academic under-performance and reduced occupational attainment. Young people with ADHD also have a greater risk for social competence deficits compared to typically developing children. Research has found that when a young person with ADHD has deficits with social competence, they are also at greater risk for experiencing family conflict, substance abuse, early school leaving and vocational problems in adulthood. These difficulties may differ for young people depending upon the ADHD presentation: children with ADHD-C exhibiting off-topic and hostile responses; while those with ADHD-I also engaged in off-topic conversations and displayed more memory related difficulties, with a pattern of social withdrawal and lower hostility. The research emphasises the importance of separating the different ADHD presentations when assessing peer rejection and social competence difficulties as they have been found to be experienced differently. Further longitudinal research is required in all areas looking at the individual child characteristics risk factors for ADHD.

## 1.7 KEY PSYCHOSOCIAL COMPONENTS FOR ADHD (FOR EXAMPLE, FAMILY FUNCTIONING, PARENTAL RELATIONSHIP FUNCTIONING, PARENTING SKILL, AND PARENTAL PSYCHOPATHOLOGY)

### 1.7.1 Introduction

Most aspects of mental illness and psychological well-being are influenced by social factors. Generally public health research has been interested in characteristics such as SES assessed by level of income or education, living or working conditions, gender and ethnicity (Marmot, Allen, & Goldblatt, 2010). Children and adolescents are not only influenced by the broader community characteristics, but also the family environment that accounts for a big proportion of their life's experiences. In the Child and Adolescent Mental Health literature a focus has been on the importance of: family functioning; parental relationship functioning; parenting skill; and parental psychopathology when looking at environmental aspects influencing the development of disorders. ADHD has been found to be both genetically and environmentally determined, with research indicating that 20-30% of phenotypic variability in symptoms are accounted for by environmental factors (Faraone et al. 2005). Most research shows that ADHD is associated with problematic family functioning, including: greater dysfunction within the family; lower levels of marital satisfaction; use of less effective parenting strategies; higher rates of parental psychopathology and stress, especially maternal Depression; and negative / conflicted parent-child relationships (Anastopoulos, Sommer, & Schatz, 2009; Deault, 2010; Johnston & Mash, 2001). These factors will be discussed.

### 1.7.2 Family Functioning

Families provide for children's needs and help them develop to become an integrated adult capable of living in society. How families function therefore, is an important variable in a child's life and has been an important area of study in the mental health field. It is often described in the literature as family dynamics. The definition of family dynamics is the scheme of family members' relations and interactions including many prerequisite elements (family arrangements, hierarchies, rules, and patterns of family interactions) (Al Ubaidi, 2017). Some dynamics are helpful and some unhelpful. "Healthy families promote the well-being and functioning of each individual family

member through the maintenance of clear and effective communication, mutually beneficial interactional patterns, clear boundaries between the generations and between family subsystems, and expectations that change over time to the internal demands of family members and external demands of the environment" (Bray, 1995). Family dynamics influence the way young people view the world and they will impact on their relationships/behaviours and their future wellbeing (Al Ubaidi, 2017). Specific to mental health, family plays a very significant role in the development of positive mental health and making a person psychologically resourceful and socially organized (David, 1978).

A dysfunctional family, is a family in which conflict, misbehaviour and even abuse on the part of individual members of the family occur continually, leading other members to accommodate such actions (Senthil, 2016). Dysfunctional families are often a result of alcoholism, substance abuse, or other addictions of parents, parents' untreated mental illnesses/defects or Personality Disorders, or the parents emulating their own dysfunctional parents and dysfunctional family experiences (Senthil, 2016). Violence and verbal abuse are typical outcomes. Individuals from dysfunctional families tend to have a higher incidence of behavioural disorders.

A major problem in the family assessment area is the lack of a unified theory of family functioning (Grotevant, 1989). There is no agreed family diagnostic system and there are many disagreements in the field about the constructs or processes that are essential to assess (Bray, 1995). The McMaster Family Assessment Device (FAD) developed by Epstein, Baldwin, and Bishop (1983) has been found to be a useful tool in the evaluation of family functioning for over 20 years in research (Grandi, Scortichini, & Fabbri, 2007). The McMaster Model is based on the six fundamental dimensions of family functioning: Problem Solving; Communication; Roles; Affective Responsiveness; Affective involvement; and Behavioural Control (Epstein et al., 1983).

### 1.7.3 Family Functioning and ADHD

Family functioning is an important factor for children with ADHD. If family members are aware of the difficulties that children with ADHD have controlling their behaviour and offer them opportunities to develop self-regulation skills and problem-solving skills, they can facilitate a positive course for the disorder (Miranda et al., 2007). However, many studies have associated poor family functioning with a child having

ADHD (Johnston & Mash, 2001). Cussen, Sciberras, Ukoumunne, and Efron (2012) found children with ADHD had lower parent-reported family quality of life (measured as emotional and time impact on the parent and impact on family activities). Mohammad pour and Kasaei (2013) compared 30 families of children with ADHD and 30 control families, finding the family functioning scores of families with an ADHD child were less healthy than those of the control group.

Families that are not responsive to the child's needs and/or lack the skills to cope with ADHD behaviour manifestations, can worsen the symptom severity (Miranda et al., 2007). Family interaction, adaptability and cohesion have been linked to the severity of the ADHD child's functional impairment and behaviour (Johnston & Mash, 2001). It has also been noted that children with ADHD with poor outcomes generally come from families with chronic family conflict and decreased family cohesion (Biederman, Milberger, et al., 1995; Cussen et al., 2012).

Little is known about the cause-effect relationship between family functioning associated with a child with ADHD and sources of stress associated with other family characteristics (for example, parents' characteristics). The results of some studies suggest that the characteristics of the child with ADHD are a main factor contributing to family stress (Anastopoulos, Guevremont, Shelton, & DuPaul, 1992; Johnston & Mash, 2001; Podolski & Nigg, 2001). Miranda et al. (2007) suggest that due to the more difficult temperament of children with ADHD and as a result of a complex conditioning process, the child becomes an aversive stimulus for their parents, which in turn increase the vulnerability of the family to stress. Some research has also suggested that the different presentations of ADHD might have different impacts on family life, with hyperactive/impulsive behaviours posing the greatest challenges to family functioning. Lacramioara and Eugene (2007) found that families of predominantly ADHD-I manifest more positive family functioning than either families of ADHD-C of ADHD-HI presentations. Longitudinal study designs are needed to help understand the various family variables involved in this complex relationships (Miranda et al., 2007).

### 1.7.4 Parental Relationship Functioning

Although there is a lot of literature that has shown that family structure is linked with child wellbeing, less is known about the extent to which positive mother-father

relationship quality is linked with children's outcomes (Goldberg & Carlson, 2014). The mother-father relationship is often perceived to be at the centre of good family functioning (Easterbrooks & Emde, 1988). Therefore, a parents' ability to communicate effectively, generate emotional closeness, and support each other's decisions likely have implications for their children's well-being and development (Goldberg & Carlson, 2014). Therefore, it is important to examine the impact that martial relationships can play on a child's development.

Psychoanalytic theory emphasises that martial relationships are important in developing a child's sense of security, and social learning theory suggests that children learn how to behave in relationships by observing parental interaction (Bandura, 1978; Fincham, 1998). The Society for Research in Child Development (2013) , looked at 251 children who reported on their exposure to marital conflict. They looked at how children's stress response system functioned by measuring respiratory sinus arrhythmia (RSA) as an index of activity in the parasympathetic branch of the body's stress response system. RSA has been linked to the ability to regulate attention and emotion. Children's ability to rapidly solve problems and quickly see patterns in new information was also measured. They found that children who had lower resting RSA and who witnessed more marital conflict, demonstrated less adaptive RSA reactivity. In addition, children with lower baseline RSA, whose stress response systems were also less adaptive, developed mental and intellectual ability more slowly.

Several behavioural problems during childhood have been linked to marital conflict and discord: lower completion of high school and attending university; greater risk of unemployment after leaving school; increased mental health problems; and criminal behaviour (Fergusson & Horwood, 1998; Fergusson, Horwood, & Ridder, 2005; McLeod & Kaiser, 2004; Needlman, Stevenson, & Zuckerman, 1991). Further research in understanding how parental relationship quality is linked to a child's long-term success is important.

The few studies that have examined the association between positive aspects of parents' relationship and children's behaviour have found that parent's supportiveness, positive affect and ability to communicate are positively linked with children's well-being (Conger, Rueter, & Elder, 1999; Goldberg & Carlson, 2014; Miller, Cowan, Cowan, Hetherington, & Clingempeel, 1993). Goldberg and Carlson (2014) found that positive

aspects of the parents' couple relationship (greater trust, empathy, and effective communication) have beneficial effects for children's externalizing and internalizing behaviours. However, these studies have limitations due to small and non-representative samples and do not include data across multiple time points (Goldberg & Carlson, 2014). Therefore, this is an area that requires further systemic study.

It is also important to note that the nature of the association between the quality of the parents' relationship and their children's behaviour may be bidirectional. For example, children's behavioural problems have been found to increase parent's stress and serve to erode parental relationships overtime (Cui, Donnellan, & Conger, 2007; Goldberg & Carlson, 2014; Schermerhorn, Cummings, DeCarlo, & Davies, 2007). These findings highlight the importance of also systemically examining how child behaviour/psychopathology affects the quality of the parental relationship over time.

### 1.7.5 Parental Relationship Functioning and ADHD

Several studies have reported a decline in marital satisfaction when parenting a child with ADHD (Anastopoulos et al., 2009; Belsky & Kelly, 1994; Cox, Paley, Burchinal, & Payne, 1999; Shapiro, Gottman, & Carrère, 2000; Zarei, Rostami, & Ghapanchi, 2010). Parenting stress can negatively impact on marital relationships causing parental conflict and marital dissatisfaction. Separation and divorce are more common in families with children with ADHD than healthy control participants (Barkley, DuPaul, et al., 1990). Carlson, Jacobvitz, and Sroufe (1995) conducted a longitudinal study and found hyperactivity at ages 6, 8 and 11 years was strongly predicted by marital relationship variables.

Johnston and Mash (2001) questioned if the poorer marital satisfaction in families was not a symptom of ADHD, but was more associated with child behavioural problems instead. Zarei et al. (2010) looked at 101 parents of children with ADHD and 60 parents of children without ADHD. They found that marital satisfaction of parents of children with ADHD was less than parents of children without ADHD. They found that parents who spent more time disciplining their ADHD child had little private couple time to help and care for each other. They hypothesised that the stressful and demanding nature of ADHD symptoms may elicit more marital miscommunications in parents. They also found that the education level of the parent was an important and protective factor in

marital satisfaction in parents of children with ADHD. Longitudinal studies are needed to provide more detailed information in this area.

### 1.7.6 Parenting Skill

One of the most important family system factors affecting children's development is parenting. Miranda et al. (2007) note that parenting is not a single concept but rather a multidimensional construct that includes aspects of the parent-child interrelationship (parent affect towards the child, general rearing style and discipline practices) as well as internal aspects like parents' feeling of competence and self-efficacy, parents' perception of their child behaviour and parental stress. All these parenting variables are closely interrelated (Hinshaw, 2002; Scheel & Rieckmann, 1998).

Hoghughi (1998) noted that parenting has three essential components: firstly **care**, which involves protecting children from harm while promoting emotional and physical health; secondly **control**, which involves setting and enforcing boundaries to ensure children's and others safety; and thirdly **development**, which involves optimising children's potential and maximising the opportunities for using it. Most recent definitions of effective parenting would include: developing and clarifying clear expectations; staying calm in the midst of turmoil when your child gets upset; consistently following through with positive and negative consequences; being a positive role model; role playing corrective behaviours; and praising your child for their appropriate behaviour (Lopez, 2004). Parenting skill is one of the most important factors in developing the parent-child relationship, in which child development unfolds, and in which the earliest foundations for risk and resilience emerge (Hawes, 2017).

However, much less is known about how children's symptoms of psychopathology impact on their mothers and fathers parenting and how parenting and child symptoms relate transactionally / interplay with one another over a span of several years. Kopala-Sibley et al. (2017) looked at parenting styles and child psychopathology over six years. They found that child externalizing symptoms at age three predicted maternal permissiveness and authoritarianism and paternal permissiveness at age six. Maternal permissiveness at age six predicted child externalizing symptoms at age nine, highlighting the importance of considering transactional models of parenting styles and child externalizing symptoms. Allmann (2018) collected data from 491 families on

mothers' and fathers' parenting styles (authoritarian, authoritative, permissive, and overprotective) and children's symptoms of psychopathology (ADHD, ODD, Depression and Anxiety). Analyses revealed that parents and children impact on each other in a bidirectional way, suggesting that child symptoms may compound over time partially because they decrease exposure to adaptive, and increase exposure to a maladaptive parenting style. Similarly, maladaptive parenting may continue to increase over time due to the persistence of child symptoms.

Further longitudinal research in this area is required, specifically looking at the role of fathers' parenting in the development of psychopathology and how children may elicit certain types of parenting from fathers (Allmann, 2018) as little is known in this area.

### 1.7.7 Parenting Skill and ADHD

Understanding the ways in which parents, through effective parenting, can help their children to regulate their emotions and behaviour, to facilitate friendships with peers and social networks, and to achieve academically is critical in understanding the pathways that shape different developmental trajectories for children with ADHD (Deault, 2010). Two areas commonly explored are: parenting stress; and the types of parent strategies used by parents. Maladaptive parenting strategies (including: less warmth and sensitivity; negative controlling parenting; inconsistent discipline; and low levels of parental involvement) and high levels of parental stress have been linked to children with ADHD (Cussen et al., 2012; Ellis & Nigg, 2009; Johnston & Mash, 2001; Keown, 2012; Modesto-Lowe, Danforth, & Brooks, 2008).

Decreased parenting confidence has been associated with ADHD (Cussen et al., 2012; Gordon & Hinshaw, 2017; Johnston & Mash, 2001). Roselló, García-Castellar, Tárraga-Mínguez, and Mulas (2003) in a study of 36 families with ADHD found that a high percentage of parents (between 75% and 88%) reported low self-efficacy, feelings of frustration, anger and rejection towards their children, and low levels of competence in their parenting role. Whalen et al. (2006) found that mothers of children with ADHD reported less quality interactions with their children, an elevated rate of anger when they were with their child, lower levels of perceived self-competence in their roles and less perceived quality of life. Miranda et al. (2007) also reported that parents of children with

ADHD perceived themselves as less competent in their parenting role than parents of children without ADHD.

McLaughlin and Harrison (2006) interviewed 150 mothers of children diagnosed with ADHD to assess their child's behaviour, their own parenting sense of competence, perceptions of social isolation and parenting practices. They found that the overall severity of their child's behavioural disturbance and parental sense of competence was associated with the use of less effective parenting practices. Barkley (1998) found that during task and free-play situations, mothers of children with ADHD were more negative and reprimanding, issued more frequent commands, and were less responsive to their children's requests for attention than mothers with children who did not have ADHD. Morgan et al. (2016) found those who were punished using spanking were significantly more likely to display severe levels of ADHD symptomatology.

Ellis and Nigg (2009) found that high levels of parental inconsistent discipline and low levels of parental involvement were associated with ADHD symptom severity. Hawes, Dadds, Frost, and Russell (2013) examined a community cohort of children with ADHD, aged 4 – 10 years, and found that across early childhood high levels of parental involvement were associated with reduced levels of hyperactivity/inattention. Across middle childhood, increases in the child's age were associated with increased levels of hyperactivity/inattention if they were exposed to high levels of inconsistent discipline. Suggesting that associations between specific parenting variables and hyperactivity/inattention may differ across the developmental periods of young people.

Poor parenting can create, maintain and exacerbate defiant behaviour (Johnston & Mash, 2001), and predict the development of oppositional and conduct problems in children with ADHD (Chronis et al., 2007; Johnston & Jassy, 2007), and their persistence (August, Realmuto, Joyce, & Hektner, 1999). Some studies suggest that ODD symptoms, not ADHD symptoms, predicted less positive parenting, including a lack of warmth and positive parental involvement, as well as reports of more negative discipline strategies and parental intrusiveness (Johnston & Mash, 2001; Kashdan et al., 2004; Pfiffner, McBurnett, Rathouz, & Judice, 2005). Evans, Sibley, and Serpell (2009) and Podolski and Nigg (2001) found that oppositional and delinquent behaviour by the ADHD child best predicted levels of caregiver strain. Both mothers' and fathers' distress were associated with oppositional symptoms and aggressive behaviour, while hyperactivity did

not impact parental distress (Podolski & Nigg, 2001). These findings suggest that preventing and treating these comorbid child behaviour problems should be a high priority when working with young people with ADHD and will be discussed further in Chapter 2.

It is important to note that the child's ADHD symptoms and parenting skill may work synergistically; a child with ADHD can impose a significant adverse effect on the family system, creating tension, stress and potentially disrupting family functioning (Johnston & Mash, 2001). Children with ADHD typically display more noncompliance, defiance, and difficulty following parental requests through to compliance (Chronis et al., 2003). The child's ADHD symptoms can influence parent behaviour and adjustment (Deault, 2010; Johnston & Mash, 2001).

Parents play an important role in delivery of pharmacological and behavioural treatments for children and adolescents with ADHD. They are responsible for administering medication and implementing the behaviour management strategies. Low parenting self-esteem and skill were associated with a poorer response to behavioural, pharmacological, and combined treatments in the Multi-Modal Treatment Study (MTA) for ADHD (Hoza et al., 2000), suggesting the importance of further research in this area.

In contrast, positive parenting predicts fewer future conduct problems and is a known protective factor for the developmental course of conduct problems in children with ADHD (Chronis-Tuscano et al., 2011). Further longitudinal research looking at risk and protective factors is needed.

### 1.7.8 Parental Psychopathology

Parent psychopathology is an important risk factor for children's functioning (Chronis et al., 2003). Theoretical models suggest that parent psychopathology may place children at risk for behavioural, emotional, and social difficulties through several processes including: shared genetics; less available or poor parenting; exposure to parents' maladaptive cognitions, affect, and behaviour; and exposure to stressful environments (Dodge, 1990; Goodman & Gotlib, 1999; McCarty & McMahon, 2003). There is extensive research linking parent psychopathology to child functioning (Breaux, Harvey, & Lugo-Candelas, 2014). Most studies have focused on maternal Depression, which has been linked with children's externalizing and internalizing problems and social

skill deficits (Chronis et al., 2007). Other areas have also looked at the importance of parental Anxiety, substance abuse, Antisocial Personality Disorder, Borderline Personality Disorder and ADHD in children's development (Breaux et al., 2014). Less research has focused on co-occurring parental psychopathology and child's functioning or parental Personality Disorders and child functioning. However some have found that parents with two co-occurring disorders had children with more difficulties than did parents who had a single disorder (Breaux et al., 2014; Carter, Garrity-Rokous, Chazan-Cohen, Little, & Briggs-Gowan, 2001).

### 1.7.9 Parental Psychopathology and ADHD

The presence of parental psychopathology is common when looking at children with ADHD (Johnston & Mash, 2001; Modesto-Lowe et al., 2008). Research reveals that mothers of children with ADHD experience higher levels of anxiety and depressive symptomatology, than do mothers of children without ADHD (Cussen et al., 2012; Mash & Johnston, 1990; Miranda et al., 2007; Nigg & Hinshaw, 1998). Mothers of children with ADHD report more self-blame and isolation (Mash & Johnston, 1990), fathers also report more depressive symptoms and perceive their families as less supportive (Brown & Pacini, 1989). Harrison and Sofronoff (2002) report that depressive symptoms in parents with children with ADHD may be associated with feelings of "learned helplessness" as a result of their inability to change their children's behaviour in spite of all their efforts. Also due to parental Depression, parents do not have the energy required to fulfil their parenting role, resulting in parents being caught in a vicious circle that is difficult to break out of. Chronis et al. (2003) report the odds of maternal lifetime Major Depressive Disorder were 2.22 times higher in the group where children had ADHD. Child ADHD was also significantly associated with paternal childhood ADHD (Biederman et al., 1992; Chronis et al., 2003) and with greater odds of maternal cocaine and stimulant dependence (Chronis et al., 2003).

Studies have reported that parental psychopathology is a risk factor for more severe and persistent child and adolescent ADHD symptoms. Morgan et al. (2016) found that those raised by mothers with depressive symptoms, experiencing emotional problems or substance abuse were significantly more likely to later display severe levels of ADHD conduct problem symptomatology. Biederman, Petty, Clarke, Lomedico, and Faraone

(2011) conducted an 11-year follow-up study to evaluate predictors of persistence of ADHD. They found 78% of children with ADHD continued to have full (35%) or a partial persistence of core ADHD symptoms: the predictors of persistence were severe impairment of ADHD; psychiatric comorbidity; and exposure to maternal psychopathology at baseline.

Parental psychopathology has been found to have negative effects on parent management practices. Kashdan et al. (2004) found that parental Anxiety was related to negative parenting practices. The authors speculated that parental Anxiety might make parents particularly vulnerable to significant distress, with reciprocal interaction patterns between parents and children contributing to negative interpersonal styles, distress and impairment of both children and parents (Kashdan et al., 2004). Chi and Hinshaw (2002) investigated the role of maternal depressive symptoms and the mother-child relationships of children with ADHD. Mothers' levels of depressive symptoms predicted negative biases in their reports of their child's ADHD symptoms, general behaviour problems, their own perception of a negative parenting style (not supported by laboratory observations) and problematic parent-child interactions (not preparing their children for upcoming demands, as well as inadequate levels of warmth and overall competence in parent-child interactions).

Several studies have noted positive associations between levels of maternal depressive symptoms and mother's ratings of child behavioural problems, usually rating their child with more externalizing and internalizing problems and lower social skills than children of parents without depressive symptoms (Boyle & Pickles, 1997; Breaux et al., 2014; Chi & Hinshaw, 2002; Chilcoat & Breslau, 1997; Fleck et al., 2015; Mineka, Watson, & Clark, 1998; Najman et al., 2000). McDermut, Haaga, and Bilek (1997) suggested that if a parent is Depressed, with distorted perceptions and cognitions and with difficulties in communication they may have problems with objective evaluations of child behaviour. This could be due to increased conflictual and/or negative parent-child interactions or distorted perceptions of the severity of the child's behaviour. This then poses a problem if the parent is the only informant about the child's emotional and behavioural disturbances (Chi & Hinshaw, 2002). Breaux et al. (2014) found that fathers who reported experiencing more types of psychopathology symptoms had children with higher maternal ratings of internalizing problems, however, did not themselves rate their children as having more difficulties. Additional longitudinal research is needed to better

understand the mechanisms underlying the relationship between reports of parent symptoms and child functioning. For example, it may be that: the parent's interpersonal styles are modelled in the parent-child relationship causing more severe difficulties in the child's behaviour; or that parents with these symptoms experience less social support, disrupting parenting and then impacting on child functioning (Breaux et al., 2014); or the parents perception of the severity of the child's behaviour is distorted.

In addition, parental Depression has also been shown to predict family discord and behaviour problems in children with ADHD (Cunningham, Benness, & Siegel, 1988). Parental psychopathology not only carries a genetic predisposition risk for children to develop mental health issues, it also carries significant environmental risks in decreased parents' perception of their parenting skills and impacts on the parent-child interactions which can lead to the development of ODD and/or CD (Biederman et al., 2011). Maternal Depression can predict conduct problems 2-8 years following the initial assessment for ADHD (Chronis et al., 2003; Chronis et al., 2007) and persistence of ODD (August et al., 1999). Some studies suggest that parental psychopathology is more likely to be associated with oppositional and conduct problems than with ADHD alone (Chronis et al., 2003; Pfiffner et al., 2005). This will be discussed further in Chapter 3.

Similarly, parent self-esteem and parenting efficacy, parental Depression and marital distress predict poorer compliance with (McMahon, Forehand, Griest, & Wells, 1981) and response to (Griest & Forehand, 1983) parent training for noncompliant and aggressive children. Also, high levels of maternal ADHD symptoms interfere with improvements shown by children with ADHD following parent training (Sonuga-Barke, Daley, & Thompson, 2002). Parents with ADHD may have trouble consistently adhering to a treatment plan, may be disruptive or inattentive during parent training session, and may forget to administer their children's medication (Weiss, Hechtman, & Weiss, 2000). Therefore, for it clinically important to assess and treat parental psychopathology at the same time as treating the child or adolescent.

Current research is looking at the bidirectional relationship between parental psychopathology / parent stress and ADHD. Breaux and Harvey (2019) examined 197 preschool children with ADHD over a three-year longitudinal study. They found greater maternal overactive parenting and life stress were predictive of more ADHD symptoms, and greater child ADHD symptoms significantly predicted greater maternal life stress and

depressive symptoms and lower warmth. They reported that child effects were evident for fathers' Depression and life stress, but these did not remain controlling for parental ADHD symptoms.

Areas for further research should include more fathers, as they have been underrepresented in the literature on parent psychopathology and child functioning (Phares, Fields, Kamboukos, & Lopez, 2005) and longitudinal studies looking at the role of parental psychopathology and child functioning that might provide stronger evidence for a causal relationship (Breaux et al., 2014).

### 1.7.10 Summary

Most recent research shows that ADHD is associated with problematic family functioning, including greater stress within the family and marital relationship, higher rates of parental psychopathology and conflicted parent-child relationships. Although a great deal of progress has been made, a lot is still unknown as to how ADHD plays out in families (Breaux & Harvey, 2019). The impact of the child's ADHD symptoms on these factors needs further exploration, as it is more likely that the child's ADHD symptoms and the family's functioning work synergistically. Literature reviews also suggest that an ADHD diagnosis alone is not enough to predict the outcomes in these domains, for they appear to be more exacerbated in children with comorbid oppositional and conduct problems. In addition, the influence of factors such as gender, culture, and ADHD presentation on the associations between ADHD and family factors remains largely unknown (Johnston & Mash, 2001). These findings suggest that interventions should target not only the child's ADHD symptoms, but also family and marital functioning, parenting practices, as well as parental psychopathology.

## 1.8 KEY COMORBID CONDITIONS FOR ADHD

### 1.8.1 Introduction

Co-occurring mental health problems are the norm, rather than the exception, among children and adolescents with ADHD in both community and clinic samples (Barkley, 2006; Becker, Luebbe, & Langberg, 2012). ADHD is consistently shown to

have a greater than chance occurrence with a number of other psychiatric conditions including Anxiety Disorders, Depressive Disorders and other Disruptive Behaviour Disorders, which include CD and ODD (Angold, Costello, & Erkanli, 1999; Becker, Luebbe, et al., 2012; Biederman, Faraone, Milberger, Curtis, et al., 1996), suggesting that ADHD places young people at risk for the development of other mental health problems (Wilens et al., 2002). Other comorbid problems associated with ADHD include motor development impairments, Developmental Coordination Disorder, poorer school performance, lower IQ, speech and language impairments and social problems (Barkley & Murphy, 2006). Marked difficulties in emotion regulation in some children with ADHD have also been described (Melnick & Hinshaw, 2000). Comorbid conditions can play a significant role in the manifestation of ADHD symptomatology and varied outcomes for the young person.

### 1.8.2 Oppositional Defiant Disorder (ODD), Conduct Disorder (CD) and ADHD

#### *1.8.2.1 Introduction*

Diagnoses involving disruptive or externalizing behaviours (CD, ODD) and ADHD are often reported together in the literature. In studies of community and clinic samples a large percentage of youth with CD or ADHD (for example, 45-70%) also meet criteria for the other disorder (Fergusson, Horwood, & Lloyd, 1991; Offord, Boyle, & Racine, 1991). Although ODD and CD are often grouped together for research purposes, differences in the ADHD literature for each disorder suggests that ODD and CD should be looked at independently. This section will explore why ODD and CD should be reported separately and discuss ADHD and CD. Chapter three will focus on ADHD and ODD.

CD is diagnosed when a person shows a repetitive and persistent pattern of behaviour in which the basic rights of others or major age-appropriate societal norms or rules are violated. DSM-5 (APA, 2013) requires three of the following fifteen criteria in the past twelve months, with at least one criterion present in the past six months: *aggression to people and animals* (often bullies, threatens, or intimidates others, often initiates physical fights, has used a weapon that can cause serious physical harm to others, has been physically cruel to people, has been physically cruel to animals, has stolen while confronting a victim, has forced someone into sexual activity); *destruction of property*

(has deliberately engaged in fire setting with the intention of causing serious damage, has deliberately destroyed others' property); *deceitfulness or theft* (has broken into someone else's house, building or car, often lies to obtain goods or favours or to avoid obligations, has stolen items of nontrivial value without confronting the victim); and *serious violations of rules* (often stays out at night despite parental prohibitions beginning before age 13 years, has run away from home overnight at least twice without returning for a lengthy period, is often truant from school beginning before age 13 years).

It has often been thought that childhood ODD is an important developmental risk factor for later CD in children with ADHD. Whittinger, Langley, Fowler, Thomas, and Thapar (2007) assessed 151 children with ADHD at ages 6-13 years and in adolescence 5 years later, finding that childhood ODD (diagnosis and severity) were almost three times more likely to develop CD in adolescence. However, other studies have differed in their findings that childhood ODD was not significantly associated with adolescent CD in children with ADHD at follow up (August et al., 1999; Biederman, Faraone, Milberger, Curtis, et al., 1996; Mannuzza, Klein, Abikoff, & Moulton, 2004). August et al. (1999) looked at the persistence and desistance of ODD in a community sample of children with ADHD using a 4-year follow-up design. A sample of disruptive children was compared to a sample of non-disruptive children. Few differences distinguished the groups at baseline, but of the 43 children with baseline diagnosis of ADHD and ODD, only 1 (2.3%) was found to have developed CD at follow-up.

ODD has not been found to be an essential precursor to CD in children with ADHD. Whittinger et al. (2007) found that there were some cases of adolescent CD in children who did not meet criteria for ODD at baseline, and some children who met diagnostic criteria for ODD but had stopped their antisocial behaviour at the time of the adolescent follow-up. Whittinger et al. (2007) also found childhood ADHD severity significantly predicted adolescent CD symptom scores, suggesting that ADHD may have independent risk effects on the development of CD.

Significant differences have been found between ODD and CD in children with ADHD in the domains of delinquency, overt aggression and ADHD symptom severity (Connor & Doerfler, 2008; Whittinger et al., 2007). Biederman, Petty, Dolan, et al. (2008) reported that the Child Behaviour Checklist (CBCL) results showing that ODD participants with CD could be distinguished from ODD without CD by their more severe

CBCL profile. CD participants were also reported to have significantly more difficulties on the CBCL Aggressive Behaviour scale compared to the ODD participants. This is consistent with the earlier work by Loeber, Green, Keenan, and Lahey (1995) reporting that physical fighting best predicted the onset of CD in children with ADHD, suggesting that ODD and CD are different disorders and should be studied separately. Comorbid ODD and ADHD will be discussed in more detail in Chapter 3.

#### *1.8.2.2 CD and ADHD*

Studies of children with ADHD consistently report a high co-occurrence with CD, some reporting as high as 50% in clinically referred samples (Stewart, Cummings, Singer, & deBlois, 1981) and 30% to 50% in community samples (Szatmari, Boyle, & Offord, 1989). Mannuzza et al. (2004) found that individuals with ADHD in childhood and no evidence of childhood disruptive behaviour did have a significantly greater rate of CD in adolescence than non-ADHD healthy control participants (43% versus 17%, $p<.001$). However, van Lier, van der Ende, Koot, and Verhulst (2007) reported that in children with ADHD approximately 4% of males and about 2% of females followed a deviant adolescent-peak CD trajectory, which accords with the point prevalence rates of CD in males and females (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003).

A significant amount of research has looked at males with ADHD in respect to CD. Some studies have shown gender-specific pathways in disruptive behaviour problems (van Lier et al., 2007). Costello et al. (2003) found a much stronger link between ADHD and CD among girls than among boys. Monuteaux, Faraone, Michelle Gross, and Biederman (2007) undertook a 5-year prospective longitudinal study of girls with (n=140) and girls without (n=122) ADHD. They found that ADHD was a significant risk factor for CD in girls. Also, that CD was associated with increased risk for academic, psychiatric, and sexual behaviour problems compared to ADHD girls without CD. van Lier et al. (2007) reported a significant sex effect showing males with ADHD and CD had consistently higher levels of conduct problems than females with ADHD and CD. Therefore, it appears that research findings are not consistent among males and females, suggesting that future research should examine gender associations separately.

Regardless of the rates of co-occurrence it is well documented that with the combined presence of ADHD and comorbid CD young people show more adverse

outcomes, for both males and females, than those with either disorder alone (Whittinger et al., 2007). Increased aggression and delinquency (Kuhne, Schachar, & Tannock, 1997; Monuteaux et al., 2007); greater academic under-achievement (Faraone, Biederman, Keenan, & Tsuang, 1991; Monuteaux et al., 2007) increased social maladjustment (Barkley, Fischer, Edelbrock, & Smallish, 1990); and lower self-esteem (Kuhne et al., 1997) have all been reported for comorbid ADHD and CD. Also, the severity of conduct problems in children with ADHD has been found to predict Antisocial Personality Disorder later in adulthood (Fischer, Barkley, Smallish, & Fletcher, 2002). Therefore, considering the poor prognosis of comorbid ADHD and CD it is important to continue systematic longitudinal research to better assess and treat these children and young people.

Recent research has examined the role of family conflict in the relationship between ADHD and CD. Sigfusdottir et al. (2017) found that the link between ADHD and CD was significantly stronger for those adolescents who had experience family conflict compared to those who had not experienced family conflict. These results suggest that family conflict moderates the association between ADHD and CD, indicating that family environment and ADHD symptoms are important when predicting CD among adolescent youth. Also, that family conflict exacerbates the effects of ADHD symptoms on CD among both females and males (Sigfusdottir et al., 2017).

#### *1.8.2.3 Summary*

ADHD and ODD and/or CD are the most common comorbidities in the ADHD population. Historically, ODD and CD have been studied together, grouped under a heading of disruptive behaviour disorders. It had been thought that childhood ODD was an important developmental risk factor for later CD in children with ADHD. However, not all studies have found this to be true. Significant differences have been found between ODD and CD in children with ADHD in the domains of delinquency, overt aggression, and ADHD symptom severity, indicating that they can be conceptualised as independent disorders and should be looked at separately. Some research has shown differences in gender-specific pathways, with girls with ADHD having an increased risk for developing CD and therefore, increased risks for academic, psychiatric, and sexual behaviour problems compared to girls with ADHD without CD. Regardless of the gender

differences, individuals with persistent ADHD and CD evince more severe ADHD symptoms and are at a higher risk of increased aggression and delinquency, greater academic under-achievement, increased social maladjustment, lower self-esteem and developing antisocial and criminal behaviour.

### 1.8.3 Depressive Disorders and ADHD

#### *1.8.3.1 Introduction*

Research has found that children and adolescents with neurodevelopmental disorders, such as ADHD, are more likely to suffer from Depression (for example, Major Depressive Disorder (MDD), Persistent Depressive Disorder (PDD) and Disruptive Mood Dysregulation Disorder (DMDD)) and Bipolar Disorders (for example, Bipolar-I and Cyclothymic Disorder). When these disorders co-occur more severe deficits and worse outcomes are found compared to those with one disorder alone. Co-occurring disorders can also impact on accurately assessing and diagnosing these young people.

The DSM-5 (APA, 2013) includes a range of disorders under the heading of Depressive Disorders, however the most common ones in childhood and adolescence are: DMDD; MDD; and PDD (Dysthymia – DSM-IV equivalent). MDD and PDD are the two primary Depressive Disorders associated with ADHD. MDD and PDD share features in common including the presence of sad, empty, and/or irritable mood, accompanied by somatic and cognitive changes that significantly affect the individual's capacity to function. They differ due to the duration and timing of depressive symptoms (APA, 2013).

#### *1.8.3.2 Major Depressive Disorder (MDD), Persistent Depressive Disorder (PDD) and ADHD*

MDD is diagnosed when five (or more) symptoms have been present during the same two week period and represents a change from previous functioning: depressed mood most of the day; markedly diminished interest or pleasure in activities; significant weight loss or weight gain; insomnia or hypersomnia; psychomotor agitation or retardation; fatigue or loss of energy; feelings of worthlessness; diminished ability to think or concentrate; and/or recurrent thoughts of death (at least one symptom must be either depressed mood or loss of interest or pleasure) (APA, 2013).

PDD (formally known as Dysthymia) is diagnosed when a person experiences symptoms of: depressed mood for most of the day, for more days than not for at least two years (one year for children and adolescents); and has two or more of the following: poor appetite or overeating; insomnia or hypersomnia; low energy or fatigue; low self-esteem; poor concentration or difficulty making decisions; and feelings of hopelessness (APA, 2013).

Typically, MDD and PDD are seen as part of the same spectrum of Depressive Disorders (APA, 2013) and most studies of ADHD comorbidity do not separate them. Children and adolescents with ADHD have been found to have a greater than chance rate of comorbid Depression (Angold et al., 1999; Biederman, Monuteaux, et al., 2008; Lundervold, Hinshaw, Sørensen, & Posserud, 2016). The point prevalence of Depressive Disorders (specifically MDD) in children and adolescents with ADHD ranges between 12% to 50% in community samples (Angold et al., 1999) and up to 60% in clinical samples (Brunsvold, Oepen, Federman, & Akins, 2008), which is 5.5 times higher than in young people without ADHD (Brunsvold et al., 2008). A study followed 123 children diagnosed with ADHD at age 4 to 6 years for up to 14 years (ages 18 to 20 years) and found children diagnosed with ADHD at ages 4 to 6 years were more likely to suffer from Depression as adolescents than those who did not have ADHD at that age (Chronis-Tuscano et al., 2010). They found that 18% of the children diagnosed early with ADHD suffered from Depression as adolescents, about 10 times the rate among those without ADHD. Girls with ADHD were also at higher risk for MDD than boys with ADHD (Chronis-Tuscano et al., 2010), which is consistent with the finding of Biederman, Monuteaux, et al. (2008) that girls are 2.5 times at higher risk.

Other factors can also increase the risk for young people with ADHD developing Depression: the relationship between ADHD and Depression appears to be stronger when it emerges in late childhood through to adolescence (Brunsvold et al., 2008); a positive family history of Depressive Disorders significantly increases the risk of these comorbidities and them continuing into adulthood (Meinzer, Pettit, & Viswesvaran, 2014); social problems (peer and family) (Drabick, Gadow, & Sprafkin, 2006); and parental psychopathology (Drabick et al., 2006).

When looking at the different presentations of ADHD, ADHD-I and Depressive Disorders have a significantly stronger association than that of ADHD-HI with

Depressive Disorders (Chronis-Tuscano et al., 2010). This relationship is also stronger in males than in females (Brunsvold et al., 2008). However, ADHD-C or ADHD-HI were at greater risk for suicidal thoughts (Chronis-Tuscano et al., 2010).

When children and adolescents have comorbid ADHD and MDD/PDD they tend to have more severe and enduring psychopathology than either disorder alone (Daviss, 2008). They tend to: have worse emotional regulation skills (Mayes, Waxmonsky, Calhoun, & Bixler, 2016); reduced levels of self-competence (Mayes et al., 2016); increased social impairments (Blackman, Ostrander, & Herman, 2005); and worse clinical outcomes (Biederman, Monuteaux, et al., 2008; Biederman, Wilens, Mick, Spencer, & Faraone, 1999; Brunsvold et al., 2008). To complicate matters, clinicians may mistake ADHD for Depression, making assessment and diagnosis challenging. Differentiating the conditions can be difficult because both disorders can involve mood problems with forgetfulness, an inability to focus, and lack of motivation.

#### *1.8.3.3 Disruptive Mood Dysregulation Disorder (DMDD) and ADHD*

DMDD is a new DSM-5 diagnosis. The core feature of DMDD is chronic, severe persistent irritability, with two prominent clinical manifestations: frequent outbursts; and severe irritability that is present between the severe temper outbursts that has been present for 12 months (APA, 2013). These temper outbursts must be inconsistent with the young person's developmental level and occur three or more times per week over a period of one year, in at least two settings. This diagnosis cannot coexist with ODD, Intermittent Explosive Disorder or Bipolar Disorder, however it can coexist with MDD, ADHD, CD and substance use disorders. The DSM-5 specifies that if individuals' symptoms meet criteria for both DMDD and ODD, they should only be given the DMDD diagnosis (APA, 2013).

This group of children show low frustration tolerance, high levels of outbursts and chronic, severe irritability that are linked to significant disruption in their social functioning (for example, family and peer relationships) and academic performance (APA, 2013). Children with ADHD and DMDD have been found to: exhibit lower level of global functioning (Mulraney et al., 2016); have higher rates of all affective disorders (76% verses 9.6%) (Ambrosini, Bennett, & Elia, 2013); poorer self-control and elevated bullying behaviour (Mulraney et al., 2016); worse social performance; (Mulraney et al.,

2016); and a higher level of negative outcomes (for example, dangerous behaviour, suicide ideation or suicide attempts, severe aggression and psychiatric hospitalization) (APA, 2013). Differences have been noted in the different ADHD presentations. The point prevalence rate of DMDD is about 2%-5% among children and adolescents, although males and school age children manifest a higher point prevalence rate of DMDD than females and adolescents (APA, 2013). Mayes et al. (2015) found 39% of children with ADHD-C have exhibited DMDD symptoms, compared to 12% of children with ADHD-I.

Previous studies have showed that children with ADHD and a high level of mood irritability were more likely to be diagnosed with ODD and Depression, than non-irritable children with ADHD (Ambrosini et al., 2013). With the new diagnosis of DMDD it is likely that children with ADHD and irritable mood might now be considered for a diagnosis of DMDD. Mulraney et al. (2016) reported that about 21.8% of children with ADHD meet criteria for DMDD, while 1.4% of children without ADHD meet these criteria.

It must be noted that the validity of DMDD as an independent diagnosis has been questioned. Mayes et al. (2015) investigated the stability of DMDD symptoms from childhood through to adolescence and found only one child with DMDD symptoms in their sample across 8 years who did not have symptoms of ODD, CD, ADHD, Anxiety and/or Depression. (Mulraney et al., 2016) also found that one in five children with ADHD in their second year of formal schooling meet criteria for DMDD and there was a very high overlap with ODD. This new category of Depressive Disorder needs more research given the current debate.

#### *1.8.3.4 Summary*

Children and adolescents with ADHD have a greater chance of developing Depressive Disorders (for example, MDD, PDD and DMDD). Other factors can also increase the risk for young people with ADHD developing Depressive Disorders: the developmental stage of the young person; a positive family history of Depressive Disorders; social problems; and parental psychopathology. The different presentations of ADHD and gender play a role in their association with Depression. ADHD and Depressive Disorders are associated with more functional impairments and worse

outcomes than each disorder alone. Further research in DMDD is required to help with the diagnostic clarification regarding DMDD and ODD.

### 1.8.4 Anxiety Disorders and ADHD

#### *1.8.4.1 Introduction*

Anxiety Disorders include disorders that share features of excessive fear and anxiety and related behavioural disturbances. Anxiety must be persistent for a diagnosis (for example, typically lasting 6 months or more, although less for children). There are several different Anxiety Disorders that differ from one another in the types of objects or situation that induce fear, anxiety, or avoidance behaviour, and the associated cognitive ideation (APA, 2013). Up to 12% of children between six to twelve years of age manifest features of one or more Anxiety Disorders (Costello, Egger, Copeland, Erkanli, & Angold, 2011). Many disorders persist in childhood, especially if not treated. Most occur more frequently in females than in males (approximately 2:1 ratio) (APA, 2013). The main types of Anxiety Disorders common in children and adolescents include: Separation Anxiety Disorder; Selective Mutism; Specific Phobia; Social Anxiety Disorder (Social Phobia); Panic Disorder; Agoraphobia; and Generalized Anxiety Disorder (GAD).

#### *1.8.4.2 Anxiety and ADHD*

ADHD and Anxiety are two common developmental mental health conditions, with a greater than chance association between them (Cox, Jr., 1982; Vance, 1997; Vance, Costin, & Maruff, 2002). Epidemiological and clinical studies suggest 25% to 33% of children with ADHD have co-existing clinically significant Anxiety (Jensen et al., 2001).

When these disorders co-exist, the associated functional impairments are more severe (Hoza, Murray-Close, Arnold, Hinshaw, & Hechtman, 2010; Tannock, 2009; Vance, 2011). Sciberras, Lycett, et al. (2014) reported that children with ADHD and Anxiety may experience a poorer quality of life with more behavioural and functional difficulties compared to those children with ADHD alone. Studies show that Anxiety adversely affects verbal working memory when comorbid with another mental health condition, such as ADHD (Bedard & Tannock, 2008). The performance of children with ADHD and Anxiety remains significantly impaired on cognitively complex tasks and high demand Working Memory tasks (Bloemsma et al., 2013; Pliszka, 1989; Sciberras,

Lycett, et al., 2014; Vance, Ferrin, Winther, & Gomez, 2013). Language impairments have been noted to be more severe in young people with ADHD and a comorbid Anxiety Disorder (Sciberras, Mueller, et al., 2014). Children with ADHD and Anxiety have been found to have significantly more emotional regulation difficulties compared to children with ADHD alone (Seymour et al., 2012) and they can experience a high level of sensitivity to negative emotions compared to typically developing children (McKay & Storch, 2011).

Some research has looked at the different ADHD presentations and Anxiety. Children with ADHD-I were found to present higher rates of baseline persistent Anxiety compared to those with ADHD-HI and ADHD-C (Jensen et al., 2001; Tannock, 2009). Other studies have also linked inattentive symptoms to higher rates of internalising problems, including Anxiety (Graetz et al., 2001). Young people with ADHD-C manifested higher trait Anxiety, while those with ADHD-I demonstrated more state Anxiety (Levy, Hay, Bennett, & McStephen, 2005).

It has also been noted that the clinical characteristics of ADHD and Anxiety may differ in the presence of a further comorbid condition such as Depression and/or ODD, CD, with ODD or CD more likely when Anxiety co-occurs with ADHD (Bilgiç et al., 2013; Tannock, 2009). Further research in this area is needed.

#### *1.8.4.3 Summary*

ADHD and Anxiety disorders are common mental health disorders for children and adolescents. There is a greater than chance association that they will occur together. When co-occurring, children and adolescents experience a poorer quality of life with more behavioural and functional difficulties compared to those children with ADHD alone. Therefore, effective treatment of both conditions is essential. More research is needed regarding ADHD and Anxiety when in the presence of a further comorbid condition such as ODD.

### 1.8.5 Autism Spectrum Disorder (ASD) and ADHD

#### *1.8.5.1 Introduction*

ASD is a neurodevelopmental disorder often comorbid with other neurodevelopmental disorders like ADHD. The DSM-5 sets out two main criteria for a diagnosis of ASD: *persistent deficits in social communication and social interaction across multiple contexts* (deficits in social-emotional reciprocity; deficits in nonverbal communicative behaviours used for social interaction; and deficits in developing, maintaining, and understanding relationships); *and restricted, repetitive patterns of behaviour, interests, or activities* (stereotyped or repetitive motor movements, use of objects, or speech; insistence on sameness, inflexible adherence to routines, or ritualized patterns of verbal or nonverbal behaviour; highly restricted, fixated interests that are abnormal in intensity or focus; and hypo or hyperreactivity to sensory input or unusual interest in sensory aspects of the environment) (APA, 2013). The current version of the DSM has no exclusion criteria for children and adolescents with ASD being comorbid with ADHD. Young people with ASD and ADHD can manifest more severe ASD and ADHD symptoms compared to ASD and ADHD alone (APA, 2013).

The point prevalence rate of ASD is about 1% of the general population and approximately 70% of this population of children and adolescents exhibit symptoms of another mental health disorder, such as Anxiety and/or Mood Disorders, Obsessive Compulsive Disorder and ADHD (APA, 2013; Brereton, Tonge, & Einfeld, 2006).

#### *1.8.5.2 ASD and ADHD in children and adolescents*

Estimates of co-occurring ADHD and ASD vary widely, as it is commonly thought that many of individuals with Autism also have ADHD symptoms. Studies looking at young people with ASD with clinically elevated levels of ADHD range between 14% - 85% (Panagiotidi, Overton, & Stafford, 2019; Stevens, Peng, & Barnard-Brak, 2016). Similarly, clinically elevated levels of autism symptoms have been reported in 18% to 67% of children with ADHD (Factor, Ryan, Farley, Ollendick, & Scarpa, 2017; Green, Sciberras, Anderson, Efron, & Rinehart, 2016; May, Sciberras, Hiscock, & Rinehart, 2016). Simonoff et al. (2008) found that 28.2% of children in a population derived sample had comorbid ADHD and ASD. It is unclear about the influence of gender on the point prevalence rate and clinical characteristics of ADHD and ASD. Study

findings have varied from “no differences” to “significant differences” between females and males (Ronald, Larsson, Anckarsäter, & Lichtenstein, 2014).

Individuals with co-occurring ASD and ADHD face more challenges than those with one condition only. When both conditions occur together the presence of each condition amplifies the symptoms of the other condition (Mannion & Leader, 2014; Matson, Rieske, & Williams, 2013). Research indicates that individuals with co-occurring ASD and ADHD face more challenges than those with one condition only: it increases the likelihood of disruptive behaviour (Sikora, Vora, Coury, & Rosenberg, 2012; Zablotsky, Bramlett, & Blumberg, 2020); more difficulties in reading other’s emotions and feelings, in holding and recalling and using information (Colombi & Ghaziuddin, 2017; Factor et al., 2017); a higher number of inattentive and hyperactive/impulsive symptoms and more stereotypic and repetitive behaviours (Green et al., 2016; Rao & Landa, 2014; Zablotsky et al., 2020); greater deficits in inhibitory control, attention and Working Memory (Berenguer-Forner, Miranda-Casas, Pastor-Cerezuela, & Roselló-Miranda, 2015); and increases the risk for further externalising and internalising “comorbidities” (Ronald et al., 2014; Tureck, Matson, May, & Turygin, 2013).

It is only in the current version of the DSM (APA, 2013) that ADHD is able to be diagnosed in the presence of ASD, due to the symptoms of both disorders overlapping significantly so research in this area is only in its infancy. Researchers have debated that it might be more helpful to evaluate their “symptoms co-occurrence, rather than complete comorbidity” (Ronald et al., 2014). Further longitudinal research in this area is needed to fully understand the comorbidity of ASD and ADHD.

#### *1.8.5.3 Summary*

ASD is a neurodevelopmental disorder characterised by persistent impairments in social functioning and restricted and repetitive patterns of behaviour. Historically, research examined ASD and ADHD separately, as being diagnosed with ASD excluded the diagnosis of ADHD. It has only been since the release of the 5$^{th}$ edition of the DSM that it is possible to have a diagnosis of co-occurring ADHD and ASD. Individuals with co-occurring ASD and ADHD have been found to face more challenges than those with one condition only: it increases the likelihood of disruptive behaviour; more challenges

in reading other's emotions and feelings; in holding and recalling and using information; a higher number of inattentive and hyperactive/impulsive symptoms and more stereotypic and repetitive behaviours; and increases the risk for further externalising and internalising comorbidities. Understanding the implications of co-occurring ADHD and ASD is in its early stages and further longitudinal systematic research is required.

## 1.9 NATURAL HISTORY OF ADHD

### 1.9.1 Introduction

Many studies note that ADHD core symptoms differ across developmental stages: hyperactivity and impulsiveness are more prevalent in childhood; while inattention is more common across childhood, adolescence and into adulthood (Cherkasova et al., 2013; Colomer-Diago, Miranda-Casas, Herdoiza-Arroyo, & Presentación-Herrero, 2012; Faraone et al., 2015). Longitudinal studies report that inattention is the most common ADHD symptom across the developmental stages.

### 1.9.2 Natural History of ADHD

ADHD is often identified and diagnosed during primary school years as the young person's inattention becomes more impairing once they start school. However, many parents generally first observe excessive motor activity when the child is a toddler, but the symptoms are often hard to distinguish from highly variable normative behaviours before the age of 4 years (APA, 2013). During childhood excessive motor activity and inattention are the most common symptoms (APA, 2013; Dulcan & Lake, 2012). In most individuals with ADHD symptoms of hyperactivity become less obvious or subside in adolescence and adulthood, but difficulties of restlessness, inattention, poor planning and impulsivity persist (APA, 2013; Cherkasova et al., 2013; Colomer-Diago et al., 2012; Faraone et al., 2015). During adolescence and adulthood, ADHD is often associated with antisocial behaviours, inattention, poor planning, difficulties in academic, occupational and relationship functioning, gambling and substance abuse (cigarette, alcohol and marijuana abuse) (Bruneau et al., 2016; Faraone et al., 2015; Massetti et al., 2008; Wilens & Spencer, 2010). A large proportion of children with ADHD remain relatively impaired

into adulthood (APA, 1013). Ramsay and Rostain (2014) and Weiss, Hechtman, Milroy, and Perlman (1985) reported that Antisocial Personality Disorder is the most common diagnosis in adults with a history of hyperactivity in childhood.

Individuals with ADHD-C typically manifest more severe ADHD core symptoms and these symptoms are relatively more stable across developmental stages compared to ADHD-HI and ADHD-I presentations (Lacramioara & Eugene, 2007; Meyer & Sagvolden, 2006; Wilens & Spencer, 2010). Young people with ADHD-C were found to have significant impairments in mathematics and more internalising (anxiety and depression) symptoms (Massetti et al., 2008). There is also a high chance that children with ADHD-HI will be diagnosed with ADHD-C by adolescence (Riley et al., 2008). Children with ADHD-I were found to have more learning difficulties and lower reading, spelling and mathematic scores over an 8-year period than children with ADHD-HI and ADHD-C (Massetti et al., 2008).

ADHD symptom persistence increases when there is psychiatric comorbidity. Biederman et al. (2011) conducted a follow-up study of boys with and without ADHD, they reported 78% of children with ADHD continued to have a full or partial persistence and impaired functioning. They found it was more predicable when psychosocial adversity and psychiatric comorbidity were present. Children with ADHD and co-occurring comorbidities were more likely to have a poorer quality of life and greater peer and family problems compared to ADHD alone (Armstrong, Lycett, Hiscock, Care, & Sciberras, 2015).

### 1.9.3 Summary

An ADHD diagnosis is relatively stable over time, although the core symptoms can differ across developmental stages: hyperactivity and impulsivity more prominent in childhood; while inattention tends to last through adolescence into adulthood, with ADHD-I the most common ADHD presentation across the developmental stages. Children with ADHD-I are more likely to have learning difficulties than children with ADHD-HI and ADHD-C. Children with ADHD-C manifest more severe ADHD core symptoms and these symptoms are relatively more stable across developmental stages, with more impairments in mathematics and more internalising symptoms. In later developmental stages, ADHD has a greater than chance association with problems in

academic, occupational and relationship functioning, and comorbid disorders. More longitudinal research is needed, especially in adulthood ADHD which is a relatively new area of study.

## 1.10 KEY TREATMENT APPROACHES FOR ADHD

### 1.10.1 Introduction

There are three main treatment approaches for ADHD: pharmacological; non-pharmacological; and combined interventions. The locally approved medications for ADHD are stimulant (for example, dexamphetamine and methylphenidate) and non-stimulant (atomoxetine) medications. There are also a range of third line medications that are used (clonidine, guanfacine, and risperidone). The non-pharmacological approaches include behavioural/psychosocial treatments (for example, family and school-based education, social skills training, Cognitive Behavioural Therapy (CBT), dietary management of ADHD, and neurocognitive interventions (for example, Neurofeedback interventions and working memory training). Pharmacological interventions are the first-line treatment for individuals with moderate to severe ADHD. However, recent review studies suggest that combined pharmacological and non-pharmacological interventions are the best to facilitate persistent improvements in ADHD symptoms and associated functional impairments.

### 1.10.2 Pharmacological Treatment for ADHD

Both stimulant (for example, methylphenidate and dexamphetamine) and non-stimulant medications (atomoxetine) are used to treat ADHD. They have been found to significantly decrease the severity of ADHD core symptoms (Faraone et al., 2015; Vance, 1997; Wolraich & DuPaul, 2010).

#### *1.10.2.1 Stimulant Medication or ADHD*

Stimulant medication is the first-line medication treatment for individuals with moderate to severe ADHD (Bello, 2015; Connor, 2015; Vance, 1997). Connor (2015) reported that stimulant medication modulates the action of dopamine by blocking the pre-

synaptic dopamine transporter, while dexamphetamine also promotes releasing of pre-synaptic dopamine. These medications aid in the regulation of executive functions subserved by neural circuits, often impaired in the ADHD population.

Stimulant medications have been shown to be effective in treating ADHD symptoms, such as short attention span, impulsive behaviour, and hyperactivity. The medication improves ADHD symptoms in about 70 percent of adults and 70-80 percent of children (Advokat & Scheithauer, 2013). When short-term clinical response is assessed quantitatively via rating scales, the effect size of stimulant treatment relative to placebo is robust, averaging about 1.0, one of the largest effects for any psychotropic medication (Faraone, Biederman, Spencer, & Aleardi, 2006; Pliszka, 2007).

Studies have reported a number of side effects of stimulant medication. Short-term physiological adverse-effects can include insomnia, loss of appetite, stomach-aches, headaches, dizziness and daytime drowsiness (Bello, 2015; Faraone et al., 2015). Stimulant medication use has been associated with an increase in effective symptoms (for example, mood lability, dysphoria, sudden severe sadness, social withdrawal and aggressive outbursts) (Vance, 1997). Minor delays in growth in height and weight has also been reported when using stimulant medication (Connor, 2015; Maiese, 2012). For children with a pre-existing cardiac condition stimulant medication should be used cautiously or with a cardiac second clinical opinion (Faraone et al., 2015; Wolraich & DuPaul, 2010). It is also advised to monitor blood pressure and heart rate for children with ADHD who have stimulant medication treatment (Faraone et al., 2015). Faraone et al. (2015) concluded that the benefits to stimulant medication use generally outweighs the adverse effects associated with using stimulant medication and they are generally "manageable".

For individuals who have persistent ADHD pharmacological treatments are typically used long term (Faraone et al., 2015; Vance, 1997).

#### *1.10.2.2 Non-Stimulant Medication for ADHD*

Atomoxetine is the current approved non-stimulant medication for treatment of ADHD. Bello (2015) and Faraone et al. (2015) describes it as a selective pre-synaptic norepinephrine transporter inhibitor that increases the synaptic levels of norepinephrine in brain regions with low dopamine transporter expression. Atomoxetine has similar

adverse effects to stimulant medication along with nausea, dry mouth and potential elevation in blood pressure and heart rate (Bello, 2015). It is generally commenced at a low dosage level with subsequent regular adjustments in order to optimally reduce the adverse effects (Faraone et al., 2015).

Current studies of atomoxetine lack uniformity in how medication effectiveness is assessed and vary in their study design parameters making it difficult to compare medication effect sizes from different studies (Faraone, Biederman, & Mick, 2006). Some studies suggest that it might be effective for the treatment of ADHD and comorbid Depressive/Anxiety Disorders (Bangs et al., 2007; Kratochvil et al., 2005). However, when compared to stimulant medication use for young people with ADHD, atomoxetine was found to be less efficacious (Faraone et al., 2015; Wolraich et al., 2011).

#### *1.10.2.3 Third Line Medications for ADHD*

Generally other third line medications are used in the treatment of ADHD when the first- and second-line treatments have not been found helpful in symptom reduction, or the individual experiences significant adverse effects. Several controlled trials have demonstrated that guanfacine, clonidine and risperidone in low doses (for example, 0.5 to 1.5mg) can significantly reduce ADHD hyperactive and impulsive symptoms, particularly in young people with intellectual disabilities and/or ASD (Faraone et al., 2015; Hazell, 2007).

Some studies have looked at the combined use of stimulant and non-stimulant medications or other third line medications for ADHD treatment where there has been limited response to one medication (Faraone et al., 2015). Although Faraone et al. (2015) warn that there have been very limited studies looking at the combined effects on cardiovascular safety.

#### *1.10.2.4 Medication Acceptance and Adherence for ADHD*

Several factors are associated with use of medication for ADHD: parent or family characteristics; child characteristics; practitioner or health system factors; and medication-related factors (Charach & Fernandez, 2013). Parent beliefs about ADHD and attitudes toward treatment are significant determinants of medication use (dosReis et

al., 2009). Some families understand that ADHD is a neurobiological condition and accept that medication is indicated, whereas for others, such treatment is not acceptable (Charach & Fernandez, 2013; dosReis & Myers, 2008). Many parents prefer implementing behavioural strategies and other non-medication strategies (Bussing & Gary, 2001; Johnston, Hommersen, & Seipp, 2008), which will be discussed further in this chapter. The relationship between parents and the healthcare provider, and the cost of medication can also impact on the decision to accept and start treatment, or to continue using it (Coletti et al., 2012; dosReis & Myers, 2008).

Certain child and adolescent characteristics have been found to impact on the use of medication: the child's age when starting; the severity of the ADHD symptoms; additional learning and behaviour difficulties'; and past experiences that medication was effective with few adverse effects (Bussing et al., 2012; Charach & Gajaria, 2008; Coletti et al., 2012). Also, how their peers perceive them using medication is important: some children recognize the benefits of medication in improving peer relationships (Singh et al., 2010); while others may avoid treatment because of stigma associated with taking medication (Bussing et al., 2012; Singh et al., 2010).

ADHD is conceptualized as a chronic disorder requiring treatment throughout childhood and adolescence (2000). In the United States the most common age to begin medication is between five and nine years, more among boys than girls (Bloom, Jones, & Freeman, 2013), with one quarter to one third of children receiving a single prescription (Williams & Taylor, 2006). Medication treatment is often short-term or intermittent, with one half to two thirds discontinuing within one year (Williams & Taylor, 2006). Studies have documented that medication nonadherence is common in children and adolescents with ADHD (Adler & Nierenberg, 2010), with teenagers less likely to continue taking psychostimulants relative to younger children. (Thiruchelvam, Charach, & Schachar, 2001) found of 71 children fifty-two percent of children adhered to stimulant treatment over the three year period they were measured. Adler and Nierenberg (2010) reviewed the literature on the prevalence, potential causes, and consequences of medication nonadherence in ADHD. They found that although medications alleviate many aspects of the disorder, associated difficulties with disorganization and planning can lead patients to have poor adherence and subsequent treatment failure. They found that treatment studies have shown that the prevalence of medication discontinuation or nonadherence is between 13.2% to 64%. They found medication nonadherence was more prevalent in

immediate-release versus extended-release psychostimulants in childhood/adolescent ADHD. Toomey, Sox, Rusinak, and Finkelstein (2012) found that the main reasons for discontinuation were "psychological side effects" (mood changes, irritability, Depression and personality changes) and "perceived inadequate effectiveness" over the longer term.

#### *1.10.2.5 Medication and Secondary Symptoms of ADHD*

While effectively treating the core symptoms of ADHD, the extent to which medication helps with improving associated impairments (such as academic performance and social performance) is still unclear. This may be due to the impact of other risk factors influencing these impairments (Faraone et al., 2015; Powers, Marks, Miller, Newcorn, & Halperin, 2008). Therefore, it is important to be cautious about using medication alone when the young person has several other risk factors present: for instance comorbid disorders; psychosocial stressors (such as, financial and medical issues); and an unstable home environment (parents with an intellectual disability and/or alcohol/substance abuse and/or mental health issues) (Faraone et al., 2015). Smith and Shapiro (2015) note that medication may assist learning to occur in the context of behavioural support and skills training, making combined interventions more effective.

### 1.10.3 Non-Pharmacological Treatments for ADHD

#### *1.10.3.1 Introduction*

Non-pharmacological treatments include a range of dietary (restricted elimination diets, artificial food colour exclusions, and free fatty acid supplementation) and psychological (cognitive training, behavioural interventions, neurofeedback and working memory training) (Faraone et al., 2015). The most recent systematic review and meta-analyses of randomized controlled trials of dietary and psychological treatments found little evidence to support the use of these treatments for the core symptoms of ADHD (Sonuga-Barke et al., 2013). However, this is not consistent with previous findings from meta-analyses of behavioural treatments for ADHD. The authors reported that better evidence for the efficacy from blinded assessments is required for behavioural interventions, neurofeedback, cognitive training, and restricted elimination diets before they can be supported as treatments for core ADHD symptoms.

Although not all research has found non-pharmacological treatments to be effective for the core symptoms of ADHD (inattention, hyperactivity and impulsivity), they can be helpful for the secondary symptoms of ADHD such as social, learning and behavioural difficulties, which can be crucial for the long-term prognosis of young people with this condition and can persist even with pharmacological treatment (Winther, 2015). They generally complement rather than replace medication for the ADHD population (Antshel et al., 2011; Faraone et al., 2015) and with good integration of pharmacological and non-pharmacological treatment approaches treatment adherence can be improved (Faraone et al., 2015).

#### *1.10.3.2 Dietary Interventions for ADHD*

Dietary interventions used for treatment of ADHD was first proposed by Feingold (1975) who hypothesised that a group of children may be more sensitive to certain dietary substances, including "artificial food colour, flavours, preservatives, dietary salicylates and potentially natural food protein", which lead them to have certain reactions that may contribute to ADHD symptoms, especially hyperactivity (Hurt & Arnold, 2014).

The two main dietary approaches are elimination diets and natural supplements (for example, fatty acids, amino acids, vitamin supplements, and mineral supplements - iron, magnesium, and zinc). Currently there is no evidence to support the effectiveness of these methods, apart from free fatty acid supplementation, for treating ADHD and more systematic evidence from "well-blinded studies" is needed (Sonuga-Barke et al., 2013). Free fatty acid supplementation produced small but significant reductions in ADHD symptoms even with properly blinded assessments, although the clinical significance of these effects remains to be determined (Sonuga-Barke et al., 2013).

#### *1.10.3.3 Behavioural Therapies for ADHD*

There is currently controversy regarding the effectiveness of behaviour modification for children with ADHD despite years of study and multiple investigations reporting the beneficial effects of the intervention. Behaviour modification is grounded in learning theory and includes principles of classical conditioning, operant conditioning, cognitive-behavioural theory, and social learning theory (Fabiano et al., 2009). Parent training programmes are psychosocial interventions aimed at training parents in

techniques to enable them to manage their children's challenging behaviour (Zwi, Jones, Thorgaard, York, & Dennis, 2011). Many approaches focus on operant procedures where antecedents and consequences of a child's behaviours are manipulated to increase desired behaviour and decrease undesirable behaviour. Parent management training includes: positive/negative reinforcement techniques; effective instruction giving; ensuring clear boundaries and setting house rules. These principles have been used for over forty years in treating childhood externalizing problems (Patterson, 1974). Since the 1970's, behaviour modification programs have been used with children diagnosed with ADHD (O'Leary & Pelham, 1978; Pelham, 1977).

Several systematic reviews have looked at the behavioural treatment literature for ADHD. Two reviews focused on disruptive classroom behaviour, both indicating that behavioural interventions for ADHD in the classroom were effective (DuPaul & Eckert, 1997; Stage & Quiroz, 1997). Other reviews have focused on the entire behavioural treatment literature for ADHD, both at home and school, finding that behaviour parent training and classroom contingency management met criteria for empirically supported treatments (Daly, Creed, Xanthopoulos, & Brown, 2007; Fabiano et al., 2009; Pelham, Jr. & Fabiano, 2008; Pelham, Jr., Wheeler, & Chronis, 1998).

Zwi et al. (2011) undertook a systematic review of the literature to determine whether parent training interventions were effective in reducing ADHD symptoms and associated problems in children aged between five and eighteen years with a diagnosis of ADHD, compared to typically developing young people with no parent training intervention. They concluded that parent training "may" have a positive effect on the behaviour of children with ADHD and may also reduce parental stress and enhance parental confidence. However, the poor methodological quality of the included studies increased the risk of bias in the results. The data concerning ADHD-specific symptoms were ambiguous and data were lacking for other important outcomes, such as school achievement and adverse effects (Zwi et al., 2011). Some studies have shown parent-based therapies for preschool children with ADHD have improved ADHD symptoms (Sonuga-Barke et al., 2018) and their mothers' sense of well-being (Sonuga-Barke, Daley, Thompson, Laver-Bradbury, & Weeks, 2001), suggesting treatment outcomes may differ depending on the child's age. Sonuga-Barke et al. (2018) compared the New Forest Parenting Program to Incredible Years Toddler programme finding both programs were effective for preschool ADHD. Also improving the quality of parenting has been

shown to have long term positive and protective effects on children with ADHD and can reduce the negative functional outcomes of ADHD symptoms (Faraone et al., 2015). Behaviour Parent Training has been shown to improve engagement with treatment, although the long-term outcomes remain unclear (Chacko, Kofler, & Jarrett, 2014). ADHD psychoeducation for parents and youth regarding the causes of ADHD; its associated morbidity; the potential for a compromised course; the rational for treatments; and plans for key life transitions has been found useful (Faraone et al., 2015; Nussey, Pistrang, & Murphy, 2013).

The most recent review in 2013, completed by (Sonuga-Barke et al.) found nonsignificant results for behavioural interventions for reduction of ADHD core symptoms, reporting that better evidence for efficacy from blinded assessments is required for behavioural interventions before they could support them as treatments for core ADHD symptoms.

In contrast, Fabiano et al. (2009) suggested that enough research demonstrates that behavioural interventions are viable and effective intervention for ADHD and suggest that efforts should be redirected from debating the effectiveness of the intervention to disseminating, enhancing, and improving the use of behavioural interventions in community, school and mental health settings. They suggest that using parent ratings of ADHD symptoms to measure ADHD treatment effectiveness is not as helpful as measures of parenting skill and stress, peer relationships, and academic functioning in school, which are validated measures predictive of long-term functioning (Pelham, Jr., Fabiano, & Massetti, 2005). Although, further evidence is warranted for behavioural intervention to be supported as a front-line treatment for core ADHD symptoms, there is evidence that these interventions benefit parenting practice and improve behavioural problems which commonly co-occur with ADHD and are often the main reason for referral (Daley et al., 2018). Positive results have also been found in relation to parental knowledge and children's emotional, social and academic functioning (Daley et al., 2018). Further research should focus on randomized control trials involving high-quality training, supervision of therapists and their practice with the child, as well as which young people with ADHD benefit the most from these behavioural interventions (Kapil Sayal et al., 2018).

#### 1.10.3.3a Other Behaviour Interventions for ADHD

Other interventions, seen in the literature, tend to be more focused on specific skill impairments for the young person at home and school, such as social skills, organisational skills, self-instructional skills, problem solving skills and behavioural interventions to improve organisation, time management and planning difficulties. Social skills training has been implemented as an additional treatment option for ADHD to help improve prosocial and socially skilled interactions. However, evidence of its effectiveness is limited (Amori Yee Mikami, 2015). Executive function training (for example, Facilitative Intervention Training and Mindfulness) have also been reported as useful (Rapport, Orban, Kofler, & Friedman, 2013). With more adults being recognised as having ADHD now, life management skills coaching has been identified as an important area to evaluate its effectiveness for people with ADHD.

Young, Bramham, Gray, and Rose (2008) found that psychological treatment, which started at the point of diagnosis, significantly facilitated adaptive coping with ADHD symptoms. They found that individuals learnt skills to anticipate future challenges and apply appropriate coping strategies that assisted with change to their self-perception and improved their view of the future. Although these interventions have been shown to be evidence based for other disorders, further systemic research of their effectiveness for ADHD is required, specifically examining what measures should be used to evaluate treatment effectiveness.

### *1.10.3.4 Neurocognitive Therapies for ADHD*

#### 1.10.3.4a Neurofeedback Interventions for ADHD

Neurofeedback interventions (a positive reinforcement, reward-based technique) has been used in the treatment of ADHD since 1976 (Arns, de Ridder, Strehl, Breteler, & Coenen, 2009). Neurofeedback is a learning process in which the brain is rewarded for positive changes in its activity (Fox, Tharp, & Fox, 2005). The response to this learning process is visual or auditory feedback. Neurofeedback is designed to change certain types of EEG activity (Barkley, 1997; Lubar, 2003). The goal of the treatment is to enhance beta and depress theta brain activity (Othmer, 2005). Neurofeedback interventions are typically conducted three times a week with 30 treatments for each child and adolescent (Duric, Assmus, Gundersen, & Elgen, 2012). Each treatment lasting 40 minutes where

the child receives feedback regarding their own brain activity, via unipolar placed sensors on the scalp measuring brain activity and a computer processing the signals as brainwave frequencies. The information is presented to the child in the form of a video game or film. When they play the game or watch the films, they produce brainwave activity that is "shaped" toward more regulated performance (Duric et al., 2012).

Some clinical trials have found that neurofeedback effectively treats the symptoms of ADHD (Duric et al., 2012; Fox et al., 2005; Monastra, 2004): improving attention; decreasing hyperactivity; increasing academic outcomes; and improving social skills (Arns et al., 2009; Monastra et al., 2005). Other studies have only found improvements in attention (Bakhshayesh, Hänsch, Wyschkon, Rezai, & Esser, 2011), and two studies with large sample sizes did not find significant improvements in core ADHD symptoms at all (Monastra, Monastra, & George, 2002; Rossiter, 2004). Duric et al. (2012) compared neurofeedback intervention to stimulant medication treatment and did not find a significant difference between the treatments in the improvement of ADHD core symptoms: they found it to improve attention and hyperactivity symptoms. These authors suggested it could be a useful treatment option for children with ADHD whose parents favoured a non-pharmacological treatment.

Studies measuring the effectiveness of neurofeedback interventions have been limited by potential confounding factors such as small studies, lack of randomization and a lack of adequate typically developing control groups (Duric et al., 2012). Many controlled studies have used either stimulant medication or waiting list groups as the control group (Rossiter, 2004). Several non-randomized studies found large effect sizes for attention and medium effect size for hyperactivity (Kropotov et al., 2005). Clinically it is a time consuming (three times a week, for 30 to 40 sessions with a psychologist) and costly intervention that may limit its accessibility for many families. In terms of a treatment option for ADHD, further systemic research is recommended.

#### 1.10.3.4b Working Memory training for ADHD

Working Memory training has been growing as a potential treatment for ADHD symptoms. Cogmed Working Memory Training (RoboMemo, Cogmed Cognitive Medical Systems AB, Stockholm, Sweden) is a tool that has been recommended for improving cognitive abilities, such as attention and reasoning, and improved response

inhibition resulting in a reduction of inattentive symptoms of ADHD (Klingberg et al., 2005). Cogmed Working Memory Training typically consists of performing working memory tasks implemented by a game like computer program. The program includes visuospatial working memory tasks (remembering the position of objects in a 4 x 4 grid) as well as verbal tasks (remembering phonemes, letters, or digits). Responses are made by clicking on displays with the computer mouse. Children undertake training for one hour a day, five days per week for five weeks. In addition, several components of the program focus on supporting the child's engagement by using contingent reinforcement (earning small rewards for successful completion of a training week), supervision by a training aide (typically a parent or guardian and a certified coach who tracks performance) who reinforces on-task behaviour, effort and completion by providing praise and encouragement.

Several randomized trials of Cogmed Working Memory Training have been conducted in children and adolescents with ADHD (Beck, Hanson, Puffenberger, Benninger, & Benninger, 2010; Chacko, Bedard, et al., 2014; Green et al., 2012; Klingberg et al., 2005). Klingberg et al. (2005) demonstrated the effects of Cogmed Working Memory Training on various aspects of trained (for example, outcomes resembling tasks similar to those used in Cogmed Working Memory Training) and non-trained cognitive tasks (for example, Stroop Task). The results demonstrated improvements in parent-rated inattention and hyperactivity/impulsivity at post-treatment, however no statistical effects were found on teacher-rated symptoms of inattention or hyperactivity/impulsivity, or on motor activity (Klingberg et al., 2005). Beck et al. (2010) reported improved parent-rated Working Memory and diminished inattention symptoms/problems at post-treatment, with no statistically significant effect observed on parent-rated ADHD hyperactivity/impulsivity symptoms or on any teacher-rated outcomes (Beck et al., 2010). Green et al. (2012) did not find any improvement in parent-rated ADHD symptoms (Green et al., 2012). Chacko, Bedard, et al. (2014) found improvements in verbal and nonverbal working memory storage, but no improvements in working memory storage plus processing/manipulation or any other outcome measures for ADHD symptoms (Chacko, Bedard, et al., 2014). Overall, the results suggest consistent effects of Cogmed Working Memory Training on trained Working Memory outcomes, but inconsistent findings for other cognitive outcomes and parent/teacher-rated ADHD symptom outcomes (for example, setting specific behavioural improvements).

Researchers have questioned the effectiveness of Cogmed Working Memory Training due to methodological implications (Chacko, Bedard, et al., 2014; Chacko et al., 2013; Hulme & Melby-Lervåg, 2012; Shipstead, Hicks, & Engle, 2012). Many researchers have noted a lack of equivalence between the active and placebo versions of Cogmed Working Memory Training (Green et al., 2012; Klingberg et al., 2005). Differences noted have been: the placebo condition that required considerably less time (and effort) to complete than the Cogmed Working Memory Training Active condition; the placebo condition had significantly less amount of parent interactions with the child than the Cogmed Working Memory Training Active condition; and other variables of the training maybe contributing to the success of the training, for example, collaborative problem-solving with parents by the coach and contingent reinforcement. Methodologically rigorous studies are needed to more appropriately evaluate Cognitive Working Memory Training as a treatment for ADHD in children (Chacko, Bedard, et al., 2014).

In conclusion, when more rigorous comparison conditions are used Cogmed Working Memory Training demonstrates effects on certain aspects of Working Memory in children with ADHD; however, it does not foster treatment generalization to other domains of functioning and should not be considered a viable treatment for children with ADHD (Chacko, Bedard, et al., 2014; Shipstead et al., 2012).

### 1.10.4 Combined Treatments for ADHD

Combined treatment approaches refer to the use of both pharmacological and non-pharmacological interventions for ADHD core symptoms and secondary symptoms. One of the most influential studies, the Multimodal Treatment of Attention Deficit Hyperactivity Disorder (MTA) study was a multisite study designed to evaluate treatments for ADHD, including behaviour therapy, medications, and the combination of the two. A group of 579 children with ADHD-C, aged 7-9 years, were randomly assigned to 14 months of controlled: medication management; intensive behavioural treatment (parent, school, and child components); the two combined; or standard community care (treatment by community providers). All four groups showed sizable reductions in symptoms over time, with children in the combined treatment and medication management groups showing significantly greater improvement than those given

intensive behavioural treatment or community care. Combined treatment proved superior where the children also displayed oppositional/aggressive symptoms and internalizing symptoms, and improved teacher rated social skills, parent-child relations and reading achievement.

It has been suggested that when medication treatment is combined with behavioural therapy a lower dosage of medication may be required for individuals with ADHD (Vance, 1997), and similarly, the behavioural intervention may be less intense when medication is simultaneously delivered (Pfiffner et al., 2014). However, more research is required to determine the effectiveness of combined pharmacological and non-pharmacological treatments for ADHD.

### 1.10.5 Summary

There are currently three main approaches for ADHD treatment: pharmacological, non-pharmacological and combined treatments. Stimulant medication (for example, dexamphetamine and methylphenidate) is the proven first line medication treatment for individuals with ADHD. Non-stimulant medication (for example, atomoxetine) and other third line medications (for example, guanfacine, clonidine and risperidone) can be effective if stimulant medication is not effectively managing the ADHD symptoms. Pharmacological interventions are not always acceptable to parents and young people and are not always associated with functional improvements (for example, social difficulties, learning and behavioural difficulties, and the quality of parenting, parenting stress, or parental psychopathology). Non-pharmacological treatment have included dietary and psychological approaches (behaviour therapy, focusing on specific skill impairment, neurofeedback interventions and working memory training). Although, there is not sufficient evidence to suggest that non-pharmacological treatments for ADHD significantly reduce the core symptoms of ADHD, they can be useful in managing significant secondary functional impairments associated with ADHD that are not optimally improved by medication. More research needs to focus on improving the methodological study designs to include larger sample sizes, randomized control trials and using adequate typically developing controlled groups. Combined pharmacological and non-pharmacological treatments for young people ADHD also requires further research.

## 1.11 CHAPTER CONCLUSIONS AND FUTURE RESEARCH DIRECTIONS

ADHD is one of the most common diagnoses for children and adolescents, although there is considerable variability in ADHD point prevalence estimation worldwide. Studies have found rates ranging from as low as 0.9% to as high as 20%. ADHD is categorised as a neurodevelopmental disorder in the DSM-5 (APA, 2013). ADHD is defined by impairing levels of inattention, disorganization, and/or hyperactivity-impulsivity that interferes with functioning or development. Three presentations of ADHD are recognised: a predominantly inattentive presentation; predominantly hyperactive-impulsive presentation; and a combined presentation when criteria for both inattentive and hyperactive-impulsive presentations are met. Two main classification systems are used for diagnosing ADHD, DSM and ICD. In Australia it is common for the DSM classification criteria to be used within the Mental Health Setting, so DSM-5 criteria will be used throughout this manuscript and for this research. Studies have shown differences in ADHD core symptoms across developmental stages: hyperactivity and impulsiveness are more prevalent in childhood; while inattention is more common across childhood, adolescence, and adulthood. Longitudinal studies demonstrate that ADHD-I is the most common ADHD presentation across the developmental stages. There are no diagnostic laboratory tests or diagnostic biological markers specific for ADHD. It is commonly diagnosed after a comprehensive assessment.

Historically, research has mainly focused on the biological nature of ADHD: It is estimated that first degree relatives in families with one ADHD member are seven times more likely to have ADHD and twin studies estimate ADHD heritability to be about 70%. But to fully understand the aetiology and developmental course of ADHD it is important to take an interactive biological and psychosocial approach. The relationship between genes and environment is transactional in nature, with ADHD being determined by genetic underpinnings but the developmental course of the disorder influenced by the interaction between biological and psychosocial risk and protective factors.

Individual child characteristics have been found to be associated with ADHD: gender; temperament (ADHD has been associated with reactive control, effortful control and the anger component of negative emotionality); intelligence (differences in Working

Memory and Processing Speed); academic achievement (lower reading, maths and spelling achievement); emotional regulation difficulties; and poor social skills, reduced peer acceptance and reduced friendship success. ADHD has also been seen to be influenced by psychosocial factors: family functioning (associated with higher ADHD severity and increased poor family functioning); increase parental relationship dysfunction; poor parenting practices; and increased parent psychopathology (Depression, Anxiety, ADHD, Antisocial Personality Disorder and increased substance abuse). A lot is still unknown about how ADHD plays out in families and the impact of these factors on ADHD, as they have been seen to be bidirectional in nature. Future longitudinal research is needed.

Co-occurring mental health problems are the norm, rather than the exception, among children and adolescents with ADHD. A greater than chance occurrence of ADHD with a number of other psychiatric conditions has been reported: Anxiety Disorders; Depressive Disorders; ASD; CD; and ODD. When a young person has comorbid conditions their ADHD symptomatology is often reported as more severe and they experience significantly more difficulties in their everyday functioning (peer and family relationships, academic achievement, and increased behavioural difficulties).

There are three main treatment approaches for ADHD: pharmacological (dexamphetamine, methylphenidate, and atomoxetine); non-pharmacological (dietary interventions, behavioural/psychosocial treatments such as cognitive behavioural therapy and social skills; neurofeedback and working memory training); and combined treatments (pharmacological and non-pharmacological interventions). Currently there is not enough evidence to suggest that non-pharmacological treatments are effective for the reduction of core ADHD symptoms. However, they can be useful in managing significant secondary functional impairments associated with ADHD. Further systemic research is needed in ADHD treatment, as pharmacological interventions are not always acceptable to parents and young people and are not always associated with functional improvements. Pharmacological interventions also do not seem to target secondary symptoms such as social difficulties, learning and behavioural difficulties, or the quality of family or marital functioning, parenting skill, or parental psychopathology.

# CHAPTER 2: Introduction to Oppositional Defiant Disorder (ODD)

## 2.1 INTRODUCTION TO OPPOSITIONAL DEFIANT DISORDER (ODD)

This chapter reviews the development of ODD. It covers: the historical context; the epidemiology of ODD, including point prevalence estimates, differences across gender, age, ethnicity and SES; diagnostic process, including the diagnostic manuals used and current diagnostic challenges; and the key clinical characteristics of ODD. The key aetiological risk factors for developing ODD will be covered including: key child characteristic components; child functional factors (temperament, IQ scores, Executive Functioning and Working Memory, academic achievement, emotional regulation, social competence); and key psychosocial components (family functioning, parenting skills, parental relationship functioning and parental psychopathology). In addition, key comorbid conditions (especially key clinical characteristics of ODD and Conduct Disorder, ODD and Depressive Disorders, ODD and Disruptive Mood Dysregulation disorder, ODD and Anxiety, and ODD and Autism Spectrum Disorder) will be explored. ODD and comorbid ADHD will be examined separately in chapter three. The developmental course (natural history) of ODD, treatment approaches including early intervention / prevention, pharmacological and non-pharmacological interventions, and future directions are also explored.

## 2.2 HISTORICAL CONTEXT OF THE DEVELOPMENT OF ODD

### 2.2.1 Introduction

Defiant and rule breaking young people are not a new occurrence, as records dating back to the 1600's describes such behaviour in children and adolescents. ODD as a formal diagnosis, however, was first introduced in the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM) (APA, 1980). Due to criticism that ODD just described "bad behaviour", small but significant changes have been made to the definition of ODD. The latest DSM-5 (2013) definition of ODD now includes that ODD behaviour is both behavioural and emotional and requires a persistent pattern of angry and irritable mood along with vindictive behaviour. This section will explore this in further detail.

### 2.2.2 ODD before the Contemporary DSM Classification

The DSM-II (APA, 1972) first described disruptive behavioural disorders in children and adolescents by using three distinct 'diagnoses': the run-away reaction; unsocialized aggressive reaction; and group delinquent reaction. However, there were not valid and reliable diagnostic criteria as such, and therefore the reliability of these 'diagnoses' was questionable (Ghosh, Ray, & Basu, 2017).

### 2.2.3 The DSMs: Contemporary Classifications of ODD

ODD was first introduced as a diagnosis in the DSM-III in 1980 as "Oppositional Disorder". This diagnosis focused on behavioural (violation of minor rules, temper tantrums, argumentativeness, provocative behaviour, and stubbornness), not affective features. The validity of the "Oppositional Disorder" diagnosis and its diagnostic threshold faced severe criticism (Mattison, Cantwell, Russell, & Will, 1979) with some researchers questioning if "Oppositional Disorder" was just bad behaviour (Rutter & Shaffer, 1980). Others suggested that "Oppositional Disorder" was really a milder form CD and it was hard to differentiate from it (Rey et al., 1988). Their primary concern was the significant overlap of "Oppositional Disorder" with CD criteria, so whenever criteria for CD were met, criteria for ODD were also met (APA, 1994). As a result, before the DSM-5, clinicians were not able to make a diagnosis of ODD if criteria for CD were met. Importantly in clinical studies, although many children who met criteria for CD also met criteria for ODD, the same was not true for community samples, where two-thirds to three-quarters of children with CD did not meet criteria for ODD (Rowe, Costello, Angold, Copeland, & Maughan, 2010). Contemporaneously, other research also demonstrated that ODD could be clearly distinguished from normative child behaviour (Keenan & Wakschlag, 2004; Rutter, Giller, & Hagell, 1998).

These issues were addressed by the DSM-III-R (1987) and DSM-IV (1994) revisions. In the 1987 edition of the DSM-III-R, "Oppositional Disorder" was revised to "Oppositional-Defiant Disorder" or ODD. DSM—III-R clarified that ODD was a separate disorder to CD, outlining that the symptoms of ODD were less aggressive than CD. Some research was also finding that ODD could be a pre-cursor to CD (Costello et al., 1996). DSM-III-R added two criteria: spiteful/vindictiveness; and angry and resentful attitude. To help differentiate ODD from normative behaviour, the frequency of

occurrence of ODD symptoms had to be disproportionate to the age and developmental stage of the child. DSM-IV did not change the diagnostic criteria significantly from DSM-III-R, except from removing swearing and changing the cut-off from five of nine, to four of eight criteria.

The new DSM-5 (2013) contained several refinements of the diagnosis of ODD. Dickstein (2010) noted that DSM-5 attempts to re-define ODD by emphasizing a 'persistent pattern of angry and irritable mood along with vindictive behaviour' rather than the DSM-IV's focus on 'negativistic, hostile, and defiant behaviour'. ODD as both a behavioural and emotional disorder is emphasised by its three categories: *vindictiveness; angry/irritable mood; and argumentative/defiant behaviour*. DSM-5 defined irritability as an 'angry/irritable mood' – with descriptors of 'loses temper, is touchy/easily annoyed by others, and is angry/resentful'. DSM-5 also included updated information about the frequency of the symptomatic behaviours and a severity rating was added. This diagnostic criterion will be discussed further in this chapter.

### 2.2.4 Summary

While bad behaviour of young people has been described through-out history, ODD is a relatively new diagnosis, being introduced in the DSM-III in 1980. ODD has a history of its symptomatology being explored within the category of CD, only being conceptualised as a separate disorder in the latest DSM-5 in 2013. ODD now describes symptoms that are necessarily both behavioural and emotional in nature.

## 2.3 EPIDEMIOLOGY OF ODD

### 2.3.1 Introduction

ODD is a relatively common childhood disorder, with a point prevalence rate ranging from 1% - 11%, with an average point prevalence estimate of around 3.3% (APA, 2013; Costello et al., 2003; Maughan, Rowe, Messer, Goodman, & Meltzer, 2004). Al-Yaman, Bryant and Sargeant (2002) reported that in Australia between 1999-2000, for children aged 1-14 years, ODD was one of the most frequent diagnoses accounting for hospital referral for mental health problems and behavioural disorders. Based on

international research, the point prevalence for ODD in non-clinical samples ranges from 2% -10% (Fergusson, Horwood, & Lynskey, 1993; Sanders, Gooley, & Nicholson, 2000).

ODD typically develops and becomes apparent in preschool years, often before the age of eight years old (APA, 2013; Connor, 2002; Fraser & Wray, 2008; Nock, Kazdin, Hiripi, & Kessler, 2007). It is unlikely to emerge after early adolescence (Rowe et al., 2010). The following section covers the key methodological factors associated with ODD prevalence rates, and how factors such as age, gender, ethnicity, and SES can affect the reported point prevalence rates of ODD.

### 2.3.2 Key Methodological Factors for ODD

Researchers have identified that prevalence rates can vary due to child characteristics and/or reporting factors: child characteristics (child's age at assessment; gender; and type of conduct problem behaviour displayed) (Sanders et al., 2000; Steiner, Remsing, & Issues, 2007) and reporting factors (such as the informant source (for example, parent verses teacher) (Grills & Ollendick, 2002); how data provided by different informants (parent, teacher or child) are combined (Kraemer et al., 2003); type of report (for example, concurrent verses retrospective); whether or not impairment is used in the definition of a case (Canino et al., 2004); and whether or not children meeting CD criteria are included (Quy & Stringaris, 2012; Steiner et al., 2007).

Which DSM version was used to diagnose an individual young person appears to have also affected ODD prevalence rates. When ODD was first included in DSM-III, the prevalence was 25% higher than when the DSM-IV revised the diagnostic criteria (Loeber, Burke, Lahey, Winters, & Zera, 2000). DSM-5 made further changes to the criteria grouping characteristics together requiring both emotional and behavioural symptomatology (APA, 2013). It will be important to assess in future studies if there is a decline in prevalence between the DSM-IV and the DSM-5 due to these further changes.

### 2.3.3 Age and ODD

Research regarding prevalence rates due to the child's age has not produced consistent findings. Costello et al. (2003) examined cross-sectional epidemiological studies of community samples of school age youth, which indicated higher prevalence

rates of conduct problems for the 12-16-year age group (7%) compared with the 4-11 year age group (4%) suggesting that prevalence rates increase with age. However, Canino et al., (2004), in a systematic review of the literature, found that children more than 12 years of age had lower ODD rates than children less than 12 years of age, consistent with a study of young people aged 9 to 17 years of age that reported higher levels of oppositional behaviour at younger ages, with aggression peaking near the middle of this age range (Lahey et al., 2000).

### 2.3.4 Gender and ODD

While numerous research studies have reported that ODD is typically more prevalent in boys than girls (Angold & Costello, 1996; Quy & Stringaris, 2012; Steiner et al., 2007; Zoccolillo, 1993), others have noted no gender differences (Lahey et al., 2000; Nock et al., 2007). Some research suggests that a gender difference may be due to age, ODD being more prevalent in boys than girls before adolescence with a ratio of 1.4: 1 (APA, 2013), with girls' prevalence increasing after puberty (Fraser & Wray, 2008). Nock et al. (2007) suggested that the sex difference in ODD prevalence may only be significant in Western cultures. Others suggest that the point prevalence rates for females may be curvilinear, with declining rates into young adulthood (24 years of age) (Leadbeater, Thompson, & Gruppuso, 2012), while the rate is stable for boys through to young men.

Since the introduction of ODD as an independent disorder, the field trials to inform the definition of this disorder have included predominantly male subjects. Some researchers propose that different ODD criteria should be used for girls. They point out that in early development (before adolescence), girls may exhibit aggression in ways that are not captured by the current definitions (for example, they tend to be less overtly aggressive and more covertly aggressive) (Steiner et al., 2007; Zoccolillo, 1993). Girls expression of anger tends to be indirect (for example, hidden, passive), verbal (as expressed in words rather than actions), and relational (as expressed in relationships) (Connolly & Bernstein, 2007; Connor, 2002; Steiner et al., 2007). Therefore, they recommend that additional definitional criteria for girls be added. Keenan, Wroblewski, Hipwell, Loeber, and Stouthamer-Loeber (2010) studied a large community sample of girls (n=2,451) longitudinally from the age 7-15 years, finding only about half of the girls

who met the criteria for CD at any phase also met the diagnosis for ODD. Rowe, Maughan, Pickles, Costello, and Angold (2002) showed in girls that ODD did not confer increased risk for the development of CD; rather, it was associated with increased risk of continued ODD, Depression and Anxiety, suggesting different outcomes of ODD in boys and girls.

Researchers have looked at how functioning and comorbidity differs in boys and girls. Munkvold, Lundervold, and Manger (2011) found no gender differences in degree of comorbid symptomology, with most children with ODD demonstrating clinical dysfunction in multiple domains. No differences in functional impairment were also found among children with ODD in middle childhood and pre-adolescence (Lourdes Ezpeleta, Keeler, Erkanli, Costello, & Angold, 2001). In contrast, others have found that boys with ODD have higher rates of aggression and externalizing symptoms (Carlson, Tamm, & Gaub, 1997), while girls are more likely to have Anxiety Disorders, Depression, self-harming behaviour, and somatic symptoms (Rowe et al., 2002; Trepat & Ezpeleta, 2011).

### 2.3.5 Ethnicity and ODD

Cross-cultural differences have been reported in prevalence data for ODD, indicating that estimates of the prevalence of ODD varies widely across countries (World Health Organization, 1992). Glorisa Canino, Lewis-Fernandez, and Bravo (1997) argued that there is evidence that cultural background influences the expression, interpretation and value given to psychiatric symptoms. DSM-IV included a section on Specific Culture, Age and Gender Features for each diagnostic category to assist clinicians evaluating variations of any given disorder that may be attributable to their culture. Prevalence rates can be affected by the degree to which ODD symptoms are considered dysfunctional and or are differentially tolerated in various cultures (Canino, Polanczyk, Bauermeister, Rohde, & Frick, 2010). For example, in Chinese and Thai culture suppression of aggression and strong emotions may lead to a lower threshold for externalizing behaviours through controlling them more quickly. Crijnen, Achenbach, and Verhulst (1999) found lower prevalence rates of externalizing syndromes in Asian countries as compared to several Western countries on the Child Behaviour Checklist (CBCL). However, in a systematic review of the literature by Canino et al. (2010) culture

was not associated with significant variability in prevalence estimates of ODD. They found that the variability in prevalence was mostly related to methodological differences across studies: the requirement of impairment for a diagnosis; and variability in diagnostic criteria.

### 2.3.6 Socio-Economic Status and ODD

ODD has been reported to be more common among children in low-income households (Hamilton & Armando, 2008; Loeber et al., 2000). Loeber et al. (2000) stated that neighbourhood and environmental factors may play a strong role in producing such symptoms, with the poorest and most violent neighbourhoods having the highest prevalence rates of ODD. Children living in poorer countries have higher exposure to poor prenatal care and poor infant nutrition, more exposure to toxic infection agents, live in disadvantaged and crime ridden neighbourhoods and are often exposed to other stressful circumstances associated with physical abuse and poverty (Canino et al., 2010), which are all risk factors that have been associated with ODD (Burke et al., 2002; Loeber et al., 2000). Low parental educational attainment and low household income are also independently associated with higher rates of ODD (Perou et al., 2013). However, as Canino et al. (2010) point out, there is evidence that protective factors related to positive family environment may lower the prevalence of ODD, even in developing or poor countries citing the results of a longitudinal study by Bird et al. (2006). Bird et al. (2006) compared the risk and protective factors among Puerto Rican children living in San Juan, Puerto Rico and the Bronx, which showed that close family attachments and strict family monitoring and supervision were associated with lower point prevalence of ODD (and CD). Frick and Kimonis (2005) argue that studies have not been able to show whether the high prevalence rates of ODD in poor or minority samples are due to differences in poverty, neighbourhood characteristics, cultural or other risk factors.

### 2.3.7 Summary

ODD is a relatively common childhood disorder, with point prevalence rates ranging between 1% - 11%. ODD typically develops in preschool years, often before the age of eight-years. However, research has not produced consistent findings regarding prevalence rates due to the young person's age, some suggesting rates increase with age,

others stating it falls after the age of twelve. Researchers have also identified that prevalence rates can vary due to other child characteristics (gender, ethnicity and SES) and reporting factors (informant source, type of report, if impairment was used as a criterion, whether CD was included, and what version of the DSM was used for classification). Further research in this area is required.

## 2.4 KEY CLINICAL CHARACTERISTICS OF ODD

### 2.4.1 Introduction

There are no laboratory tests or diagnostic markers specific for ODD. A diagnosis is made by a comprehensive assessment by a qualified professional. Researchers have noted some diagnostic challenges given that it is not uncommon for children or adolescents to display oppositional behaviours. This section will explore the process for diagnosing ODD, the difference in the diagnostic manuals (DSM-5 and ICD-10), and the challenges in diagnosing ODD.

### 2.4.2 Diagnostic Process for ODD

ODD is commonly diagnosed after a comprehensive assessment involving gathering information from multiple informants, over one or more sessions with a qualified professional (Paediatrician, Psychiatrist, Psychologist, or Mental Health Clinician). The same as for ADHD, information is often collected using a combination of: clinical interview with history taking and screening for associated comorbidities; observation; examination; and a variety of assessment tools (checklists, behaviour questionnaires, and/or rating scales). It is also important to gain an adequate understanding of contributing factors (such as: family functioning; parental relationship functioning; parenting style; and parental psychopathology).

The American Academy of Child and Adolescent Psychiatry (Steiner et al., 2007) issued a practice parameter for assessing ODD stating the diagnosis requires a comprehensive diagnostic evaluation, which includes an interview with the child or youth, the primary caregiver, and collateral informants, such as teachers. They reported that standardized reporting tools might be helpful for gathering data from informants but

did not specify a specific tool. The recommendations emphasized the importance of the clinician's relationships with both the family and young person and the clinician being considerate of cultural needs for both assessment and treatment rapport.

Other evaluations, such as tests of intelligence (for example, the Wechsler Intelligence Scale) and educational achievement (for example, the Wide Range Achievement Test-4) are also used to help rule out intellectual deficits and learning disabilities (Fletcher et al., 2005).

### 2.4.3 Diagnostic Manuals

The two main classification systems used for diagnosing ODD are the International Classification of Diseases, tenth revision (ICD-10) and Diagnostic and Statistical Manual of Mental Diseases, fifth edition (DSM-5). The ICD-10 (World Health Organization, 1992) categorises ODD under Mental and Behavioural disorders – behavioural and emotional disorders with onset usually occurring in childhood and adolescence, codes F90-98. In the current DSM (APA, 2013) ODD is categorised within Disruptive, Impulse-Control, and Conduct Disorders, codes 313.81 – 312.9.

In ICD-9, ODD was included under the diagnosis of Conduct Disorders, but not very well defined. ODD, in ICD-10 remains under the category of Conduct Disorders and requires that the overall criteria for Conduct Disorder be met but requires less severe symptoms. ICD-10 suggests that a continuum between ODD and CD exists. Conduct Disorders are characterized by a repetitive and persistent pattern of dissocial, aggressive, or defiant conduct. The behaviour needs to amount to major violations of age-appropriate social expectations (it should be more severe than ordinary childish mischief or adolescent rebelliousness) and should be an enduring pattern of behaviour (six months or longer).

DSM-5 describes ODD under Disruptive, Impulse-Control, and Conduct Disorders (APA, 2013) outlining these disorders as ones that include conditions involving problems in the self-control of emotions and behaviours. What separates them from other disorders are these problems are manifested in behaviours that violate the rights of others (for example, aggression, destruction of property) and / or bring the individual into significant conflict with societal norms or authority figures (APA, 2013). Other disorders

that are listed under this category are: Intermittent Explosive Disorder; CD; Antisocial Personality Disorder; Pyromania; and Kleptomania.

The DSM-5 (APA, 2013) states the main features of ODD are frequent and persistent patterns of behaviour, requiring four symptoms from any of the following categories: *angry / irritable mood* (often loses temper; is often touchy or easily annoyed; is often angry and resentful); *argumentative / defiant behaviour* (often argues with authority figures; often actively defies or refuses to comply with requests from authority figures or with rules; often deliberately annoys others; often blames others for his or her mistakes or behaviour); or *vindictiveness* (has been spiteful or vindictive at least twice within the past 6 months). These behaviours must have been occurring for at least 6 months and exhibited during interaction with at least one individual who is not a sibling. These behaviour difficulties occur outside of normal age-appropriate limits and are also considered to be outside a range that is normative for the individual's developmental level, gender, and culture. The difficulties need to be associated with distress in the individual or others in their immediate social context, and/or impact negatively on social, educational, occupational, or other important areas of functioning. The behaviours cannot occur exclusively during a psychotic illness, substance use, Depressive, or Bipolar Disorder. Also, the criteria are not met for Disruptive Mood Dysregulation Disorder. The current severity specifiers are: mild (symptoms are confined to only one setting (for example, at home, at school, at work, with peers); moderate (some symptoms are present in at least two settings); or severe (some symptoms are present in three or more settings). DSM-5 criteria for ODD will be used for this study.

### 2.4.4 ODD Diagnostic Challenges

Diagnosing ODD can be challenging as it is not uncommon for preschool children or adolescents to display oppositional behaviours at times. Therefore, the criteria for diagnosis that become important to help determine the diagnosis of ODD are that "there is a persistent pattern of oppositional and disruptive behaviours towards authority figures, for at least 6 months, and these cause distress to the family system and are impacting on the child's social and educational functioning" (Fraser & Wray, 2008). Clinicians must have a comprehensive understanding of normative presentations of these behaviours in

the different age groups in terms of frequency and duration to assess whether a clinical problem is present (Matthys & Powell, 2018).

It is not uncommon for parent and teacher ratings of child behaviours to be discrepant. Chen et al. (2017) questioned if this was due to parenting stress levels. They recruited 299 preschool children from community and clinical referrals and found that scores reported by parents were higher for each of the symptoms examined than those reported by teachers, and the degrees of agreement between informants ranged from low to moderate. The parental distress factor of parenting stress was associated with parent ratings, suggesting that parental distress should be considered when parent ratings scores show significant discrepancies from that of teacher rating scores.

As mentioned in the gender section of this chapter some researchers have questioned whether the diagnostic criteria are clinically relevant for use with females, whether gender-specific criteria and thresholds should be developed.

There is limited research that has looked at ODD in adulthood. Barry, Marcus, Barry, and Coccaro (2013) found that 28.7% of adults in a clinical sample meet ODD criteria. Research has shown that ODD does persist from adolescence into adulthood, but some have questioned if the diagnostic symptoms are the same: irritability symptoms were found to persist, but it is unclear if the behavioural symptoms (for example, arguing and defying, deliberately annoying others, blaming others) persist (Leadbeater & Homel, 2015). Further systematic longitudinal research in this area is required.

### 2.4.5 Summary

The two main classification systems used for the diagnosis of ODD are the DSM-5 and the ICD-10. In Australia, the DSM-5 is the most commonly used in mental health services. A diagnosis is usually made after a comprehensive assessment by a qualified professional. However, professionals face challenges in diagnosing ODD as it is not uncommon for preschool children and adolescents to display challenging and oppositional behaviour suggesting the professional must be experienced in understanding behaviour of a typically developing young person and ensure that there is a persistent pattern of behaviour, which has been occurring for at least six months, that causes significant distress to the family, school and/or wider social system of the young person.

Gender, age, and rater differences have also been noted and need consideration when making a diagnosis.

## 2.5 KEY AETIOLOGICAL RISK FACTORS FOR ODD

### 2.5.1 Introduction

Historically, there have been limited investigations specifically looking at the aetiological factors linked with ODD (Goldstein & DeVries, 2017). This may be due, in part, to a tendency to view ODD as a lesser variant of CD (Greene & Doyle, 1999; Hinshaw, Lahey, & Hart, 1993; Kuhne et al., 1997). Most of the data available on Disruptive Behaviour Disorders is contained in the body of research termed "conduct problems" (Biederman, Faraone, Milberger, Jetton, et al., 1996; Hinshaw et al., 1993; Steiner et al., 2007) making it difficult to interpret findings specifically related to ODD (Biederman, Faraone, Milberger, Jetton, et al., 1996; Greene et al., 2002). However, understanding the aetiology of a disorder is an important step in developing and implementing effective prevention and intervention strategies (Tremblay, 2010).

There is no one specific factor that has yet been identified as directly causing ODD (Goldstein & DeVries, 2017). Most researchers agree that the causes of ODD are multifactorial, arising out of a complex mix of increased risk and decreased protective factors present across multiple domains (for example, individual, family, peer and school), and exerting different levels of influence over time (Burke et al., 2002; Connor, 2002; Liberman, 2008; Loeber, Burke, & Pardini, 2009; McCart & Sheidow, 2016; Patterson, DeBaryshe, & Ramsey, 1989).

Several risk factors that have been identified for ODD are child biological factors (involving genetics, neuroanatomy, neurotransmitters, neurophysiology and neuroendocrinology), child functional factors (including temperament, IQ, Working Memory, academic achievement, emotional regulation, and social competence), school risk factors (for example, poor classroom management, high levels of classroom aggression, large class sizes, and poor school-home communication); and psychosocial risk factors (family functioning, parental relationship functioning, parenting stress, poor parenting skill / ineffective parenting, and parental psychopathology). Less is known

about protective factors (Burke et al., 2002), with good parenting practices, engagement and attachment with school, clear supervision and expectations, and a close and trusted relationship with a trustworthy adult being noted as protective against deviant behaviour. This section will explore the child functional factors and psychosocial risk factors for developing ODD that are relevant for the aims and hypotheses of this thesis. Whilst crucial, the biological factors noted above are beyond the focus of this thesis.

### 2.5.2 Individual Child Characteristics for ODD

Most of the research in this area has included ODD into a broader category of Disruptive Behaviour Disorders, including CD. This section will cover temperament, IQ, Executive Functioning and Working Memory, academic achievement, emotional regulation and social competence and their associations with ODD. Where the literature has explored ODD as a separate diagnosis it will be noted.

#### *2.5.2.1 Temperament and ODD*

Temperament is regarded as a constitutional facet of child development that may be observed very early in childhood and when dysregulated, may evoke maladaptive parenting (Lytton, 1990) and may facilitate the progression from early disruptive disorders to CD (Cole & Zahn-Waxler, 1992; Kingston & Prior, 1995). ODD has been strongly associated with temperamental risk factors (Connor, 2002; Loeber et al., 2009). Irritability and oppositional behaviour have been found to predict psychiatric disorders at follow-up (Stringaris, Cohen, Pine, & Leibenluft, 2009; Stringaris & Goodman, 2009). ODD has been reported to have temperamental profiles including high negative emotionality and activity; high novelty seeking; low persistence, and low effortful control (Lavigne, Gouze, Hopkins, Bryant, & LeBailly, 2012; Melegari et al., 2015; Stringaris et al., 2010). Rabinovitz et al. (2016) found anger/frustration acts as a risk factor for the development or persistence of behavioural difficulties through its influence on the poor development of control mechanisms.

ODD core traits, such as oppositional behaviour and negative emotionality, are considered measures of temperamental domains (Sanson & Prior, 1999; Wakschlag et al., 2007). Given that the symptoms of angry/irritable mood are among ODD symptoms it is no surprise that some research has found that negative affect predicted ODD (Antúnez,

de la Osa, Granero, & Ezpeleta, 2016; Martel, Gremillion, & Roberts, 2012). Sanson and Prior (1999) undertaking a longitudinal study reported that early temperament (specifically negative emotionality, intense and reactive responding, and inflexibility) was predictive of externalizing behaviour problems by late childhood. Wichstrøm, Penelo, Rensvik Viddal, de la Osa, and Ezpeleta (2018) assessed a community sample of 3 and 4-year old's over four waves of data collection. They found that negative affect (anger-frustration, discomfort, fear, sadness, low soothe-ability) predisposed children to symptoms of ODD (and ADHD, Anxiety and Depression), and low effortful control (attention focusing, inhibitory control, low-intensity pleasure, perceptual sensitivity) predisposed children to ODD symptoms (and ADHD).

Researchers have looked at the temperament dimension daring (a tendency to have positive socio-emotional responses to risky or novel experiences) from the developmental propensity model that outlines three independent dimensions of temperament – daring, negative emotionality, and prosociality; finding childhood daring positively predicted ODD/CD symptoms (Bai & Lee, 2017; Castellanos-Ryan, Rubia, & Conrod, 2011; Hampson, Andrews, & Barckley, 2008; Lahey et al., 2008; Lahey & Waldman, 2003).

However, some researchers have not found temperament to predict ODD in childhood. Fagot and Leve (1998) did not find a difficult temperament reported by parents at age two predictive of ODD (or CD) or reported by teachers at age five. Good parent management skills have also been found to demonstrate effects in moderating the relationship between children's temperament and disruptive behaviour disorders (Guo & Mrug, 2018). Studies have found that children are more likely to display externalizing problems if they have experienced harsh parenting or received high maternal control or low maternal positive guiding (Bayer, Sanson, & Hemphill, 2006; Rubin, Burgess, Dwyer, & Hastings, 2003). Antúnez et al. (2016) found a difficult child temperament was more likely to develop into ODD in the presence of parental psychopathology. They found that children with more difficult temperaments (for example, higher negative affect, poorer effortful control) had higher levels of ODD symptoms when parental Depression and Anxiety were also higher. Further longitudinal research investigating the associations of temperament, parent management skills and parental psychopathology is required to help us understand the impact of these factors on young people developing ODD.

Early research has demonstrated gender differences in temperament: female infants were less approaching to novel stimuli than male infants (Prior, Sanson, & Oberklaid, 1989); males were found to be more active than females (Eaton & Enns, 1986); boys displayed lower effortful control than girls (Else-Quest, Hyde, Goldsmith, & Van Hulle, 2006); and more externalizing problems in boys than girls in toddlerhood and later life stages (Juliano, Stetson Werner, & Wright Cassidy, 2006). However, gender differences in the relationship between temperament and ODD are not consistent. Further longitudinal studies of gender-specific effects of temperament on ODD are needed (Guo & Mrug, 2018; Sanson & Prior, 1999).

Moreover, replication and extension of temperament studies are required to be help identify children at risk for emergent conduct problems (Bai & Lee, 2017), specifically ODD separately from CD, to see if the pathway differs. Future research could assess the additive and multiplicative influences of other child traits, including pro-sociability and negative emotionality (Lahey & Waldman, 2003) and how temperament is linked to other ODD risk factors, as it could be a possible key marker for at-risk children given it is distinguishable from an early age (Burke et al., 2002). Research is also needed to look at how temperament and the family environment impact on ODD. Because temperament is malleable, interventions targeting the affective, attentional, and behavioural regulatory components of temperament may reduce disruptive behaviours (and other psychopathology) in children (Wichstrøm et al., 2018).

#### *2.5.2.2 Cognitive Function (IQ Score) and ODD*

Understanding the cognitive profiles of young people with ODD alone is difficult as there are limited studies in this area, many studies having combined ODD and CD into a single entity. In a review by Hogan (1999) examining the relationship between ODD/CD and IQ, 60% of the studies found significantly negative associations, however 80% of these studies did not control for ADHD. When ADHD was controlled for, 73% of the studies did not find ODD/CD to be linked to lower IQ. Frick et al. (1991) reported nonsignificant differences between young people with CD and clinic controls on full scale IQ. Forssman, Eninger, Tillman, Rodriguez, and Bohlin (2012) did look at ODD alone and did not find that cognitive functioning difficulties were associated with ODD behaviours.

Some researchers have explored how lower IQ may predict future ODD/CD using longitudinal study designs, finding that lower IQ was associated with greater risk for CD (Fergusson et al., 1993; Loeber et al., 1995). However, when environmental factors were controlled for (for example, SES and parental psychopathology) there were no differences on IQ measures between boys who did and did not develop CD (Lahey et al., 1995; Loeber et al., 1995). Lahey et al. (1995) found that high verbal IQ was related to a decrease in CD symptoms over time for boys in a clinic-referred sample, suggesting that high verbal IQ could be a protective factor.

In contrast, other researchers found that very young girls with conduct problems, compared with those without such problems, tended to have higher scores on measures of intelligence (Fagot & Leve, 1998; Sonuga-Barke, Lamparelli, Stevenson, Thompson, & Henry, 1994). Hence, further systematic research of the association of ODD and gender, and ODD separately from CD, needs to be undertaken.

#### *2.5.2.3 Executive Functioning, Working Memory and ODD*

Adequate functioning in emotionally charged or stressful situations requires flexibly adapting to constantly changing environments, in which Working Memory (holding information in mind - necessary for considering alternatives, making action plans and reasoning) is one important mechanism (Schoorl, van Rijn, de Wied, van Goozen, & Swaab, 2018). Most studies in this area have looked at young people with Disruptive Behaviour Disorders (combining CD and ODD), however it is not clear if ODD and CD are affected by deficits in Working Memory at the same level and separate assessments of both groups are needed (Matthys, Vanderschuren, Schutter, & Lochman, 2012).

Studies have revealed that Working Memory in children with ODD may be compromised, contributing to their antisocial and aggressive behaviour. Several studies have found that boys with ODD showed impairments in Working Memory (Séguin, Arseneault, & Tremblay, 2007; Syngelaki, Moore, Savage, Fairchild, & Van Goozen, 2009; Xu, Jiang, Du, Li, & Fan, 2017). Rhodes, Park, Seth, and Coghill (2012) found that boys with ODD demonstrated impaired memory performance in the areas of verbal memory tasks; spatial memory tasks; on the storage and central executive working memory factors; and the long-term memory factor. They suggested that verbal memory

difficulties were more closely associated with ODD than ADHD symptoms. Schoorl et al. (2018) suggested that these deficits might also be modulated by stress. They found that in a stressful situation, deficits in adaptation to the environment in boys with ODD became more prominent.

Although Working Memory deficits in young people with ODD have been found, to what extent they exist independently of ADHD comorbidity is controversial (Dolan & Lennox, 2013; Morgan & Lilienfeld, 2000; Pennington & Ozonoff, 1996). Some studies have found that impairments are more pronounced in those with ADHD and comorbid ODD, than ADHD alone (Dolan & Lennox, 2013). Some studies have controlled for ADHD and not found impairments in Working Memory for young people with ODD (Dolan & Lennox, 2013; Fairchild et al., 2009; Oosterlaan, Scheres, & Sergeant, 2005; Thorell & Wåhlstedt, 2006), while others have found impairments (Saarinen, Fontell, Vuontela, Carlson, & Aronen, 2015; Schoorl et al., 2018; Syngelaki et al., 2009).

#### *2.5.2.4 Academic Achievement and ODD*

Research indicates that young people with school difficulties, including low academic achievement and frequent truancy, are at very high risk for Disruptive Behaviour Disorders (Janosz, Le Blanc, Boulerice, & Tremblay, 2000). Given academic achievement is an important predictor of SES later in life, and wellbeing and health in adulthood (Huisman et al., 2005), it is important to understand its association with ODD in young people. The literature examining ODD alone is limited, most studies historically reporting on Disruptive Behaviour Disorders together (ODD, CD and ADHD) or ODD/CD).

ODD has been found to be associated with poorer functioning in school settings. Greene et al. (2002) examined 643 children and adolescents with ODD alone, 262 youth with ODD and CD, and 695 psychiatric comparison subjects. They found that while rates of repeated grade and remedial assistance did not differ among the three groups, the likelihood of placement in special classes did. They found that youth in both ODD and ODD/CD groups had a significantly greater likelihood of placement in special classes at school than the psychiatric comparison subjects. Harpold et al. (2007) interviewed a sample of adults with ADHD and ODD obtained through retrospective recall and found ODD was associated with a history of repeating school grades.

Reading disorders have been demonstrated to be related to ODD (Burke et al., 2002; Maguin, Loeber, & LeMahieu, 1993; Smart, Sanson, & Prior, 1996). However, Clark, Prior, and Kinsella (2002) found the reading levels of adolescents with ODD/CD were equivalent to those without behaviour problems. Gender differences for young people with ODD have been found regarding reading problems: for boys, disruptive behaviour was found to be a risk for later reading problems, but not vice versa; for girls, however, early reading problems were a predictor of adolescent disruptive behaviour (Maughan, Pickles, Hagell, Rutter, & Yule, 1996).

It has been questioned if the difficulties associated with negative school outcomes are due to comorbid ADHD, rather than ODD. Burke, Rowe, and Boylan (2014) recruited 177 boys between the ages of seven to twelve years from general psychiatric clinics. They found that ODD symptoms alone were not predictive of antisocial behaviour or low educational attainment. Liu, Huang, Kao, and Gau (2017) examined the impact of ADHD and ODD/CD on various school functions. The results showed that youths with ADHD had poorer performance across different domains of school functioning. Youths with ADHD and ODD/CD had more behavioural problems, but similar academic performance than those with ADHD only. They revealed that ADHD impaired academic performance, while ODD/CD aggravated behavioural problems, suggesting that the ODD/CD may specifically contribute to social difficulties in these young people rather than academic difficulties.

Parent functioning and parenting skill have been found to impact on child and adolescent academic outcomes. Babinski, Waschbusch, King, Joyce, and Andrade (2017) examined associations between parenting behaviours and school performance in children with externalizing behaviour problems (ADHD, ODD or CD). A medium size association was found between maternal parenting and school performance domains. They found that paternal positive involvement mitigated the negative effects of dysfunctional maternal behaviour on spelling achievement and peer functioning. These findings suggest it is important to consider parent functioning and parenting skill in the school performance of children with externalizing problems and by addressing parenting difficulties more adaptive school functioning and outcomes may be enhanced. Further longitudinal research investigating multiple aspects of academic achievement and their links with the development of ODD is required.

#### *2.5.2.5 Emotional Regulation and ODD*

It is not uncommon for young people with ODD to have emotion regulation difficulties. Research reports that children with externalizing problems display more negative emotion than healthy control participants in frustrating situations (Cole, Zahn-Waxler, & Smith, 1994; Gilliom, Shaw, Beck, Schonberg, & Lukon, 2002). Studies looking at ODD alone are limited, as most studies have combined ODD and CD together.

Researchers have found that young people with externalizing disorders are deficient in social information processing: they underutilize pertinent social clues; misattribute hostile intent to peers; generate fewer solutions to problems; and expect to be rewarded for aggressive responses (Dodge, 1991; Fontaine, 2006; Fontaine, Yang, Dodge, Pettit, & Bates, 2009; Pettit & Mize, 2007). van Bokhoven, Matthys, van Goozen, and van Engeland (2005) found that children with externalizing behaviour disorders demonstrated both lower baseline arousal and greater reactivity to peer provocation than children without behaviour disorders. Therefore, putting them at risk of displaying proactive aggression when faced with the opportunity to use aggression for instrumental gain, but also reactive aggression if provoked by a peer. Schoorl et al. (2018) found young people with ODD/CD, compared to children with other psychiatric conditions, had poorer emotion regulation reported by parents. However self-reports did not reveal any difference, indicating that the boys with ODD/CD do not perceive themselves as having impairments in regulating their emotions (they have a reduced awareness of this difficulty). Cavanagh, Quinn, Duncan, Graham, and Balbuena (2014) assessed 4,380 children using SNAP rating scales. They found that ODD items grouped together with emotional lability and irritability items, which did not group with CD, suggesting that ODD maybe more closely related to difficulties of emotional regulation than CD.

Parents can play a crucial role in assisting children to regulate their emotions. As children learn to self-regulate, parent's roles in modelling and coaching them how to regulate is essential and predicts children's emotion regulation capacity (Calkins, Smith, Gill, & Johnson, 1998). Without good modelling and coaching, particularly around anger, young people's risk for externalising problems increase (Cole, Teti, & Zahn–Waxler, 2003; Gilliom et al., 2002). Parental emotion coaching shows positive effects on young people's emotion regulation and externalizing problems (Dunsmore, Booker, & Ollendick, 2013; Dunsmore, Booker, Ollendick, & Greene, 2016; Shortt, Stoolmiller, Smith-Shine, Eddy, & Sheeber, 2010), emphasizing the importance of teaching parent's

emotion coaching skills as part of treatment. Beauchaine (2015) argues that externalizing disorders result from the combination of temperamental qualities with emotional control deficits, shaped through socialization processes, including poor parenting and early life stressors (such as poverty and neglect).

#### *2.5.2.6 Social Competence and ODD*

Behaviours associated with ODD (frequent temper outbursts, excessive arguing, defiance of rules and requests) can significantly impact on social interactions with peers, family members and teachers (Greene et al., 2002). Oliver, Barker, Mandy, Skuse, and Maughan (2011) looked at the associations between trajectories of conduct problems and social-cognitive competencies through childhood into early adolescence in a prospective population-based cohort. They found that social-cognitive deficits among those with early-onset persistent conduct problems were particularly apparent: 40.6% of boys and 24.3% of girls with persistent conduct problems. In general, most researchers have found children with disruptive behaviour disorders have poorer social relationships than non-disruptive peers, seen in conflicting interactions and frequent peer rejection, which continues into their adolescence (Campbell, 1990; Emond, Ormel, Veenstra, & Oldehinkel, 2007; Oliver et al., 2011; Pope, Bierman, & Mumma, 1989; Vitaro, Tremblay & Bukowski, 2001; Yoon, Hughes, Cavell, & Thompson, 2000).

Young people with Disruptive Behaviour Disorders have been found to make the same number of attempts at social interaction with their peers, but they are less successful in their interactions (Snyder, Horsch, & Childs, 1997). Research has found that young people with Disruptive Behaviour Disorders show marked differences in their behavioural responses in social situations and have greater social impairment (Dodge, McClaskey, & Feldman, 1985; Greene et al., 2002; Matthys, Maassen, Cuperus, & van Engeland, 2001; Webster-Stratton, Reid, & Hammond, 2001). They lack positive communication skills required for successful group interactions and are less likely than other children to be prosocial (Hughes, White, Sharpen, & Dunn, 2000).

Children with Disruptive Behaviour Disorders have been found to be more hostile towards peers, to interpret others' behaviours as more hostile (for example, people are out to get me, people will treat me badly), to be hypervigilant to cues of threat or hostility, and to miss out on benign or reconciliatory cues more than typically developing children

(Crick & Dodge, 1994; Muris, 2006; Schniering & Rapee, 2004). Frankel and Feinberg (2002) found that a diagnosis of ODD was associated with increased hostility towards peers and decreased resistance to provocation by peers. Horsley, de Castro, and Van der Schoot (2010) however, argue that young people with ODD do attend to benign or reconciliatory cues, but they ignore this information and encode only the hostile or threatening ones, as they do not fit their expectations. Similarly, Troop-Gordon, Gordon, Vogel-Ciernia, Ewing Lee, and Visconti (2018) reported that children with aggressive behaviour problems paid as much visual attention to relevant social cues as their peers, but then selectively remembered and recalled only the cues that fit a hostile interpretation. Kempes, de Castro, and Sterck (2008) found young people with Disruptive Behaviour Disorders misinterpret reconciliatory emotional signals (for example, regret or sympathy) as lies, resulting in escalating peer conflicts. In addition, de Castro, Verhulp, and Runions (2012) found that boys with Disruptive Behaviour Disorders had different goals and experienced different emotional tendencies than other children, focusing on goals directed at punishing peers for alleged wrong doing.

Several studies have looked at how children view themselves in social interactions (social self-view). They have found that children with Disruptive Behaviour Disorders do not generally hold negative social self-views, as they appear to overestimate their own social competence (Diamantopoulou, Rydell, & Henricsson, 2008; Webster-Stratton & Lindsay, 1999). Boys with Disruptive Behaviour Disorders focused on concrete and external qualities, adopted an egocentric bias in describing their peers and had less prosocial goals (Matthys et al., 1995; McDonald & Lochman, 2012).

Affiliation with like peers further impacts on the behaviour and social role of a child or adolescent with disruptive behaviour problems (Burke et al., 2002). Young people with Disruptive Behaviour Disorders typically have associations with deviant peers (for example, delinquent and/or substance using friends) (Dodge, Dishion, & Lansford, 2007). Cross-sectional and longitudinal studies have established a positive relationship between disruptive behaviour problems and deviant peer affiliation in youth (Liberman, 2008; Morse et al., 2013; Patterson, Dishion, & Yoerger, 2000). Exposure to deviant peers may lead to the initiation of delinquent behaviour in boys (Elliott & Menard, 1996; Keenan, Loeber, Zhang, Stouthamer-Loeber, & van Kammen, 1995), or may enhance pre-existing delinquency (Coie & Miller-Johnson, 2001). Although the

relationship is reciprocal, Matsueda and Anderson (1998) found that conduct problems have a stronger effect on peer associations than the converse.

Social competence is also impacted upon by other risk factors: low intelligence puts children at risk for developing maladaptive social-cognitive process and disruptive behaviour problems (Hyde, Shaw, & Moilanen, 2010); children with limited Working Memory capacities have been found to have impaired social problem solving skills (Yaghoub Zadeh, Im-Bolter, & Cohen, 2007); children with low inhibition evaluate aggressive responses more positively (Van Nieuwenhuijzen et al., 2017) and being exposed to adverse social experiences (for example, peer rejection, harsh parental discipline, community violence) develops deviant social-cognitive dispositions which predicts disruptive behaviour problems (Dodge & Pettit, 2003; Troop-Gordon & Ladd, 2005). Further systematic longitudinal research is required to look more closely at the interactions of different risk factors and their contribution to poor social competence in young people with ODD.

#### *2.5.2.7 Summary*

It is important to look at the individual child characteristics that might put young people at risk of developing ODD. Factors such, as temperament, cognitive functioning, Working Memory, academic achievement, emotional regulation, and social competence have been explored.

ODD has been associated with temperament risk factors including high negative emotionality and activity, high novelty seeking, low persistence and low effortful control. Yet, there have been inconsistent findings among researchers, some not finding difficult temperaments as predictors of ODD. Parenting (harsh parenting, high maternal control, or low maternal positive guiding) and parental psychopathology have also been found to moderate the relationship between children's temperament and Disruptive Behaviour Disorders. Gender differences have been noted, and future longitudinal studies of gender-specific effects on temperament are needed. Further longitudinal studies are also required to identify if temperament plays a different role in the pathways to ODD compared with CD.

There are limited studies to help us understand the cognitive profiles of young people with ODD alone as many studies have combined ODD and CD into a single entity

and have often not controlled for comorbid ADHD. Some studies have found that lower IQ predicts future ODD/CD. However, when environmental factors were controlled, there was no difference on IQ measures for those who developed Disruptive Behaviour Disorders. There is also limited research in the area of gender differences, some researchers finding young girls with conduct problems tended to have higher scores on measures of intelligence. Further longitudinal research is required to look carefully at the relationship between IQ and ODD.

Most studies looking at Executive Functioning and Working Memory in young people with Disruptive Behaviour Disorders have combined ODD and CD. Therefore, it is not clear if ODD and CD are affected by deficits in these areas in the same way. Some studies have found that boys with ODD showed impairments in Working Memory. Although to what extent Working Memory deficits exist independently of ADHD comorbidity remains controversial, as some studies that have controlled for ADHD have not found impairments in Working Memory. Some studies have reported that verbal memory difficulties may be more closely associated with ODD than ADHD symptoms, especially in stressful situations. Further systematic longitudinal research in this area for ODD is required.

Academic achievement is an important predictor of SES later in life, wellbeing, and health in adulthood. Research indicates that young people with school difficulties (low academic achievement and frequent truancy) are at very high risk for Disruptive Behaviour Disorders. However, the literature for ODD alone is limited, most studies reporting on Disruptive Behaviour Disorders together. ODD has been associated with poorer functioning in school settings and negative school outcomes. Some researchers have questioned if these difficulties are due to comorbid ADHD, rather than ODD alone. Parent functioning and parenting skill have also been found to impact on young people with ODD and their academic performance, finding positive involvement could mitigate negative effects on some academic outcomes. Future systematic longitudinal research of the association between ODD and a range of academic achievement measures is required.

Research reports that children with externalizing problems display more negative emotion than children without disruptive behaviour problems. Studies have found that young people with externalizing disorders are deficient in social information processing (underutilize pertinent social clues, misattribute hostile intent to others, generate fewer

solutions to problems and expect to be rewarded for aggressive responses). Children with ODD have been found to have poorer emotion regulation skills compared to children without ODD. Some researchers suggest that ODD is more closely related to difficulties of emotional regulation than CD. Parents with good emotion coaching skills have been found to show positive effects on young people's emotion regulation and externalizing problems, suggesting that ODD and emotion regulation difficulties can be shaped through positive socialization processes.

Young people with ODD have been found to have poorer social relationships than non-disruptive peers, seen in conflicting interactions and frequent peer rejection. Research has found that young people with Disruptive Behaviour Disorders show marked differences in their behavioural responses in social situations and have greater social impairment. They lack positive communication skills required for group interactions and are less likely than other children to be prosocial. They are found to be more hostile towards peers and to interpret others' behaviours as more hostile. Several studies have found that children with Disruptive Behaviour Disorders do not generally view themselves as having social difficulties and appear to overestimate their own social competence. Young people with Disruptive Behaviour Disorders typically associate with deviant or like peers which can lead to an increase in their delinquent behaviour.

Young people with ODD have been found to have deficits in many different areas that are likely to contribute to difficulties in overall functioning and place them at risk for persistent ODD. More systematic longitudinal research is needed to look at ODD separately from CD, as little is known about young people specifically with ODD. Further longitudinal research is also required to understand the relationship between, cognitive functioning, working memory, temperament, deviant peers, environment, and social competence in ODD.

### 2.5.3 Key Psychosocial Risk Factors for ODD

Several studies have well documented the significant linkage between family context and child psychological development (Hetherington & Martin, 1979). Family factors, such as poor family functioning, parental relationship functioning, poor parenting practices, and familial psychopathology have all been noted as risk factors for developing Disruptive Behaviour Disorders (Burke et al., 2002; DeLisi & Vaughn, 2014; Greene et

al., 2002; Marmorstein, Iacono, & McGue, 2009; Matthys & Lochman, 2016; Pardini & Frick, 2013). These will be discussed in the following section.

#### *2.5.3.1 Family Functioning and ODD*

Young people who live in families with unstable family structures and deficits in family functioning are more likely to show Disruptive Behaviour Disorders. Numerous studies have examined the extent to which family disruption and "non-traditional" family forms are associated with variations in rates of child behaviour problems. Epidemiological studies in a range of Western societies provide evidence of elevated rates of ODD among children in "non-traditional" family settings or who have experienced family disruption (Farbstein et al., 2010; Ford, Goodman, & Meltzer, 2004).

Poor family functioning, less cohesive families and more family conflict have been associated with ODD (Greene et al., 2002; Lucia & Breslau, 2006). Harvey, Metcalfe, Herbert, and Fanton (2011) examined the role of family experience in the early development and maintenance of ODD symptoms in preschool-age children with behaviour problems, finding the importance of early family functioning in the development of ODD. Lavigne, Dahl, Gouze, LeBailly, and Hopkins (2015) assessed a community sample of 344 children and found family conflict was a significant predictor of parent-rated symptoms of ODD, but not teacher-rated ODD symptoms. Greene et al. (2002) found that young people with ODD had significantly greater impairment in family functioning than did subjects without ODD.

Cox and Paley (2003) proposed that families are a dynamic and interactive system consisting of interdependent subsystems, including whole-family factors, parent-child subsystem, co-parenting subsystem, and marital subsystem. Therefore, different associations of multi-level family factors and child ODD symptoms should be explored together. A few longitudinal studies have looked at the complex interactions among several of the disruptive behaviour risk factors. Lavigne et al. (2015) examined the associations of contextual (stress and family conflict), parent (Depression), parenting (hostility, support, and scaffolding), and child factors (receptive vocabulary; negative affect; effortful control; inhibitory control; attachment; and sensory regulation). They found that higher scores on family risk factors (family conflict, parent hostility in parenting) and child temperament were positively associated with child ODD symptoms.

They also looked at both parent and teacher ratings of child ODD symptoms, showing that there was poor agreement between parents and teachers suggesting that ratings by different raters should not be considered to be equivalent.

Similarly, Smeekens, Riksen-Walraven, and van Bakel (2007) looked at four domains of factors in a longitudinal study: (a) parent-child interaction and parent-child attachment, (b) child characteristics (temperament and cognitive abilities), (c) parental characteristics (personality), and (d) contextual characteristics (SES, partner support, and stressful life events). They found that multiple family factors (parent-child interaction, parent-child attachment, and various parental, child, and contextual characteristics) served as predictors of child later externalizing behaviour problems. Generally, studies looking at multiple interactions between risk factors indicate that when families experience high conflict and poor relationships, they are more likely to reduce parental monitoring over time. Reduced monitoring allowed more time for young people to increase their time with deviant peers and less time at school, increasing the risk of academic failure (McCart & Sheidow, 2016).

However, these models were too complex to demonstrate the hierarchy of these family factors. Lin et al. (2013) proposed a three-level model which delineated the mechanism of ODD development (based on the Family System Theory (Cox & Paley, 2003) and the Bioecological Model (Bronfenbrenner, 2009)). This model divided family factors into three levels, including the whole level, the dyadic level (including, couple dyadic and parent-child dyadic levels) and the individual level (including, parental individual and child individual levels). They found three levels of family factors showed different associations with affective and behavioural ODD symptoms. Family factors at the whole level were less correlated with ODD symptoms than factors at the dyadic and individual levels. Also, multi-level family factors showed different associations with two dimensions of child ODD symptoms in that all family factors were more closely linked to affective ODD symptoms. Only the most proximal factors (the factors directly related to children—parent-child relationship and child emotion regulation) showed significant association with child behavioural ODD symptoms. The present study highlighted the value of studying child affective and behavioural ODD symptoms in the family context. They suggested that future research should focus on family factors at the dyadic and individual levels, and ODD symptoms should be divided into two separate dimensions that differentially depict affective and behavioural ODD symptoms. They also

recommended that further research using a longitudinal design (they used a cross-sectional study method) is needed to examine the direction of the associations between multilevel family factors and child ODD symptoms and potential mediating effects.

#### *2.5.3.2 Parental Relationship and ODD*

The parental relationship has been acknowledged as the core of family solidarity and the key element in determining the quality of family life (Erel & Burman, 1995). Parental discord and conflict have been seen to be more significant to child maladjustment than family intactness (Amato & Keith, 1991; Emery, 1982; Harold & Conger, 1997). Research has linked parental relationship quality to both children's internalizing and externalizing problems (Amato & Keith, 1991; Davies et al., 2002; Fincham, Grych, & Osborne, 1994). Where parental relationship quality is poor, research has found increased externalizing problems in children (for example, CD, aggressiveness and delinquency/antisocial behaviour); increased internalizing problems in children (for example, Depression, Anxiety, withdrawal); increased sibling and peer social problems (Stocker & Youngblade, 1999); and cognitive or academic problems (Grych & Fincham, 1990).

Several studies have found that parental relationship conflict is associated with heightened rates of children with Disruptive Behaviour Disorders (Bornovalova, Blazei, Malone, McGue, & Iacono, 2013). Erath and Bierman (2006) reported that exposure to parental relationship conflict was upsetting to children and appeared to elicit child maladjustment. They found links between aggressive parental relationship conflict, high rates of parental disagreement and child aggressive-disruptive behaviour at home and school.

Merikangas et al. (2010) found that parental divorce or separation were associated with increased risk of behavioural disorders in 13-18-year-olds, even when ethnicity, urbanicity, parental education and poverty were controlled. In a meta-analysis of studies of parental divorce by Amato and Keith (1991), divorce was associated with some decrements in children's functioning across a range of outcomes, including associations with conduct problems and delinquency. Studies investigating children in step-families report higher rates of disruptive behaviour problems and delinquency (Apel & Kaukinen, 2008; Coleman, Ganong, & Fine, 2000). However, some researchers have argued that

family breakdown is associated with a range of other factors (including, parental antisocial tendencies, and/or marital conflict while together) and that children in divorcing families manifest poor adjustment before their parents' divorce (Jaffee, Strait, & Odgers, 2012), suggesting that it is not the divorce itself, but the numerous factors associated with the divorce (Zimet & Jacob, 2001).

Some researchers argue it would be useful for further research to assess if some children are more vulnerable to the effects of family transitions than others, these might include characteristics such as gender; temperament; SES status; ethnic differences; higher IQ; maternal education; maternal sensitivity; family functioning prior to a family breakdown; the role of parental distress; and disruptions to family relationships, routines and parenting (Maughan, Rowe, & Murray, 2018).

Parkes, Green, and Mitchell (2019) argue that it is also important to understand the protective effects of positive dimensions of couple relationships on children's externalizing behaviour. They looked at the supportiveness in the dyadic couple relationship and the contribution of 'co-parenting' (couples' support for one another's individual parenting) on a child's behaviour. They found that couple supportiveness was associated with reduced externalising problems eight to ten years later, suggesting helping parents to support each other and improve their co-parenting may lessen the longer-term impact of couple relationship problems on the children.

Further research in this area is needed to fully understand the impact of parental relationship dysfunction on the development of ODD. Zimet and Jacob (2001) suggest that different dimensions need further investigation in relation to parental relationship conflict, including: *frequency*, how often the children are exposed to parental relationship conflict; *intensity*, low intensity (a calm discussion) to high intensity (physical violence); *content*, certain topics of conflict might be more or less threatening to children; *resolution*, from no resolution to a complete resolution (which has less negative reactions observed in children). They also recommend (since mechanisms are rarely so simple that they are fully explained by a single moderator or mediator) that multiple factors need to be looked at. They noted that the magnitude of the association between parental conflict and child maladjustment is only in the small to moderate range, accounting for approximately 10% of child outcome variance (($r$=0.46, Erel and Burman (1995); 0.33, Jouriles and Farris (1992)). They recommend factors including *modelling* (the cognitive model of the child

that is learned by imitating the behaviours of their parents); *the child's cognitive factors* (the children's perceptions when exposed to marital conflict via their appraisals, attributions, and coping techniques); *emotional insecurity* / *quality* of the parent-child interactions and *changes* in the quality of the parent-child relationship *over time*; the child's *temperament*; the *stage of development/age* of the child; and *gender*. Other researchers recommend also including factors such as: parental psychopathology, especially Antisocial Personality Disorder (Bornovalova et al., 2013); and the child's genetic make-up (Canino et al., 2010; Moffitt, 2003).

#### *2.5.3.3 Parenting Skill and ODD*

Numerous studies demonstrate that poor parenting / maladaptive parenting is related to Disruptive Behaviour Disorders (Bornovalova et al., 2013; Burke et al., 2002; Haapasalo & Tremblay, 1994; Niu, Liu, & Wang, 2018). Factors that have been correlated with children's disruptive or delinquent behaviour are: the degree of parental involvement; poor parent-child relationship; parent-child conflict management; poor monitoring; and harsh and inconsistent discipline (Burt, McGue, Krueger, & Iacono, 2005; Jaffee, Caspi, Moffitt, & Taylor, 2004; Tang, Lin, Chi, Zhou, & Hou, 2017). Stormshak, Bierman, McMahon, and Lengua (2000) found that punitive discipline, physically aggressive punishment, and low parental warmth/involvement by parents were common risk factors among children with oppositional and aggressive behaviours.

Bornovalova et al. (2013) found mother's parenting behaviours had a higher impact on child Disruptive Behaviour Disorders than the father's parenting behaviours. Although, Niu et al. (2018) found adolescents' perception of corporal punishment as 'normal' buffered the association between parental corporal punishment and adolescents' externalizing problem behaviours, emphasizing the importance of considering how the child and adolescents' perceptions may influence the effects of parental harsh discipline on developing ODD.

There may also be a bi-directional relationship between parenting behaviour and child behaviour. Researchers suggest that children are at higher risk of developing Disruptive Behaviour Disorders due to a social learning and reciprocal process: over time children learn that oppositional and aggressive behaviours are effective ways to avoid undesired activities (for example, going to bed, doing chores), and parents become

increasing disengaged from attempting to control their child's behaviour, thus developing families with a lack of warmth, high rates of conflict, and poor parental monitoring (McCart & Sheidow, 2016; Shaw, Owens, Giovannelli, & Winslow, 2001; Snyder & Patterson, 1995). This model suggests that child behaviour can modify parenting behaviours in maladaptive ways over time (Deater-Deckard, 2000).

Research has found it difficult to separate parenting behaviour from parental psychopathology in the contributions to the development of Disruptive Behaviour Disorders in children (Burke et al., 2002; Kaplan & Liu, 1999). Kaplan and Liu (1999) suggest while both poor parenting and parental psychopathology contribute to the development of these disorders, parental psychopathology may be a stronger determinant of disruptive behaviours in children than parenting behaviour. Smeekens et al. (2007) also recommend that parental personality (for example, ego-resiliency: resourceful adaptation to changing circumstances, including problem-solving strategies and the ability to perform under stress) needs further exploration as a potential predictor of ODD in children, rather than a strict parental psychopathology diagnosis alone.

The role that child gender differences play is also unclear in the literature. Most of the parenting research has been undertaken with boys. However, parents have been found to interact differently with boys compared to girls (Keenan & Shaw, 1995). Further longitudinal research is needed in child gender differences and the development of ODD.

Some researchers have noted that there is a need for comprehensive models that include both risk and protective factors to explain the relationship between parenting and ODD (Burke et al., 2002). They suggest factors including the severity of punitive physical punishment; culture; the nature of the relationship between the parent and child (Deater-Deckard & Dodge, 1997) and child's contributions (Pike, McGuire, Hetherington, Reiss, & Plomin, 1996).

#### *2.5.3.4 Parental Psychopathology and ODD*

Parental psychopathology has been established as a contributing factor to behaviour problems in children and adolescents, including ODD (Breaux et al., 2014; Harold, Elam, Lewis, Rice, & Thapar, 2012; Knutson, DeGarmo, Koeppl, & Reid, 2005). Research has primarily focused on maternal Depression, specifically in relation to the development of ODD, but it is important to note this is not the only type of parental

psychopathology associated with ODD. ODD also tends to occur in families with a history of parental ADHD, parental Disruptive Behavioural Disorders, Substance Use Disorders, Mood Disorders, and Antisocial Personality Disorders, suggesting that a vulnerability to develop ODD may be inherited (Bornovalova et al., 2014; Bountress & Chassin, 2015; Chronis et al., 2003; Hirshfeld-Becker et al., 2008; Shaw, Vondra, Hommerding, Keenan, & Dunn, 1994). Hirshfeld-Becker et al. (2008) found parents with comorbid disorders, such as Depression and a Disruptive Behaviour Disorder, are even more likely to have a child with a Disruptive Behaviour Disorder.

Maternal Depression is commonly linked to ODD in children and adolescents (maybe since Depression is one of the most common mental health disorders) (Barry, Lindsey, Fair, & DiSabatino, 2018). Many studies have connected maternal Depression and children and adolescents with externalizing behaviours (Hirshfeld-Becker et al., 2008; van der Molen, Hipwell, Vermeiren, & Loeber, 2011). van der Waerden et al. (2015) reported that the more severe and chronic the mother's Depression was, the more severe the behaviour problems in the child were. Maternal Depression has been found to predict both concurrent and future externalizing behaviours in children (Blatt-Eisengart, Drabick, Monahan, & Steinberg, 2009). Maternal Depression was found to predict poorer treatment outcomes among children with a Disruptive Behaviour Disorder (Muratori et al., 2015). However, less research has looked at the effect of paternal Depression on behaviour problems and has found conflicting results. Tully, Iacono, and McGue (2008) did not find paternal Depression to predict CD; however, Ohannessian et al. (2005) and Ramchandani et al. (2008) found that it did predict child ODD and CD.

Most research assumes parental psychopathology plays a causal role in the development of ODD. However, child behaviour problems and parental psychopathology often have a bidirectional relationship, with higher levels of parental distress being linked to more child behavioural problems, and more frequent problematic child behaviours being associated with increased parental distress over time (Neece, Green, & Baker, 2012). Parental psychopathology therefore may also serve to maintain ODD once established through a reciprocal process (Kazdin & Wassell, 1999; McKee, Colletti, Rakow, Jones, & Forehand, 2008). Parental psychopathology is not only a predictor, but also a risk factor, interacting with other predictors (difficult temperament and poor parenting skill/harsh discipline) to increase the risk of developing ODD and the severity of ODD (McKee et al., 2008).

Some researchers found that parental psychopathology can be a barrier to accessing treatment (Kazdin & Wassell, 1999) and treatment outcomes (Kazdin & Wassell, 2000). Owens et al. (2002) found that parental mental health was a barrier for parents to access services to get help to manage their child's difficult behaviours. Kazdin and Wassell (2000) found the higher level of parental psychopathology predicted not only barriers to participation in outpatient treatment, but also therapeutic change among clinically referred children. These studies highlight the importance of future research into the aspects of parental psychopathology and treatment of parental psychopathology that could form part of the optimal intervention package for young people with ODD.

#### *2.5.3.5 Summary*

Key psychosocial risk factors (family functioning, parental relationship functioning, parenting skill, and parental psychopathology) have been explored as risk factors for developing ODD. However, again research is limited for ODD independent from the other Disruptive Behaviour Disorders. Young people who live in families with unstable family structures and deficits in family functioning are more likely to show Disruptive Behaviour Disorders. Epidemiological studies have found evidence of elevated rates of ODD among children in "non-traditional" family settings and who have experienced family disruption. Several studies have found parental relationship conflict is also associated with heightened rates of children with Disruptive Behaviour Disorders, including ODD. However, some researchers argue, that family breakdown is associated with a range of factors (including, parental antisocial tendencies and marital conflict while together) and that children in divorcing families manifest poor adjustment associated with the whole divorce process. Some research has focused on the quality of the family functioning and family conflict as predictors of ODD, finding poorer family functioning and increased conflict predicted parent rated symptoms of ODD.

Parenting practices and parenting skill have both been associated with oppositional defiant symptoms. A number of studies show that poor parenting / maladaptive parenting is related to Disruptive Behaviour Disorders. Factors such as, the degree of parent involvement, poor parent-child relationships, parent-child conflict management, poor monitoring and harsh and inconsistent discipline are common risk factors among children with oppositional and aggressive behaviours. Studies have also

found that young people's behaviour can modify parenting behaviours in maladaptive ways over time, suggesting a reciprocal relationship.

Parental psychopathology has been established as a contributing factor to behaviour problems in children and adolescents, including ODD. A lot of research has focused on maternal Depression specifically in relation to the development of ODD, but it is important to note that parental ADHD, parental Disruptive Behavioural Disorders, Substance Use Disorders, Mood Disorders and Antisocial Personality Disorder also have been found as risk factors for developing ODD. Research has found child behavioural problems and parental psychopathology to have a bidirectional relationship, with higher levels of parental distress being linked to more child behavioural problems, and more frequent problematic child behaviours being associated with increased parental distress. Parental psychopathology is important to address in treatment as it has been seen as a barrier to children with ODD accessing services and treatment outcomes.

Current emphasis has been placed on interactive models of the risk factor contribution to the development of ODD and further longitudinal research is needed to look at how ODD is multi-factorially determined. Further systematic longitudinal research is needed to fully understand the impact of parental relationship dysfunction on the development of ODD and its interaction with other factors such as parenting skills, parental psychopathology, child's cognitive factors, child's temperament, and gender. In addition, further research is needed in the area of protective factors and exactly how they mitigate the impact of ODD risk factors.

## 2.6 KEY COMORBID CONDITIONS FOR ODD

### 2.6.1 Introduction

Young people with ODD have significantly higher rates of comorbid psychiatric disorders (Steiner et al., 2007). Researchers often report that ODD co-occurs with ADHD, CD, Mood Disorders (MD, PDD), DMDD, Anxiety Disorders, and ASD. In a population-based study, Angold et al. (1999) found that of young people with ODD, 14% of children had comorbid ADHD, 14% have comorbid Anxiety Disorder, and 9% have comorbid Depressive Disorder. More recently research has focused on ODD as a distinct

psychiatric disorder, independent of CD, but research in this area is limited. This section will cover the common comorbid disorders with ODD.

### 2.6.2 Conduct Disorder and ODD

DSM-IV precluded making a diagnosis of ODD in the presence of CD as the literature was not clear whether ODD and CD could be comorbid (Steiner et al., 2007). Historically, there were questions if they represented two parts of a single continuum or construct (Moffitt et al., 2008). However, there is significant evidence that they are two distinct disorders (Pardini & Frick, 2013; Rowe et al., 2010), and the majority of children with ODD do not develop CD (Lahey & Loeber, 1994; Loeber et al., 2000; Rowe et al., 2010). ODD has been found to have different socio-environmental and genetic correlates than CD (Dick, Viken, Kaprio, Pulkkinen, & Rose, 2005; Hudziak, Derks, Althoff, Copeland, & Boomsma, 2005). It is now accepted and defined in DSM-5 that ODD and CD are different diagnoses and often co-occur in youth (Angold et al., 1999; Burke, Waldman, & Lahey, 2010; Nock et al., 2007; Rowe et al., 2010).

Rowe et al. (2010) examined the links between ODD, CD and their young adult outcomes in a longitudinal dataset of 1420 individuals aged nine to twenty-one years of age. They found key differences: CD largely predicted behavioural outcomes; whereas ODD showed greater prediction to emotional disorders in early adult life; the irritable and headstrong dimensions in ODD symptoms showed prediction to later behavioural and emotional disorders. Overall, they reported that their results emphasise the value of retaining separate ODD and CD diagnoses in DSM-5. Steiner et al. (2007) found that young people with ODD and comorbid CD have higher rates of Mood Disorders and social impairment than those with ODD alone.

Given the recent clarification of CD and ODD should be investigated as distinct disorders, research into the comorbidity of CD and ODD is only in its early stages. Further research is required to better explain the processes that lead to co-occurring or developmental associations between CD and ODD (Lochman & Matthys, 2018).

### 2.6.3 Major Depressive Disorder (MDD), Persistent Depressive Disorder (PDD: formally known as Dysthymia) and ODD

Depression is commonly associated with ODD and can develop as early as preschool age (Nock et al., 2007; Rowe et al., 2010; Steiner et al., 2007). Angold et al. (1999) found that up to 9% of young people with ODD also have Depression. Stringaris and Goodman (2009) reported that those with angry and irritable symptoms of ODD are at higher risk of comorbid Mood Disorders.

Several studies have often combined Disruptive Behaviour Disorders (ODD and CD) and Mood Disorders (MD and PDD) when examining outcomes. Ezpeleta, Domènech, and Angold (2006) reported that, in a sample of 382 children attending a psychiatric outpatient clinic, 92 had Depressive Disorders without ODD/CD, 165 ODD/CD without a Depressive Disorder and 125 had both. They found comorbidity accentuated depressive and emotional symptoms and functional impairment, young people with comorbid Depression and ODD were more globally impaired in school, the home and in relationships with other people.

There is some evidence to suggest that ODD, and not CD, may best explain the comorbidity between Disruptive Behaviour Disorders and depression (Burke & Loeber, 2010). Burke, Loeber, Lahey, and Rathouz (2005) examined the developmental comorbidity among boys in a clinic-referred sample and found that over ten assessment waves, ODD symptoms predicted depression symptoms in the following year. They reported that after accounting for ODD and for negative life events, CD symptoms were no longer predictive of Depression. Rowe, Maughan, and Eley (2006) also found that after accounting for negative life events, delinquency was not predictive of Depression, whereas oppositionality showed a direct relationship with later Depression, and ODD in adolescence was found to predict Depression in adulthood (Copeland, Shanahan, Costello, & Angold, 2009).

Burke, Hipwell, and Loeber (2010) looked at the dimensions of ODD and their prediction of later CD and Depression in a community sample of 2451 girls over a five-year period. They reported that dimensions of negative affect, oppositional behaviour and antagonistic behaviour were found within ODD symptoms, and negative affect predicted later Depression (oppositional and antagonistic behaviour predicted CD). They noted that: ODD typically has its onset before CD and Depression; changes in ODD

symptoms predicted changes in symptoms of CD and Depression from one year to the next; and ODD in childhood and adolescence predicted Depression in adulthood. Emerging evidence suggests that there are affective and behavioural dimensions of ODD symptoms, and those affective ODD symptoms best predicted later Depression requiring further longitudinal research (Burke & Loeber, 2010).

### 2.6.4 Disruptive Mood Dysregulation Disorder (DMDD) and ODD

DSM-5 prohibits a diagnosis of ODD being given if criteria for Disruptive Mood Dysregulation Disorder (DMDD) are also met. There is overlap between the two disorders. DMDD was created to capture the same symptoms as the irritability dimension of ODD, with some qualifiers regarding the frequency or severity with which they manifest (Burke, Derella, & Johnston, 2018). The core feature of DMDD is described in DSM-5 as "chronic, severe persistent irritability", which has two manifestations, "frequent temper outbursts" and "chronic, persistently irritable or angry mood" (APA, 2013). The DSM-5 states that most children who meet criteria for DMDD will also meet criteria for ODD (APA, 2013), which is consistent with the findings of Copeland, Angold, Costello, and Egger (2013). The reverse has not been found, with researchers reporting 15% to 58% of those meeting criteria for ODD also meeting criteria for DMDD (APA, 2013): Copeland et al. (2013) found that 23 to 38% of those with ODD would meet criteria for DMDD; Axelson et al. (2012) reported that 58% of those with ODD would meet criteria for DMDD.

Some researchers report that there is inadequate literature to substantiate the diagnosis of DMDD and to differentiate it from ODD, given there is such high co-occurrence between them (Axelson et al., 2012; Dougherty et al., 2016; Freeman, Youngstrom, Youngstrom, & Findling, 2016). Burke et al. (2014) and Mayes et al. (2015) suggest there is a difficulty in distinguishing between DMDD and the irritability dimension of ODD (Burke et al., 2018). Mayes et al. (2016) conducted a study on 665 children aged six to twelve years of age in the general population. They found that more than 90% with DMDD had ODD, and 65% of ODD children had symptoms of DMDD. They also found that DMDD did not increase the risk of comorbid psychopathology (Anxiety, Depression, CD, ADHD) independently of ODD.

Further longitudinal research in this area is required. However, if researchers are prohibited from diagnosing ODD, if DMDD criteria are met, it is not clear how they would be expected to represent the prognostic risks associated with the behavioural symptoms of ODD in future studies (Burke et al., 2018).

### 2.6.5 Anxiety Disorders and ODD

Anxiety is commonly associated with ODD and can develop at an early preschool age (Boylan, Vaillancourt, Boyle, & Szatmari, 2007; Nock et al., 2007; Steiner et al., 2007). Angold et al. (1999) found that up to 14% of persons with ODD also have Anxiety. In a more recent study, Nock et al. (2007) found at least 50% of participants with ODD had comorbid Anxiety or Depression.

Some researchers have looked at how different factors interact for young people with ODD and co-occurring Anxiety. Martín, Granero, Domènech, and Ezpeleta (2017) undertook a study to identify factors related to comorbid ODD and Anxiety disorders. They assessed 622 children longitudinally at three and five years of age. They found that high levels of the child's negative affectivity and the mother's aggressive behaviour, and high scores in the father's psychopathology measurements were related to the presence of comorbid ODD and Anxiety at three years of age. High scores in approach-positive anticipation, fears in boys and aggressive behaviour, and low scores for smiling and laughter were predictive of comorbidity at five years of age. These findings suggest that parental responses, parent psychopathology and temperament traits may be factors in explaining longitudinal ODD and Anxiety comorbidity. Further longitudinal research in this area is required.

### 2.6.6 Autism Spectrum Disorder (ASD) and ODD

There is limited research in the area of ODD and ASD. However, comorbidity between the two disorders is common (Gadow, Devincent, & Drabick, 2008; Guttmann-Steinmetz, Gadow, & Devincent, 2009). Some researchers have questionned whether ODD behaviour among children with ASD reflects the same disorder seen in children without ASD (Kaat & Lecavalier, 2013). While others have reported that comorbid ODD does appear to present differently from other ASD related behavioural difficulties and that ASD symptomotology is not uniquely associated with ODD behaviours, suggesting

that the co-occurrence of the two disorders is a true comorbid presentation (Gadow et al., 2008; Mandy, Roughan, & Skuse, 2014).

Salazar et al. (2015) studied a clinical sample of 101 young children (ages 4.5-9.8 years) with ASD and found that boys were 3.9 times more likely to also have ODD. Mandy et al. (2014) found that in mainstream school, over half of the children with ASD met the criteria for ODD as well. A comorbid diagnosis of ASD and ODD has been found to be associated with a more severe clinical presentation and poorer general functioning (Gadow et al., 2008; Guttmann-Steinmetz et al., 2009). Further longitudinal research in this area is required.

### 2.6.7 Summary

Young people with ODD have significantly higher rates of comorbid psychiatric disorders. When ODD is comorbid with other conditions young people present with a more severe clinical presentation and increased functional impairments (at school, home and in relationships). In the last decade, research has put to rest questions about the validity of the ODD disorder and the issue of whether it is meaningfully distinct from CD. Research is now separating ODD and CD, making it easier to now understand the differences between them. Research has become focused on the dimensions within ODD of chronic irritability and oppositional behaviour, but further investigation into these areas is required. High comorbidity has also been found between ODD and Mood Disorders, Anxiety and ASD. There is limited research in the area of DMDD and ODD, with DSM-5 prohibiting a diagnosis of ODD being given if criteria for DMDD are also met. Given changes to diagnosic categories in DSM-5, further research into ODD and its comobidities is required, especially in the areas of CD, DMDD and ASD. Future systemic research should also explore how different factors interact for young people with ODD, co-occuring disorders, temperament and parental factors, such as parenting skill and psychopathology.

## 2.7 NATURAL HISTORY OF ODD

### 2.7.1 Introduction

Two developmental pathways to ODD have been explored: early onset and the late starter pathway. The early onset pathway has been described as having a more detrimental trajectory than the late starter pathway. Historically, ODD has been recongised as a significant predictor of CD and Anti-Social Personality Disorder. However, this has recently being challenged, especially for different genders. Typically ODD has been seen as a disorder of childhood, although recent studies have been exploring ODD into adulthood. This section will look at the natural history of ODD in further detail.

### 2.7.2 Natural History of ODD

Developmental theorists have suggested that a child on the "early starter/ early onset pathway" (where the behaviours develop before preschool years) has a two-to-threefold risk of developing conduct problems in adolescence or an Antisocial Personality Disorder in adulthood (Connor, 2002; Fraser & Wray, 2008; Loeber & Farrington, 2000; Tremblay, 2000). Research suggests that the primary developmental pathway for serious CD in adolescence and adulthood appears to be established during the preschool period (Webster-Stratton & Reid, 2018). Lavigne et al. (2001) reported that preschool children with ODD were also more likely to exhibit other additional disorders several years later including ADHD, Anxiety, or Mood Disorders.

The late starter pathway involves little oppositional behaviour during childhood, but the behaviours become more observable during adolescence. This pathway is often associated with family stresses (unemployment, divorce), which may cause disruption in the family management practices, monitoring of the young person's activities and their involvement with an inappropriate peer group. The prognosis for the late starter pathway is generally more positive (Webster-Stratton & Reid, 2018).

ODD has long been recognized as a significant predictor of CD (Burke et al., 2002; Loeber et al., 1995), although there now is significant evidence to challenge this. Rowe et al. (2010) conducted a longitudinal study, of both girls and boys, aged 9-21 years

finding that nearly half of all the children with a diagnosis of CD did not previously meet criteria for a diagnosis of ODD. They found that an ODD diagnosis was found to be a risk factor for the development of CD in boys, but a diagnosis of ODD in girls was not significantly predictive of later development of CD. Burke, Waldman, et al. (2010) combined data from three longitudinal data sets and showed that a substantive proportion of youths with adolescent-onset CD did not have an ODD diagnosis in their childhood. These studies suggest that ODD and CD are two distinct disorders, and challenge the previous hierarchical approach (Ghosh et al., 2017).

There has been scant research investigating which children go on to develop CD (Steiner et al., 2007). With the introduction of the two distinct features of ODD in DSM-5 - the *affective symptoms* (irritability, temper tantrums, and resentful attitude) and the *behavioural features* (defiance, vindictiveness and arguing) - researchers have become interested in exploring the differences. Kolko and Pardini (2010) conducted a longitudinal study of clinic-based children (n=177) aged six to eleven years over a three-year period. They found that the irritability facet of ODD was associated with internalizing problems, whereas defiance (for example, vindictiveness) predicted CD symptoms and externalizing problems. Similar findings were also reported by Rowe et al. (2010) in a much larger sample, showing irritability symptoms predicted Anxiety disorders. Mikolajewski, Taylor, and Iacono (2017) found the 'irritable' dimension predicted internalizing problems, and the 'headstrong/hurtful' dimension predicted substance-use disorder symptoms. The irritability dimension has been found to 'not' predict Bipolar Disorder, CD, ADHD and substance abuse (Vidal-Ribas, Brotman, Valdivieso, Leibenluft, & Stringaris, 2016). Stringaris and Goodman (2009) go further and propose that there are at least three dimensions of ODD. In a cross-sectional general population study, they found an additional dimension, 'hurtful', and found it to be associated with callous traits and aggressive symptoms. These studies suggest that underlying subtypes or dimensions of ODD are being recognised, but the actual number are still uncertain (Ghosh et al., 2017) and their relationship to developmental pathways are only just beginning to be explored.

Historically, ODD has been characterised as a disorder of childhood. However, there is a growing body of evidence that suggests that prevalence rates of the disorder are stable into late adolescence and trajectories of symptoms persist into young adulthood (Boylan et al., 2007; Burke et al., 2014; Leadbeater et al., 2012; Maughan, Rowe, et al.,

2004). ODD was found to remit in roughly half of the ODD population studied after 3 years by Biederman, Monuteaux, et al. (2008) and Bunte, Schoemaker, Hessen, van der Heijden, and Matthys (2014). Nock et al. (2007) looked at lifetime prevalence of ODD in a nationally representative sample of adults (n=3,199) and estimated it to be 10.2% (males 11.2%; females 9.2%). Burke et al. (2014) after controlling for parent reported symptoms of ADHD, CD, Depression and Anxiety, found ODD symptoms from childhood through adolescence predicted: poorer functioning at age 24 years with peers; poorer romantic relationships; a poorer paternal relationship; and not having anybody who would provide a recommendation for a job. These results are consistent with others that have found that ODD is associated across the lifespan with functional impairments that go beyond noncompliance in the parent-child relationship (Burke et al., 2014; Leadbeater et al., 2012).

ODD has also been seen as a predictor of other psychopathology in adulthood: Depression (Burke, 2012; Stringaris et al., 2009); Borderline Personality Disorder (Burke & Stepp, 2012); and some evidence suggesting Antisocial Personality Disorder (Langbehn, Cadoret, Yates, Troughton, & Stewart, 1998), while others finding no association with Antisocial Personality Disorder (Burke, Waldman, et al., 2010; Leadbeater et al., 2012; Loeber, Burke, & Lahey, 2002). Further longitudinal research is required looking at the underlying dimensions of ODD and different developmental pathways.

### 2.7.3 Summary

Current literature is beginning to explore the pathways of ODD in more specific detail. Challenges have been made to assumptions that ODD leads to CD and Antisocial Personality Disorder and is primarily a disorder of childhood. Researchers are exploring different features of ODD (affective symptoms and behavioural features) to help understand their association with different trajectories for young people with ODD: affective symptoms (irritability) are associated with internalizing problems; whereas behaviour (defiance) predict more externalizing problems. Further longitudinal research is required to explore the different pathways for ODD. This has been made easier now ODD is seen as a separate disorder and is recognised as requiring research independently of the other Disruptive Behaviour Disorders. Research is required to look at which

children are at risk of developing CD and Antisocial Personality Disorder, compared to those who are at risk of developing Depressive and/or Anxiety disorders. Further longitudinal research is required to look at gender differences and the lifelong functional impairments of adult ODD.

## 2.8 KEY TREATMENT APPROACHES FOR ODD

### 2.8.1 Introduction

Given the substantial impact of ODD on individuals, families and society, it is important that children receive treatments that have measurable and long term functional impacts (Kaminski & Claussen, 2017). Both internationally, and in Australia, the current policy direction is towards earlier intervention and prevention for ODD, because it addresses early risk factors, before secondary risk factors have developed (Webster-Stratton & Reid, 2018). The aim is to counteract risk factors (individual child factors, building more effective family functioning, disengaging adolescents from deviant peer networks, enhancing their school involvement) and strengthen protective factors, thereby helping to prevent a developmental trajectory towards aggressive and violent behaviours (McCart & Sheidow, 2016; Webster-Stratton & Reid, 2018). This section will outline the early intervention and prevention literature, as well as evidence-based treatment approaches for young people with a diagnosis of ODD.

The American Academy of Child and Adolescent Psychiatry (AACAP) published psychiatric practice parameters for ODD in 2007, recommending the "minimal standard" of delivering a parent intervention based on one of seven empirically tested behavioural parent therapies, with medication potentially helpful as an adjunct treatment (Steiner et al., 2007). While parent group programs are recommended, especially for children under eight years of age, older children might benefit more from multicomponent treatment approaches (Ghosh et al., 2017; Knapp, Chait, Pappadopulos, Crystal, & Jensen, 2012; Scotto Rosato et al., 2012). Typical approaches that have been used to treat ODD include: Parent Management Training; individual psychotherapy; family therapy; Cognitive Behavioural Therapy; and social skills training (Steiner et al., 2007). Treatments should be specifically tailored to the individual child, and different treatment techniques are applied for pre-schoolers, school aged children, and adolescents (Kaminski & Claussen,

2017; Steiner et al., 2007). There is evidence that early intervention is preferable and prevents progression into more problematic conditions. Clinicians should also consider and treat comorbid psychiatric conditions when treating ODD (Connolly & Bernstein, 2007).

The American Academy of Child and Adolescent Psychiatry (Connolly & Bernstein, 2007) points out that regardless of what treatment you use, successful treatment of ODD requires two important therapeutic conditions: the establishment of therapeutic alliances with the child and family; and being culturally sensitive. Establishing therapeutic alliances with both the child and the family is not an easy task, for children are usually brought in by parents and often are not in agreement about the nature of the problems and frequently lack the motivation to resolve them. Clinicians also need to constructively raise issues regarding the effectiveness of parenting without making the parent feel accused or judged. Cultural issues need to be actively considered in ODD treatment due to the substantial body of literature on different standards of obedience and parenting in ethnic subgroups. It is important to be mindful that there may be a mismatch in the family/clinician backgrounds especially regarding the topic of discipline (Portes, Dunham, & Williams, 1986; Steiner et al., 2007; Walker-Barnes & Mason, 2001). To help establish these two therapeutic conditions, it is important for clinicians to clarify their role as helpers, empathize with both the child and the family, and be willing to explore cultural differences with the families they are working with.

### 2.8.2 Early Intervention and Preventative Treatments for ODD

Many researchers in the field of ODD believe that prevention is the key in ODD intervention (Burke et al., 2002; Connor, 2002; Hinshaw & Anderson, 1996; Rutter et al., 1999). Prevention intervention has included: consultation; home visits to high-risk families; parent management strategies; psychoeducational packages targeting social skills, conflict resolution, and anger management; cognitive interventions; vocational training; academic skills training; and school-based interventions. Interventions vary according to the young person's age and can be delivered in schools, clinics, and other community locations.

#### *2.8.2.1 Preschool Children*

For preschool children, there is evidence that programs such as Head Start (a comprehensive early childhood education, health, nutrition, and parent involvement service for low-income children and their families) have assisted in improving academic scores, reducing the probability of a child repeating a grade and preventing future delinquency (Connor, 2002; Deming, 2009; Garces, Thomas, & Currie, 2002).

#### *2.8.2.2 School Aged Children*

For school aged children, parent management strategies are the most empirically supported programs. Two programs have been comprehensively evaluated: The Triple P-Positive Parenting Program; and The Incredible Years Program. Sanders, Turner, and Markie-Dadds (2002) developed the Triple P- Positive Parenting Program, combining universal and indicated intervention levels (targeting parenting skills and other family adversity factors such as marital conflict, Depression and high levels of parenting stress). It has undergone a number of effectiveness trials finding it reduced disruptive behaviours in children, which were maintained overtime, with further improvements in long-term follow-up (Connell, Sanders, & Markie-Dadds, 1997; de Graaf, Speetjens, Smit, de Wolff, & Tavecchio, 2008; Sanders, 1999). The Incredible Years (BASIC) program, developed by Webster-Stratton and colleagues, has been well supported in a series of randomized control trials. The Incredible Years parent component works on improving parent management skills, increasing family support networks, and strengthening home-school bonds. The child component teaches skills related to empathy, problem-solving, anger control, friendship skills, communication skills and school skills. The BASIC program was shown to improve: parental confidence; increase positive parenting strategies; reduce harsh and coercive discipline; and reduce child conduct problems, compared to wait-list control groups (Webster-Stratton & Reid, 2018). Webster-Stratton and Hammond (1997) studied families of children with early-onset conduct problems (4 to 8 years of age) and randomly assigned them to one of four conditions: a parent training treatment group (PT); a child training group (CT); a combined child and parent training group (CT + PT); or a waiting-list control group (CON). They found that all three treatment groups had resulted in significant improvements in comparison with typically developing control participants. CT and CT + PT children demonstrated significant improvements in problem solving, as well as conflict management skills. PT and CT +

PT parents and children had significantly more positive interactions, compared with CT parents and children. They found at follow-up, child conduct problems at home had significantly lessened over time, and at one-year follow-up the CT + PT group produced the most significant improvements in child behaviour. Other researchers have replicated this work and also found a positive impact of the program (Scott, Spender, Doolan, Jacobs, & Aspland, 2001; Spaccarelli, Cotler, & Penman, 1992; Taylor, Schmidt, Pepler, & Hodgins, 1998). Burke et al. (2002) reported that other helpful preventive interventions for this age group include psychoeducational packages targeting social skills, conflict resolution, and anger management strategies.

#### *2.8.2.3 Adolescent Prevention / Early Intervention*

For prevention in adolescence, psychoeducation packages including cognitive interventions skills training, vocational training and academic preparations appear to reduce ODD symptoms (Burke et al., 2002; Connor, 2002; Rutter et al., 1999). The Adolescent Transitions Program (ATP; (Dishion & Kavanaugh, 2002)) has developed a three-level blended prevention model that incorporates universal (developing a positive behaviour support plan that bridges home and school for a young person and provides parent education), selected (for at risk adolescents, undertaking a family interview, providing home visits, and providing parenting strategies), and indicated strategies (providing direct professional support to parents including: face-to-face intervention; school monitoring; parents groups; behavioural family therapy; case management; and referral services). ATP matches intervention intensity to family and youth need. Evaluation of the program reports that it can significantly contribute to the reduction of adolescent problem behaviour and substance use (Dishion, Kavanagh, Schneiger, Nelson, & Kaufman, 2002).

#### *2.8.2.4 School-Based Prevention Programs for ODD*

Several studies have reported that school-based prevention programs are helpful in reducing ODD symptoms (Burke et al., 2002; Loeber & Farrington, 2000). Some focus on students and are designed to: reduce and prevent aggression and violence directly; or to develop social, affective, and/or academic competence in order to reduce risk (Greene, Ablon, Goring, Fazio, & Morse, 2004). Others focus on changing the school ecology

rather than modifying the behaviour of individual young people directly (Greenberg, Domitrovich, & Bumbarger, 2001).

Kellam and colleagues conducted several large-scale prevention trials to test the impact of two classroom interventions: The Good Behaviour Game (GBG) and Mastery Learning (ML) (Kellam et al., 2011). The GBG is a classroom management program promoting positive social relationships and adherence to classroom rules, by dividing the class into teams that compete for rewards. ML is an educational program requiring a majority of students in the class to master specific reading and maths skills before being introduced to more complex skills in the same areas. Both programs were found to be effective when implemented for one or two years. GBG was shown to reduce aggressive behaviour and the ML to improve basic learning skills, thereby positively affecting risk factors for developing behavioural disorders (Kellam, Ling, Merisca, Brown, & Ialongo, 1998; Tingstrom, Sterling-Turner, & Wilczynski, 2006). Ialongo et al. (1999) found that in early adolescence youth who participated in the combined program were less likely than typically developing control youth to meet diagnostic criteria for CD, to have received mental health services, and to have been suspended from school.

Olweus (1993) also developed a school based national program aimed at reducing school bullying. It is a comprehensive intervention targeting change at the school level (through policy and other initiatives); at the classroom level (through the establishment of rules to reduce bullying and opportunities to discuss alternative behaviour to antisocial behaviour); and at the individual and family level (by promoting communication between home and school, and discussing incidents of bullying with perpetrators, victims, and their families). Results showed that the program reduced incidents of bullying by half or more in elementary and middle school children, and improved student satisfaction with school; and reduce antisocial conduct in general (fighting, stealing, vandalism, and truancy) (Olweus, 1994; Olweus, 1996).

Felner, Ginter, and Primavera (1982) developed a program called the School Transitional Environment Project (STEP) designed to facilitate transition to a new school environment for students at risk of failing academically and dropping out. STEP has two major components involving restructuring the physical and social environment (students remain in small groups for their homeroom periods and academic subjects) and restructuring the role of the homeroom teacher (homeroom teacher's act as administrators

and guidance counsellors). Homeroom teachers helped students to adjust to their new school environment, facilitate communication between home and school, and monitor students' social adjustment and academic progress throughout the year. Students in STEP were found to have reduced stress and Anxiety, and better social and academic adjustment compared to the typically developing control group. Over a four-year follow-up period, STEP students continued to be better adjusted socially and academically and had much lower dropout rates than non-participants (Feiner et al., 1994).

The Incredible Years Program also has a school-based component (Webster-Stratton et al., 2001; Webster-Stratton, Reid, & Hammond, 2004). The goals of the teacher training program are to: improve teachers' classroom management skills (proactive teaching approaches and effective discipline); increase teachers' use of academic, persistence, social and emotional coaching with students; strengthen teacher-student bonding; increase teachers' ability to teach social skills, anger management, and problem-solving skills in the classroom; improve home-school collaboration, behaviour planning, and parent-teacher bonding; and build teachers' support networks. Kirkhaug et al. (2016) assessed the teacher program and found significant decreases in conduct problems and other problem behaviour among the intervention children, including noncompliant and off task behaviour, compared to the typically developing control group. Reinke, Herman, and Dong (2018) conducted a randomised control trial (RCT) to evaluate its efficacy on student social behaviour and academic outcomes with 105 teachers from kindergarten to third grade. They found that it reduced student emotional dysregulation, increased prosocial behaviour, and increased social competence. Students also showed significant improvements in social and academic competence.

### 2.8.3 Pharmacological Treatments for ODD

Pharmacological treatment is not generally used for young people with ODD alone, and the effectiveness of medication treatment is not well established. Medication is usually prescribed for aggression management or coexisting disorders that the young person may have, such as ADHD, Anxiety or Depression. Psychopharmacological treatment is found to be most effective when paired with another treatment plan, such as individual intervention or multimodal intervention (Burke et al., 2002).

Research in behavioural disorders has again commonly focused on a diagnosis of CD with aggression management difficulties, not ODD for pharmacological treatment. Clinical experience has suggested that mood stabilizers, the typical and atypical antipsychotics, clonidine, and the stimulant medications may be useful for the treatment of children and adolescents with CD (Burke et al., 2002). Most studies that have used RCTs to assess the effectiveness of medication use in CD have been undertaken with inpatient young people with aggression difficulties. Two studies conducted RCTs to assess the effectiveness of lithium with placebo and reported that at therapeutic levels, lithium was efficacious and safe for the short-term treatment of aggressive inpatient children and adolescents with CD (Malone, Delaney, Luebbert, Cater, & Campbell, 2000; Sweeney, Forness, Kavale, & Levitt, 1997). However, a third study did not find a difference between lithium and the placebo (Rifkin et al., 1997). Other medications that have been found useful were haloperidol (although not well tolerated) (Campbell et al., 1984); molindone; and thioridazine (Greenhill, Solomon, Pleak, & Ambrosini, 1985), whereas carbamazepine (Cueva et al., 1996) was not found to be useful. In outpatient settings, studies have found the following medications helpful for young people with CD in the management of aggressive behaviours: risperidone (Findling et al., 2000); methylphenidate (Klein et al., 1997); and clonidine (Connor, 2002; Hunt, Minderaa, & Cohen, 1985). Other studies have suggested that lithium, the typical and atypical antipsychotics, and the stimulant medications maybe useful for the treatment of youth with CD. However, these findings cannot be generalized because they included small samples of aggressive youths, only looked at treatment completers, and did not consider the presence of comorbid disorders (Burke et al., 2002).

Pringsheim, Hirsch, Gardner, and Gorman (2015) did a systematic review and meta-analysis of RCTs of antipsychotic medications, lithium, and anticonvulsant medications for aggression and conduct problems in youth with ADHD, ODD, and CD. They found that there was moderate quality evidence that risperidone has a moderate-to-large effect on conduct problems and aggression in youth with sub-average IQ and ODD, CD, or Disruptive Behaviour Disorder not otherwise specified, with and without ADHD. They also found high quality evidence that risperidone had a moderate effect on disruptive and aggressive behaviour in youth with average IQ and ODD or CD, with and without ADHD. They outlined the evidence supporting the use of haloperidol, thioridazine, quetiapine, and lithium in aggressive youth with CD is of low to very low quality, and the

evidence supporting the use of divalproex or carbamazepine in aggressive youth with ODD or CD is of low to very low quality. They concluded that except for risperidone, the evidence to support the use of antipsychotic medications and mood stabilizers is of low quality for the treatment of ODD/CD.

There is high comorbidity between ODD and ADHD. A few studies have reported the positive effects of stimulant medications or atomoxetine in the treatment of ODD associated with ADHD (Turgay, 2009). Chapter one outlines the pharmacological treatments for ADHD. Newcorn, Spencer, Biederman, Milton, and Michelson (2005) examined the responses of ODD symptoms to atomoxetine finding that youth with ADHD and comorbid ODD showed statistically significant improvement in ADHD, ODD, and quality of life measures. Treatment response was similar in youths with and without ODD, except the comorbid group showed improvement compared with placebo. They concluded that atomoxetine treatment improved ADHD and ODD symptoms in youths with ADHD and ODD, although the comorbid group may require higher doses.

Challenges arise when prescribing medication to this cohort. Firstly, side effects (sedation, cognitive effects, hypotension, extrapyramidal symptoms, tardive dyskinesias, and obesity) should be weighed against the possible benefits of the pharmacological treatment (Burke et al., 2002). Secondly, given the high risk for substance abuse in youths with Disruptive Behaviour Disorders, caution should be taken when prescribing stimulant medications to this population (Burke et al., 2002). Thirdly, adherence to treatment should be monitored because it is usually low in youths with Disruptive Behaviour Disorders (Burke et al., 2002).

### 2.8.4 Non-Pharmaceutical Treatments for ODD

There is considerable research investigating the effects of psychosocial treatments for young people with ODD. More recent systematic reviews separate treatment for disruptive young people in early/middle childhood (ages 5-11 years) and adolescents (ages 12-19 years), as they require slightly different targets for intervention. This is due to: the type of behaviour problems exhibited by young people varies significantly with age (mild oppositional behaviours are common in early childhood, whereas aggression becomes more prevalent in adolescence) (Kaminski & Claussen, 2017); the risk factors for behaviour problems are present in multiple domains (for example, individual, family,

peer and school); and those domains have different levels of influence over time, with peer influences and school experiences becoming more relevant over time (Kaminski & Claussen, 2017; Patterson et al., 1989).

#### *2.8.4.1 Early / Middle Childhood*

There have been three main reviews of the literature for evidence based psychosocial treatments for Disruptive Behaviour Disorders in children: Brestan and Eyberg (1998); Eyberg, Nelson, and Boggs (2008); and Kaminski and Claussen (2017). The two earlier reviews focused on a brand name program approach. They both reported that *the Parent Management Training Oregon model,* (designed to train parents to manage their child's externalizing behaviour through behaviour contracts and contingencies from problem behaviour) reached the level of a well-established treatment. *The Incredible Years Parent Training Program* (Webster-Stratton et al., 2004) was listed as probably efficacious, along with 14 other treatment programs. These programs have core components in common: they promote parent competencies and strengthen families by increasing positive parenting, parent-child attachment, and parenting self-efficacy.

This is consistent with the recommendations set out in the American Academy of Child and Adolescent Psychiatry (2007) Practice Parameter for the Assessment and Treatment of Children and Adolescents With Oppositional Defiant Disorder (Steiner et al., 2007) which included Incredible Years (Webster-Stratton & Reid, 2003); Triple P-Positive Parenting Program (Hoath & Sanders, 2002); Parent-Child Interactional Therapy (Brinkmeyer & Eyberg, 2003); Helping the Noncompliant Child: Parenting and Family Skills Program (McMahon & Forehand, 2003); COPE (Cunningham, 2006); Defiant Children (Barkley, 1997); and The Adolescent Transitions Program (ATP) (Dishion & Kavanaugh, 2002).

The more recent review of evidence-based programs for five to twelve-year-old children, Kaminski and Claussen (2017) took a more generic treatment approach and wanted to identify the common factors across effective treatment programs. Two treatment families achieved the highest distinction of well-established treatments: *group parent behaviour therapy***;** and *individual parent behaviour therapy with child participation.* They described parent behaviour therapy as being a treatment approach that taught parents to be more effective behavioural reinforcers. This type of therapy

typically focused on strengthening the parent-child relationship so that the child is more motivated to behave in a way that the parents wants; and providing the parents with more effective child behaviour management strategies (Hanf, 1969, cited in Kaminski & Claussen, 2017). The strategies are based on social learning principles: positive reinforcement (attending to desirable behaviours); withholding positive reinforcement (planned ignoring or time-out for undesirable behaviours); and relationship enhancing strategies including providing the child with positive attention, engaging in joint activities, and communication skills (Kaminski & Claussen, 2017). They also found the following treatment approaches were probably efficacious: group parent behaviour therapy and group child behaviour therapy; group parent behaviour therapy with child participation and family problem-solving training; individual parent behaviour therapy; group parent focused therapy; group child-centred play therapy; and individual child-centred play therapy (Kaminski & Claussen, 2017).

#### *2.8.4.2 Adolescents*

McCart and Sheidow (2016) completed an evidence-based review of treatments for youth aged thirteen years and older. They reported that well-established treatments included ones that combined behavioural therapy, Cognitive-Behavioural Therapy (CBT) and Family Therapy. Two examples of such programs are: *Multisystemic Therapy* (MST) (Henggeler, Schoenwald, Borduin, Rowland, & Cunningham, 2009) and *Treatment Foster Care Oregon* (TFCO; formerly Multidimensional Treatment Foster Care (MTFC), (Chamberlain, 2003). MST is an intensive family and community based intervention (twenty-four hours a day, seven days a week) for children and young people aged eleven to seventeen which includes: increasing the skills and resources of parents and carers to manage their young person's behaviour more effectively; improving family relationships; assisting young people to have positive friendships; increasing the young person's involvement with education and/or training; helping the young person participate in positive activities; and creating a support network of extended family, neighbours and friends to help the parents and carers maintain the changes. TFCO is an alternative to residential treatment, to help prevent incarceration among high-risk adolescents and youth. The goal of TFCO is to provide young people with skills, resources, supervision, and structure necessary to reduce delinquency and promote more prosocial behaviours. The program relies on the involvement of trained foster families, therapists, and case

managers, with a goal to return youth to their biological or adoptive families who are also involved throughout the process. Each young person receives an individualized program that emphasizes behavioural skills training, discipline and positive reinforcement, and positive affirming relationships. McCart and Sheidow (2016) listed probably efficacious treatments as Cognitive-Behavioural Therapy (CBT) based ones, such as: The Aggression Replacement Training and Positive Peer Culture, Equipping Youth to Help One Another (Gibbs, Potter, & Goldstein, 1995); Solution-Focused Group Program (Shin, 2009); and Functional Family Therapy (Sexton & Alexander, 2002).

#### *2.8.4.3 Limitations of Psychosocial Interventions for ODD*

Although there is strong evidence that psycho-social inventions are successful in the treatment of ODD, several limitations of these treatments have also been noted.

Firstly, a substantial number of parents who receive parent management training do not fully comply with implementation or drop out of treatment altogether (Greene, Ablon, & Goring, 2003; Prinz & Miller, 1994). Estimates of attrition rates for children referred to outpatient clinics for conduct problems range from 40 to 60% (Kazdin, 1995). Frey and Snow (2005) review of the literature states the attrition from parent training groups is consistently at or above 40%, even when financial incentives are offered and when childcare, refreshments, and transport are provided. In the Fast Track study parents missed an average of 43.7% of parent sessions (Orrell-Valente, Pinderhughes, Valente, & Laird, 1999) and in the Head Start Program 40% of mothers in the randomly assigned intervention group attended less than three parent training sessions and were considered "non-attenders" in statistical analyses (Reid, Webster-Stratton, & Baydar, 2004). Most studies examining the efficacy of parent management training present data only for those who remained in treatment, rather than those who began treatment.

Although large effects from behavioural treatments have been show in studies children may not achieve full normalization of functioning (Pfiffner & Haack, 2014). Studies have produced statistically significant changes in oppositional behaviour, but very few studies have reported clinically significant changes (for example, daily improvement in each child's functioning across school and home domains) (Kazdin, 1997). Data has shown that a significant percentage of children are not functioning within the normal range when such treatment is completed (Dishion & Patterson, 1992) and 30

to 40% of those children remaining in treatment continued to have behaviour problems in the clinical range at follow-up (Kazdin, 1993). There is also limited data on the long-term sustainability of treatment effects (Furlong et al., 2012). Although treatment effects can persist for at least several months after treatment ends, beyond that time periodic treatment may be necessary (Pfiffner & Haack, 2014). Little is known about the processes or mechanisms through which parent management training improves outcomes (Kazdin, 2010).

Questions of generalizability have also been raised. Outcomes from behavioural interventions tend to be setting specific, so that behavioural interventions implemented in one setting (for example, home) often do not generalize to another setting (for example, school), without behavioural intervention in that setting as well (Owens, Murphy, Richerson, Girio, & Himawan, 2008). A lot of the research for Disruptive Behaviour Disorders is undertaken under research conditions and not implemented and evaluated in "the real world". Researchers have noted that it is important to see if these programs are transportable to community treatment settings (La Greca, Silverman, & Lochman, 2009). There is also not a high take up of evidence-based programs in the routine clinical care of young people (Ng & Weisz, 2016; Weisz et al., 2012).

Therefore, some researchers are advocating for alternative treatments that better address the needs of these children and parents to be developed and evaluated (Greene et al., 2004). Programs other than parent management training may be better suited for: parents with significant psychopathology (such as, anger management problems, ADHD, Depression and substance abuse); limited cognitive capacity; those in highly conflicted marital/partner relationships; or those parents unlikely or unable to attend weekly sessions (Chronis, Chacko, Fabiano, Wymbs, & Pelham, 2004; Pfiffner & Haack, 2014).

#### *2.8.4.4 Summary*

Effective treatment of ODD is important given the substantial impact it has on individuals, families and society. The current policy direction is towards early intervention / prevention for ODD. Numerous treatment programs have been developed at universal, targeted and indicated levels of intervention. When looking at evidence based interventions for young people diagnosed with ODD, both pharmocological treatment and psychosocial treatments have been explored. Pharmocological treatment

generally target aggression and comorbid conditions that the young person has. Systematic reviews of evidence based psychosocial (non-pharmacological) treatments have been published focusing on early/middle childhood (ages 5-11 years) and adolescence (ages 12-19 years), as they require slightly different targets for intervention. Overall psycho-social interventions (parent behaviour therapy; child behaviour therapy; and school based interventions) have been found effective in changing behaviour and in the treatment of ODD. As a young person increases in age, multisystem interventions are required for more effective outcomes. Researchers have suggested that future interventions need to take into account child temperament dimensions, cognitive and/or academic deficits that the young person may have, family functioning, parental relationship dysfunction and parental psychopathology, as these may impact on the outcomes of the intervention (Greene et al., 2004). More research is needed regarding the type, timing, and dosage of specific programs for different populations.

## 2.9 CHAPTER CONCLUSIONS AND FUTURE RESEARCH DIRECTIONS

Defiant and rule breaking young people are not a new occurrence. Records dating back to the 1600's describe such occurrences. ODD as a formal diagnosis however, was first introduced in the DSM in 1980. The current version of DSM-5 defines ODD as a frequent and persistent pattern of behaviour of angry/irritable mood, argumentative/defiant behaviour, and/or vindictiveness. ODD is a relatively common childhood disorder, with point prevalence ranging from 1% - 11%, with an average prevalence estimate of around 3.3%. ODD typically develops and becomes apparent in preschool years, often before the age of eight years. Researchers have identified that prevalence rates can vary due to child characteristics and reporting factors.

There are no laboratory tests or diagnositic markers specific for ODD and it is diagnosed after a comprehensive assessment by a qualified professional. Making a diagnosis can be challenging as it is not uncommon for preschool children and adolescents to display challenging behaviour, suggesting the professional must be experienced in understanding behaviour of a typically developing young person and ensure that there is a persistent pattern of behaviour that is causing significant distress to the family and

system of the young person. Gender differences have also been noted and need consideration when making a diagnosis.

Most researchers agree that the causes of ODD are multifactorial, arising out of a complex mix of risk and protective factors, present in multiple domains (for example, individual, family, peer and school) which exert different levels of influence over time. Research in the area of ODD has been limited due to ODD generally being researched under the heading of Disruptive Behaviour Disorders, including CD. Further longitudinal research is required specifically looking at ODD. Research looking at the important child functional factors such as temperament, IQ, Working Memory, academic achievement, emotional regulation and social competence, is limited for ODD and shows mixed results. Family factors, such as poor family functioning, low parental relationship functioning, poor parenting practices and familial psychopathology have been noted as risk factors for developing Disruptive Behaviour Disorders. However, further systematic longitudinal research is required to look at the risk factors for ODD independently to CD. Current emphasis is being placed on interactive models of the risk factors contribution to the development of ODD and further research is need to look at how ODD is multifactorially determined. Also further research is needed in the area of protective factors and how they ameliorate the impact of risk factors.

Young people with ODD have significantly higher rates of comorbid psychiatric disorders. Researchers often report that ODD co-occurs with ADHD, CD, Mood Disorders (MD and PDD), Anxiety Disorder and ASD. More recently research has focused on ODD as a distinct psychiatric disorder, independent of CD. Generally, research has found that when ODD is comorbid with another disorder the young person presents with a more severe clinical presentation, highlighting the importance of a comprehensive assessment to ensure comorbid disorders are not missed and are treated. Further systematic longitudinal research is also required to explore the different pathways for ODD development. This is easier now ODD is viewed as a separate disorder and is recognised as requiring research independently of the other Disruptive Behaviour Disorders. Longitudinal research is required to look at which children are at risk of developing CD and Antisocial Personality Disorder, compared to those who are at risk of developing Depression and Anxiety. Futher longitudinal research is also required in adult ODD and the impact on functioning of these individuals.

Given the impact of ODD on the individual, family and society, it is important that children receive treatments that have measurable and long term functional impacts. Both internationally and in Australia, the curent policy direction is towards early intervention and prevention for ODD, because it addresses early risk factors, before secondary risk factors have developed. Pharmacological treatments generally are used to target comorbid conditions, and are not generally used for young people with ODD alone, as the effectiveness of medication treatment is not well established. Non-pharmacological treatments include: parent mangement training (to promote parent competencies and strengthen families by increasing postive parenting, parent-child attachement and parenting self-efficacy); Cognitive Behavioual Therapy; and Multisystemic Therapy. Most researchers recommend intervention taking a whole of systems approach (individual, parents, school and community) for the treatment of ODD.

# CHAPTER 3: Introduction to Comorbid Attention Deficit Hyperactivity disorder (ADHD) and Oppositional Defiant Disorder (ODD)

## 3.1 Introduction to Comorbid Attention Deficit Hyperactivity Disorder (ADHD) and Oppositional Defiant Disorder (ODD)

Attention Deficit Hyperactivity Disorder (ADHD) is consistently shown to have a greater than chance occurrence with several other mental conditions including Anxiety Disorders, Depressive disorders and other Disruptive Behaviour Disorders, which include CD and ODD (Angold et al., 1999; Biederman, Faraone, Milberger, Jetton, et al., 1996). ADHD comorbidity has shown to be associated with higher delinquency rates (Connor & Doerfler, 2008), increased risk for alcohol problems and drug use (Molina & Pelham, 2003) and higher costs for treating comorbid ADHD (Foster et al., 2007; Guevara, Lozano, Wickizer, Mell, & Gephart, 2001; Jensen et al., 2005).

One trajectory for children with ADHD is to develop comorbid ODD and/or CD; known to have a greater than chance association (Angold et al., 1999). Children with combined ADHD and ODD and or CD have been found to show more severe ADHD symptoms (Hurtig et al., 2007), greater social dysfunction (Kuhne et al., 1997), greater academic underachievement (Faraone et al., 1991), more behaviour problems as well as a greater general impairment in daily functioning (Crawford, Kaplan, & Dewey, 2006), lower self-esteem (Kuhne et al., 1997) and higher rates of antisocial behaviours, including juvenile and adult criminal conviction outcomes. There are limited studies that have looked at ODD and CD separately in young people with ADHD. Therefore, it is important to understand the risk and protective factors for young people with ADHD in relation to developing ODD.

ADHD and ODD can cause significant impairment in family, school, and social functioning, and prognosis is even worse when these disorders co-occur (Biederman, Petty, Monuteaux, et al., 2008; Carlson et al., 1997). Children or adolescents who have ADHD and ODD are typically more aggressive, have more negative behavioural symptoms of ODD, underachieve more severely in the academic domain, and are rejected at higher rates by peers (American Academy of Child and Adolescent Psychiatry, 2009; Carlson et al., 1997; Steiner et al., 2007). Biederman, Petty, Monuteaux, et al. (2008) found that girls with co-morbid ADHD and ODD were more likely to be placed in a special classroom and have higher rates of suspensions. Girls tend to have more severe

and persistent behavioural problems and are more likely to have additional co-morbid Mood Disorders and higher risk of substance use and abuse (Connor, Steeber, & McBurnett, 2010; Steiner et al., 2007). Steiner et al. (2007) hypothesized that ADHD facilitates the onset of ODD and can hasten the transition to CD. Gadow et al. (2007) found that adults who attended mental health clinics with co-morbid ADHD and ODD had higher rates of unemployment, while community adults with this comorbidity had lower rates of marriage.

A number of studies have found that the hyperactivity/impulsivity aspect of ADHD and ODD are highly correlated with each other (Burns & Walsh, 2002; Gomez, Harvey, Quick, Scharer, & Harris, 1999). Burns and Walsh (2002) through a longitudinal study reported that the hyperactivity/impulsivity factor, and not the inattentive factor of ADHD, influenced the development of ODD behaviour. They also found impulsivity levels were a risk factor, but child adaptability and interpersonal skills served as protective factors, predicting with 85% accuracy which children had a co-occurring diagnosis of ODD and which did not. Therefore, it is important to separate the subtypes of ADHD in future research.

This chapter will look at comorbid ADHD and ODD: the epidemiology of ADHD and ODD; key clinical characteristics of ADHD and ODD; the diagnostic process for comorbid ADHD and ODD; the diagnostic challenges; key aetiological risk factors (child characteristics and psychosocial factors); key comorbid conditions; natural history of ADHD and ODD; and key treatment approaches (pharmacological and non-pharmacological). Future research directions will be discussed and the rationale, aims and hypotheses for the current study will be presented.

## 3.2 EPIDEMIOLOGY OF ADHD AND ODD

Historically diagnoses involving disruptive or externalizing behaviours (CD and ODD) and ADHD are often reported together in the literature, making it difficult to get a clear picture of ADHD and ODD without CD. ADHD and ODD are among the most common childhood disorders and frequently co-occur, each affecting 10% of children and adolescents (Bloom et al., 2013; Nock et al., 2007). High rates of comorbidity between

ADHD and ODD have been noted: 14 to 50% (Angold et al., 1999; Nock et al., 2007; Waschbusch & King, 2006). In adults with ADHD, ODD is diagnosable in 43% and is present predominantly when ADHD co-occurs with high levels of emotional dysregulation, which is associated with greater impairment (Reimherr et al., 2010).

### 3.2.1 Age and Comorbid ADHD and ODD

While ADHD usually first presents in childhood and persists into adulthood (Biederman & Faraone, 2005), ODD can emerge during different developmental stages and may or may not persist over time (Nock et al., 2007). This is relevant to the development of comorbid ADHD and ODD, as which developmental stage it emerges in may have a different pattern of symptom course, related impairment, and prognosis (Hudec & Mikami, 2018).

In *early childhood* the point prevalence rate for comorbid ADHD and ODD has been estimated at 2.4% (Lavigne, Lebailly, Hopkins, Gouze, & Binns, 2009). Although a low prevalence rate, these children are at significant risk for future comorbid ADHD and ODD (Bunte et al., 2014; Harvey, Breaux, & Lugo-Candelas, 2016).

Across all of *childhood* comorbid ADHD and ODD has been estimated at 48% (Pardini & Fite, 2010). This comorbidity is associated with a high risk for poor future outcomes, through being more likely to experience continued difficulties with impulsivity, hyperactivity and mental disorders in adolescence and adulthood (Biederman et al., 2011; Harpold et al., 2007).

During *adolescence*, there appears to be three pathways: (1) children with ADHD may develop and then discontinue ODD in adolescence, they tend to display less severe ODD symptoms and typically have better outcomes (Moffitt, Caspi, Harrington, & Milne, 2002); (2) children with ADHD develop ODD in adolescence and it persists over time, generally leading to a poorer prognosis later (Barker, Oliver, & Maughan, 2010); and (3) children with ADHD already have comorbid ODD in childhood that persists into adolescence. This latter group generally displays the most severe behavioural problems and has the poorest prognosis of all three groups (Waschbusch, 2002).

There are very few studies looking at ADHD and ODD in *adulthood*, and it is rare for ADHD and ODD to develop for the first time in adulthood (Reimherr et al., 2010).

Generally adult ADHD and ODD is associated with a significant impairment in interpersonal and occupational functioning, and may progress to aggressive or criminal behaviour (Young et al., 2015).

In summary, future research needs to examine ADHD and comorbid ODD separately in childhood and adolescence, using longitudinal study designs.

### 3.2.2 Gender and Comorbid ADHD and ODD

Most studies have focused on boys; only a few studies have looked at comorbid ADHD and ODD in girls. Different point prevalence rates have consistently been noted between boys and girls (Biederman, Petty, Monuteaux, et al., 2008; Carlson et al., 1997; Munkvold et al., 2011; Trepat & Ezpeleta, 2011). Carlson et al. (1997) found equivalent rates of comorbidity across gender, while some researchers found the risk was higher for girls (Costello et al., 2003; Loeber & Keenan, 1994). Costello et al. (2003) found a much stronger link between ODD and ADHD among girls than boys, with the longitudinal link of ADHD predicting the onset of ODD found only in girls.

Biederman, Petty, Monuteaux, et al. (2008) found that girls with ADHD were more likely than typically developing girls to have ODD. They compared girls who had ADHD alone, to those with ADHD and ODD at baseline, over a five-year period. They found that ADHD and ODD significantly increased the risk for ODD and Major Depression at follow-up. Both groups of girls with ADHD had an increased risk for CD and Bipolar Disorder at follow-up (Biederman, Petty, Monuteaux, et al., 2008).

In summary, future research examining ADHD and comorbid ODD must assess the impact of gender.

### 3.2.3 Ethnicity and Comorbid ADHD and ODD

Co-morbidity rates tend to be similar across geographical locations. Rates recorded for the various countries include: 35% for the United States (Nock et al., 2007); 29.5% for England (Maughan, Rowe, et al., 2004); and 25% for China (Qu, Jiang, Zhang, Wang, & Guo, 2015). However, rates were lower in Turkey, 9.4%, (Ercan, Bilaç, Uysal Özaslan, & Rohde, 2015) and in a Danish sample, 16.5%, (Jensen & Steinhausen, 2015) possibly due to variability in sample populations, study methods, differences in diagnostic

methodology and cultural norms (Hudec & Mikami, 2018), which will be discussed further under diagnostic challenges.

### 3.2.4 Summary

Comorbidity between ADHD and ODD is high and can cause significant impairment in family, school, and social functioning. Prevalence rates, symptoms and prognosis can differ depending on gender and developmental stage. Due to limited studies addressing ADHD and ODD comorbidity (without CD) further systematic research in this area is needed that takes these factors into account.

## 3.3 Key Clinical Characteristics for Comorbid ADHD and ODD

### 3.3.1 Introduction

Assessing comorbid conditions can be complex. However, an accurate assessment is important because the co-occurring disorders of ADHD and ODD can lead to a greater risk for poor outcomes and have implications for treatment. The following section will cover the diagnostic process for comorbid ADHD and ODD; the diagnostic manuals relevant to ADHD and ODD; and the diagnostic challenges for comorbid ADHD and ODD.

### 3.3.2 Diagnostic Process for Comorbid ADHD and ODD

There are no diagnostic laboratory tests or diagnostic biological markers specific for ADHD and ODD. Both are diagnosed after a comprehensive assessment of gathering information from multiple informants, over one or more sessions with a qualified professional (Paediatrician, Psychiatrist, Psychologist, or Mental Health Clinician). Information is collected in the same way that has already been described in the previous two chapters: a clinical interview with history taking and screening for associated comorbidities; observation; examination; and a variety of assessment tools (checklists, behaviour questionnaires, and/or rating scales). It is important to gain an adequate understanding of contributing factors (such as, family functioning, marital relationship

functioning, parent management skills, and parental psychopathology). Other evaluations, such as tests of intelligence (for example, Wechsler Intelligence Scale) and educational achievement (for example, Wide Range Achievement Test – 4) are also often helpful to rule out intellectual deficits and learning disabilities (Fletcher et al., 2005).

### 3.3.3 Diagnostic Manuals

The diagnostic criteria for ODD and ADHD are outlined in the Diagnostic and Statistical Manual of Mental Disorders, fifth edition (DSM-5) (APA, 2013) and the International Classification of Diseases, tenth revision (ICD-10) (World Health Organization, 2004). The history and criteria for both disorders have already been discussed in Chapter 1 and Chapter 2. Typically, in Australia DSM-5 criteria are used for mental disorder diagnoses and will be used in this study.

### 3.3.4 Diagnostic Challenges for Comorbid ADHD and ODD

The overlap between ADHD and ODD symptoms and behaviour, and developmental considerations can cause diagnostic challenges (Hudec & Mikami, 2018): for example, a young person who doesn't seem to listen or follow through on instructions (ADHD) may also be seen as defying or refusing to comply with requests (ODD); leaving their seat (ADHD) might be seen as defying rules (ODD); or interrupting others (ADHD) could be classified as deliberately annoying others (ODD). Therefore, a comprehensive assessment including multi-informants' assessment ratings, clinical interview and observation is recommended. Barkley (2010) suggested that the high rate of comorbidity and increasing focus on irritability symptoms in ODD has led some to suggest that the overlap reflects a core dimension of ADHD symptomology, such as emotional impulsivity, rather than emotion dysregulation rooted in ODD. Burke et al. (2018) argue that measures of emotional impulsivity included in the DSM-5 irritability dimension of ODD (touchy, angry, and lose temper) will make it difficult to attribute emotional impulsivity entirely to ADHD, and ADHD and comorbid ODD will need to be considered more, especially in the adult population.

Assessing ADHD and ODD during developmental stages is challenging because some of the diagnostic behaviour could be age appropriate (Hudec & Mikami, 2018). Professionals making a diagnosis of ADHD, ODD, or comorbid ADHD and ODD must

have a comprehensive understanding of the normative presentations of these behaviours in the different age groups in terms of frequency and duration to assess whether both disorders reach clinical severity (Matthys & Powell, 2018). Using rating scales with appropriately normed reference groups can assist with this challenge. However, it is important for multiple informants to provide information and complete rating scales, due to parent and teacher ratings often differing and the impact and influence of parents own psychopathology on their severity rating (as previously discussed).

The difference in the prevalence rates of ADHD and ODD in girls, compared to boys, maybe due to girls being underdiagnosed (Biederman, 2005). As mentioned in chapter 2, females tend to express their antisocial behaviour via relational aggression, as compared to males who are more physically aggressive (Abikoff et al., 2002). Due to this, symptoms are less likely to be noticed and treated, more likely to escalate (Abikoff et al., 2002) and then only recognised later in adolescence. Therefore, girls referred with ADHD need a comprehensive assessment of comorbid disorders so ODD is not missed in childhood, due to its different behavioural expression compared to boys.

Finally, Lachman and Bass (1985) described the halo effect as a type of immediate judgement discrepancy, or cognitive bias, where a person making an initial assessment of another person will assume ambiguous information based upon concrete information. If a young person has a diagnosis of ADHD then a reporter may inflate and endorse symptoms of ODD (Hartung et al., 2010), resulting in an inaccurate diagnosis of comorbid ADHD and ODD. Therefore, it is important to not only rely on information provided by checklists or behaviour rating scales: a comprehensive interview and observations are important to add to the diagnostic process.

### 3.3.5 Summary

The criteria for ADHD and ODD are set out in DSM-5 and ICD-10. Typically, DSM-5 diagnostic criteria are used in Australia and will be used in this study. When making a diagnosis of comorbid ADHD and ODD an experienced clinician needs to undertake a comprehensive assessment, using multiple informants for behavioural questionnaires/checklists, clinical interview, and observation. Many factors must be kept in mind during the assessment, including: the overlap of symptoms between the disorders; the developmental stage of the young person; gender differences; and the halo effect. It

is important not to miss diagnosing comorbid ADHD and ODD because of the increased risk of poorer outcomes for those with this comorbidity and its treatment implications.

## 3.4 Key Aetiological Risk Factors for Comorbid ADHD and ODD

### 3.4.1 Introduction

Relatively few studies have investigated risk factors for comorbid ODD in ADHD. The high rates of comorbidity that exist between ADHD and ODD may be attributable to similar aetiological factors across the disorders, increasing the likelihood of both disorders emerging for children and adolescents experiencing certain high risk life circumstances (Hudec & Mikami, 2018). Reported risk factors for ODD, which are arguably also implicated in the development of comorbid ODD, include both risk factors overlapping with those reported for ADHD and risk factors specific for ODD (Noordermeer et al., 2017). Specific risk factors for co morbid ODD in ADHD, compared with ADHD, include low levels of parental education, parenting styles (harsh or inconsistent parenting), deviant peer affiliation, parental psychopathology and exposure to poor family functioning (Deault, 2010; Loeber et al., 2000; Modesto-Lowe et al., 2008; Richards et al., 2015). This section will explore the aetiological risk factors for comorbid ADHD and ODD covering: child functional factors (temperament, cognitive functioning, executive functioning and Working Memory, academic achievement, emotional regulation ability, and social competency); and key psychosocial components (family functioning, parental relationship functioning, parenting skill, and parental psychopathology).

### 3.4.2 Individual Child Characteristics

#### *3.4.2.1 Temperament and Comorbid ADHD and ODD*

Very few studies have looked at the temperament of young people with comorbid ADHD and ODD. Three temperament characteristics have been found to be common in young people with comorbid ADHD and ODD: high levels of activity; decreased levels of task persistence; and decreased levels of self-directedness. Stringaris et al. (2010)

explored the temperamental antecedents to ODD and its comorbidities in a longitudinal study. They found ODD and ADHD were predicted by high levels of activity, whereas ODD alone was found to be preceded by temperaments of both emotionality and activity. Kim et al. (2010) found that children and adolescents with both ODD and ADHD showed decreased levels of task persistence and self-directedness compared to those with ODD only. They found those with ODD only showed temperament and character profiles of high novelty seeking, low self-directedness, and low co-cooperativeness. Further longitudinal research in this area could explore the temperamental antecedents to comorbid ADHD and ODD compared to ADHD or ODD alone, which may aid understanding of their different pathways and assist with targeting specific treatment for individuals with ADHD and ODD.

#### *3.4.2.2 Cognitive Functioning (IQ Scores) and Comorbid ADHD and ODD*

A few studies have assessed if ADHD and ODD/CD is associated with greater intellectual impairment than ADHD or ODD/CD alone. Most studies found evidence of greater impairment in groups with ADHD (either alone or with ODD/CD) relative to groups with ODD/CD alone (Anderson, Williams, McGee, & Silva, 1989; Taylor, Chadwick, Heptinstall, & Danckaerts, 1996). However, it is hard to know if this would be the case if ADHD and ODD was looked at without CD due to limited studies in this area. When compared to typically developing control participants, research has found ADHD, CD/ODD, and ADHD+CD/ODD had lower FSIQ scores, suggesting that those with comorbid ADHD and ODD/CD did not have greater intellectual impairments than those with ADHD or ODD/CD alone (Dolan & Lennox, 2013; Jensen et al., 2001; Matthys, Cuperus, & Van Engeland, 1999). Hogan (1999) argued that if a young person with comorbid ADHD and ODD had intellectual deficits it could be better explained by ADHD. Hogan (1999) reviewed 27 studies examining the relationship between ODD/CD and IQ and when ADHD symptoms were controlled for, 73% of the studies did not find ODD/CD to be uniquely linked to lower IQ.

A few studies have reported mixed findings for comorbid ADHD and ODD (without CD). Speltz, DeKlyen, Calderon, Greenberg, and Fisher (1999) studied a group of preschool clinic referred boys with ADHD and ODD finding significant deficits in

Verbal IQ, language abilities and Executive Functions compared to a matched comparison group. Jin and Wang (2004) found that ADHD children, with or without ODD had similar cognitive functioning in FSIQ, VCI and PCI, and both groups were significantly lower than the typically developing control group. In contrast, Skogan et al. (2014) found no difference on IQ measures between those with ADHD, ADHD+ODD, ODD and typically developing control participants. Forssman et al. (2012) investigated 120 adolescents from the general population with ADHD and ODD and found that cognitive functioning was associated with ADHD behaviours, rather than ODD.

Overall, studies examining young people with comorbid ADHD and ODD/CD suggest that they do not appear to have greater deficits than those with ADHD alone. Undoubtedly, more longitudinal research is required looking at more specific aspects of cognitive functioning and comorbid ADHD and ODD to understand if particular IQ difficulties: increase the risk for ADHD and ODD behaviours; ADHD and ODD behaviours increase the risk for IQ difficulties; or if ADHD better explains the risk for IQ difficulties than ODD.

#### *3.4.2.3 Executive Functioning, Especially Working Memory and Comorbid ADHD and ODD*

Executive dysfunction has been found in both ADHD and ODD samples, but it is unclear what the implications for comorbid ADHD and ODD are. Little research has been conducted to determine the specific associations between Executive Function components such as Working Memory and comorbid ADHD and ODD. It is unclear if these disorders are exclusive of each other, with only ADHD affecting Executive Functioning, or if both ADHD and ODD affect Executive Functions in different ways, both separately and together (Sarkis, Sarkis, Marshall, & Archer, 2005). Some studies have found that Executive Functioning impairments are more pronounced in those with comorbid ADHD and ODD, than ADHD alone (Dolan & Lennox, 2013), while others suggest the deficits are unique to ADHD (Ezpeleta & Granero, 2015; Oosterlaan et al., 2005).

Qian, Shuai, Cao, Chan, and Wang (2010) examined the Executive Functioning of children with ADHD, ADHD + ODD and a typically developing control group. They used a performance based Stroop and Trail-making test (measuring ability to sequence;

ability to shift cognitive set; and processing speed), and a BRIEF parent report of Behavioural Regulation (three scales: Inhibit; Shift; and Emotional Control), Metacognition (five scales: Initiate; Working Memory; Plan/Organise; Organisation of Materials; and Monitor) and a Global Executive Composite score for everyday life scenarios. Both the ADHD and ADHD + ODD groups performed worse than the typically developing control participants in the Stroop and Trail-making test and the BRIEF. The ADHD + ODD group were rated worse than the ADHD group on the BRIEF, but the two groups showed no significant difference in the Stroop and Trail-making test. These findings suggest children with ADHD display executive dysfunction in performance-based tests and everyday life scenarios. However, children with ADHD + ODD showed more severe executive dysfunction in everyday life scenarios than those with ADHD alone.

Oosterlaan et al. (2005) assessed three components of Executive Function (verbal fluency, Working Memory, and planning) in 99 children age 6-12 years, finding comorbidity did not increase the severity of executive dysfunction. They reported that independent of ODD/CD, ADHD was associated with deficits in planning and Working Memory, but not in verbal fluency. They found no Executive Functioning deficits associated with ODD/CD. They reported the presence of comorbid ADHD accounts for the Executive Functioning deficits in children with ADHD+ODD/CD, suggesting the deficits are unique to ADHD. Sarkis et al. (2005) also found no relationship between comorbid ADHD diagnosis and Executive Function, suggesting comorbidity was not found to add additional impairment to the Executive Functioning of ADHD children.

The findings have been more consistent when looking specifically at Working Memory for comorbid ADHD and ODD. Skogan et al. (2014) investigated the association between symptoms of ADHD and/or ODD and two core Executive Functions (inhibition and Working Memory) from a population-based sample of 1045 three-year-old children. Children with comorbid ADHD and ODD performed at a significantly lower level than typically developing children on the Executive Functioning measures. Rhodes et al. (2012) assessed memory functioning boys with ADHD only, ADHD and ODD, ODD only and typically developing boys. All three clinical groups demonstrated impaired memory performance. Boys with ODD and ADHD and ODD (but not ADHD alone) performed poorly on Verbal Memory tasks, whilst all three clinical groups showed impaired performance on Spatial Memory tasks. All three clinical groups performed

poorly on the storage and Central Executive Working Memory factors and the Long Term Memory factor. Lin and Gau (2017) also found young people with ADHD and ODD had deficits in Verbal Memory and response inhibition, compared to those with ADHD only. They also found that young people with ADHD, and ADHD comorbid with ODD, had impaired Spatial Working Memory and Short-Term Memory. Taken together, these studies suggest that Verbal Memory difficulties are more closely associated with ODD than ADHD symptoms and that ADHD and ODD represents a true comorbidity. Future longitudinal research is needed to systematically investigate the association of specific aspects of Verbal and Visual Spatial Working Memory with ADHD and comorbid ODD compared to ADHD alone and ODD alone.

#### *3.4.2.4 Academic Achievement and Comorbid ADHD and ODD*

There are limited studies investigating the impact of comorbid ADHD and ODD on academic achievement. Findings from these studies are mixed. Some studies suggest that: comorbid ADHD and ODD is associated with worse academic outcomes; others suggest that ADHD only is associated with worse academic achievement; while some show no difference. Sayal, Washbrook, and Propper (2015) investigated the impact of ADHD and ODD behaviours at age 7 to 16 years on academic achievement in a population-based sample. They found that both ADHD and ODD behaviours were associated with worse academic outcomes at age 16. Liu et al. (2017) examined the impact of ADHD and ODD/CD on various school functions. They assessed youth with ADHD+ODD/CD, ADHD only and a typically developing control group. The results showed that youth with ADHD had poorer performance across different domains of school functioning. Youth with ADHD+ODD/CD had more behavioural problems, but a similar academic performance to those with ADHD only. They found that ADHD impaired academic performance for those youth with ADHD with or without ODD, and that comorbid ODD/CD may specifically contribute to behavioural difficulties in youth with ADHD. Clark et al. (2002) assessed four groups of adolescents (ADHD only, comorbid ADHD and ODD/CD, ODD/CD only, and a normal community control group) finding the ADHD group had the poorest reading score (using the WRAT3) and this was significantly lower than that of the ODD/CD and control groups. The WRAT3 reading score of the comorbid ADHD+ODD/CD group was also lower than that of the control group. However, standard scores on the WRAT3 reading subtest were within the average

limits for each group (ranging from the 37th percentile for the ADHD group to the 65th percentile for the control group), suggesting no reading difficulties were found for any of the groups. Further systematic research is required to understand the outcome on specific academic literacy and numeracy performance measures for ADHD and ODD compared to ADHD alone and ODD alone.

#### *3.4.2.5 Emotional Regulation and Comorbid ADHD and ODD*

It is not uncommon for children with ADHD and ODD to display emotional regulation difficulties (see chapter 1 and chapter 2). However, there is limited research investigating emotional regulation deficits in comorbid ADHD and ODD (without CD). Some research suggests that emotion regulation problems may be more linked to ODD than ADHD symptoms (Schoorl, van Rijn, de Wied, van Goozen, & Swaab, 2016; Sobanski et al., 2010). Bunford et al. (2018) found that young people with ADHD did have emotion dysregulation difficulties. However, it did not differ as a function of ADHD subtype or comorbid ODD. Schoorl et al. (2016) examined emotional regulation difficulties in boys with ODD/CD and ADHD to understand to what extent emotional regulation in the ODD/CD group was correlated with severity of ADHD traits. They found emotional decision making within the ODD/CD group was not related to variation in attention deficit traits. Further longitudinal research in ADHD and ODD is required to understand the impact of the comorbidity on emotional regulation of children and adolescents.

#### *3.4.2.6 Social Problems and Comorbid ADHD and ODD*

Research commonly reports that children and adolescents with ADHD frequently experience difficulties in social functioning (Becker, Langberg, Vaughn, & Epstein, 2012) and their peer relationships (Becker, Luebbe, et al., 2012; Gadow & Nolan, 2002). When ADHD is comorbid with ODD it has been reported to be associated with greater social impairment than ADHD alone (Becker, Langberg, et al., 2012). Kuhne et al. (1997) found greater social adjustment difficulties / poorer social functioning skills in the ADHD+ODD group than in the ADHD only group, on both parent and teacher ratings. In a laboratory study of social problem-solving, boys with Disruptive Behaviour Disorders and ADHD had problems encoding social cues and generating responses,

whereas boys with Disruptive Behaviour Disorders more often selected aggressive responses to problems and felt more confident in their ability to carry out an aggressive response (Matthys et al., 1999). Çakaloz, Akay, and Günay (2007) assessed children with ADHD, ADHD and ODD and typically developing control participants, using both parent and teacher reports. They found that both the ADHD and ADHD+ODD groups had significantly more peer-relationship difficulties compared to the control participants in social problems. Frankel and Feinberg (2002) assessed children referred for friendship problems with ADHD, ADHD and ODD, ODD and children referred with neither disorder. Both parents and teachers were given questionnaires to rate. They found the diagnosis of ADHD was associated with increases in classroom disruption and decreased resistance to provocation by peers. While the diagnosis of ODD was associated with increased hostility towards peers, decreased resistance to provocation by peers and decreased respect for adults. Hence, both groups express deficits in social functioning, although they may differ in their presentation.

Becker, Luebbe, et al. (2012) undertook a literature review to look at the impact of ADHD and co-occurring externalizing symptoms (ODD and CD) on the peer functioning, friendship, and social skills/ competence domains of young people with ADHD. They found some studies that suggested co-occurring externalizing problems exacerbate peer problems. However, the majority of studies found co-occurring externalizing problems had no effect on peer problems, consistently in the friendship domain of peer functioning. Studies were mixed in terms of demonstrating either no effect or exacerbation across social skills/competence and peer status. Therefore, they concluded that the problems in peer relations, friendship and social skill/competence domains among youth with ADHD cannot be wholly attributable to co-occurring behaviour problems (Becker, Luebbe, et al., 2012).

Becker, Luebbe, et al. (2012) also found mixed results depending upon who the reporter was and where the sample was taken from. When social skills/competence were assessed with parent report measures no difference was found (Booster, Dupaul, Eiraldi, & Power, 2012; Jensen et al., 2001; Ostrander, Crystal, & August, 2006). When teacher reports of social skills/competence were used significant differences were found with comorbid ADHD and ODD, showing a decrease in social skills/competence (Booster et al., 2012; Carlson et al., 1997; Mikami & Lorenzi, 2011). They also found a difference between samples of comorbid ADHD+ODD and peer problems: treatment-referred

children with ADHD and ODD showing no significant difference (Jensen et al., 2001) compared to school-based samples reporting a reduction in social skills/competence (Carlson et al., 1997; Mikami & Lorenzi, 2011). Becker, Luebbe, et al. (2012) speculate that the treatment referred children with ADHD did not show teacher-reported differences because these children were already experiencing significant social impairments in the school setting that were not affected by comorbid ODD. They suggested that longitudinal studies are required to examine the possibility that co-occurring ODD symptoms in childhood may have long term adverse effects on friendship functioning, which is not readily identified in cross-sectional studies.

#### *3.4.2.7 Summary*

There are high rates of comorbidity that exist between ADHD and ODD, which may be attributable to similar aetiological factors across the disorders. However, it has been difficult to gain a clear picture of the risk factors for comorbid ADHD and ODD and the impact of the comorbidity on young people's functioning due to limited research in the area. Some temperament differences have been found in comorbid ADHD and ODD (high levels of temperament activity, decreased levels of task persistence and decreased levels of self-directedness) compared to ODD only. Young people with comorbid ADHD and ODD do not appear to have greater cognitive deficits than those with ADHD alone, some researchers suggesting that the deficits are better explained by ADHD alone. Executive Functioning studies generally have found mixed results for comorbid ADHD and ODD, some suggesting Executive Functioning impairments are more pronounced in those with comorbid ADHD and ODD than ADHD alone, while others have found no difference. In contrast, Working Memory studies broadly have shown more consistent results, suggesting that young people with comorbid ADHD and ODD performed significantly lower for both long term and short-term memory tasks, compared to ADHD alone, or typically developing control participants. The impact of comorbid ADHD and ODD on academic achievement has also produced varied results, some studies suggesting comorbidity is associated with worse academic outcomes, while others have found that ADHD only is associated with worse academic achievement, and moreover others have reported no difference. Most studies in comorbid ADHD and ODD, in relation to emotional regulation and social competence, have shown no greater difficulties have occurred due to the comorbidity. Future longitudinal systematic studies are required to

look at the key aetiological child risk factors for young people with ADHD and ODD (separately from CD) and assess if the comorbidity increases the impact on more specific and carefully defined aspects of functioning in these areas.

### 3.4.3 Key Psychosocial Risk Factors for Comorbid ADHD and ODD

#### *3.4.3.1 Introduction*

The effects of ADHD are not confined to the child or adolescent alone but can also affect their family context. ADHD and ODD share similar environment risk factors. Studies of families of children with ADHD and comorbid conduct problems have found associations between disturbances in family, disrupted parent-child relationships, increased levels of parenting stress and parental psychopathology (Johnston & Mash, 2001). This section will cover the key psychosocial risk factors for comorbid ADHD and ODD (including family functioning, parental relationship, parenting skill and parental psychopathology).

#### *3.4.3.2 Family Functioning and Comorbid ADHD and ODD*

There are very few studies that have investigated the relationship with comorbid ADHD and ODD and overall family functioning. Barkley, Anastopoulos, Guevremont, and Fletcher (1992) compared two groups of clinic-referred adolescents (one with ADHD, one with ADHD and ODD) to a community typically developing control group. They found that adolescents with ADHD and ODD reported more conflicts at home, while their mothers reported greater negative interactions, greater personal distress and less satisfaction in their marriages than in the control group.

Generally, studies have looked at ADHD with comorbid ODD and CD collapsed into one Disruptive Behaviour Disorder construct. Kiliç and Sener (2005) investigated family functioning in children with comorbid ADHD and ODD/CD, finding these families scored high at the level of unhealthy functioning in the roles and behaviour control subscales of the McMaster Family Assessment Device (FAD) for family functioning (The FAD has an overall general functioning subscale and includes 6 dimensions of family functioning: problem solving; communication; behaviour control; affective involvement; affective responsiveness; and roles). İmren, Arman, Gümüstas, Yulaf, and Çakıcı (2013) also using the FAD, assessed children and adolescents with

ADHD alone and comorbid ADHD and ODD/CD. They found ADHD families scored high at the level of unhealthy functioning in the problem solving, roles, affective involvement, general functioning, and behaviour control subscales of the FAD. Problem solving behaviour and general functioning were significantly poorer than control families and they had more difficulties in area of roles. When ADHD was comorbid with ODD or CD all areas of family functioning, as measured by the FAD, were scored high at the level of unhealthy functioning. Additionally, general functioning and affective responsiveness were significantly poorer than ADHD without ODD or CD comorbidity. Thus, overall, the families of children and adolescents with ADHD and ADHD comorbid with ODD or CD, had poorer family functioning in most of the subscales of the FAD.

Some researchers have argued that the family functioning problems of young people with ADHD appear to mostly originate from Disruptive Behaviour Disorder features such as rule-breaking, aggressive and antisocial behaviours (Barkley et al., 1992). They report that most of the negativity in the family interactions associated with ADHD are related to comorbid disruptive behaviours in the children (Barkley, 1998; Barkley et al., 1992). Thus, comorbidity of ADHD with Disruptive Behaviour Disorders may increase the levels and types of family-related impairments of psychosocial functioning.

Harvey et al. (2011) argue that there is some evidence that ADHD may place children at risk for developing ODD symptoms by disrupting family functioning. They found that children with ADHD manifest less improvement in ODD symptoms and poorer family functioning during preschool years than children without ADHD. They did not find evidence that the effects of family adversity were greater among children with comorbid ADHD and ODD.

The majority of research designs that explore the relationship between family psychosocial variables and oppositional or conduct problems in children with ADHD use a cross-sectional design to contrast the patterns of associations among families (Deault, 2010) which are generally less useful in separating cohort effects and studying ‘causal’ risk factor association (Kelsey, Petitti, & King, 1998). Longitudinal research designs are more powerful in being able to achieve these outcomes. They need to be applied in future to understand family functioning deficits more fully in young people ADHD with comorbid ODD.

#### *3.4.3.3 Parental Relationship and Comorbid ADHD and ODD*

Literature has focused on the impact of marital interaction on children's developmental outcomes (Cummings & Davies, 2010; Liu & Wang, 2015) and the influence of children's mental health issues on marital interaction (Kouros, Merrilees, & Cummings, 2008; Lin et al., 2017). Studies have shown a link between the severity of child behaviour and interparental discord/interparental conflict and report greater discord among parents of youths with ADHD and comorbid ODD or CD, than among parents of youths with ADHD alone or without ADHD (Barkley et al., 1992; Wymbs, Pelham, Gnagy, & Molina, 2008).

Wymbs, Pelham, Gnagy, et al. (2008) studied adolescents with comorbid ADHD and ODD also reporting a higher prevalence of frequent and unresolved conflict between their parents, than in families of adolescents with ADHD only. Wymbs, Pelham, Molina, et al. (2008) found that both parent (lower maternal education level, higher paternal education and antisocial behaviour) and child ODD/CD behaviour ratings each increased the risk of divorce between parents of youths diagnosed with ADHD in childhood. They found that the severity of disruptive behaviour (ODD/CD) increased the risk of marital dissolution, which was also consistent with the findings of Wymbs, Pelham, Molina, et al. (2008).

However, there are no studies examining parental relationship dysfunction of young people with comorbid ADHD and ODD (separately from CD). Longitudinal studies are needed to further our understanding of the risks and impact of young people with comorbid ADHD and ODD on parental relationship dysfunction and vice versa.

#### *3.4.3.4 Parenting Skill and Comorbid ADHD and ODD*

There are limited studies that have investigated parenting skill and comorbid ADHD and ODD (without CD). Studies have investigated the dynamics of parent-child interactions that are associated with comorbid ADHD and ODD (not CD). Edwards, Barkley, Laneri, Fletcher, and Metevia (2001) found that parents of adolescent boys with comorbid ADHD and ODD reported poorer communication with their adolescents, including greater parent-child conflict, anger intensity and aggressive tactics compared to parents of typically developing adolescents. They also reported that the adolescents with comorbid ADHD and ODD reported higher levels of anger in family conflicts and poorer

communication with both parents. Kashdan et al. (2004) found ODD symptoms, not ADHD symptoms, predicted less positive parenting, including a lack of warmth and positive parental involvement, as well as reports of more negative discipline strategies and parental intrusiveness. Richards et al. (2015) found that maternal warmth was positively associated with prosocial behaviour and negatively with antisocial behaviour, while maternal criticism was positively associated with antisocial behaviour and negatively with prosocial behaviour.

Freitag et al. (2012) looked at the psychosocial risk factors that influenced symptom severity and psychiatric comorbidity in children with ADHD. They assessed nine psychosocial risk factor domains: (1) abnormal intra-familial relationship patterns; (2) psychiatric disorder or disability in parent or sibling; (3) distorted communication within the family; (4) abnormal parenting; (5) parental separation/divorce or institutional education outside the family; (6) acute life events independent of the child; (7) movement, migration and discrimination; (8) adverse school circumstances; and (9) acute life events due to child psychopathology. They found that familial risk factors, comprising abnormal intra-familial relationships (such as lack of warmth toward the child, scapegoating, child maltreatment, constant arguments and fights between adults, difficult communication pattern, parental separation/divorce and isolated family) showed a trend towards increasing risk for comorbid CD. They also found current adverse parenting conditions (such as lack of supervision and teaching/education, and aversive constraints on the child) were risk factors for comorbid CD in children with ADHD, independent of child hyperactive-impulsive symptoms or parental ADHD. Similarly, Pfiffner et al. (2005) found negative, inconsistent and detached parenting was a risk factor for comorbid CD in children with ADHD. Hurtig et al. (2007) also reported a lack of parental interest in adolescents' activities increased the risk for comorbid ADHD and conduct problems in adolescents. However, it is unclear if these findings are true for comorbid ADHD and ODD, or just ADHD and CD.

Most studies focussed on parent skills have examined their association with boys' externalizing behaviours. In contrast, Peris and Hinshaw (2003) studied a sample of girls and looked at familial factors associated with externalising symptoms in ADHD. The results indicated that mothers' elevated emotional expression (reflecting high degrees of criticism and over-involvement) predicted ADHD status. They found that emotional expression was more strongly related to ADHD symptoms than ODD/CD

symptoms. These results are generally inconsistent with other findings reflecting strong associations of poor parenting skills with conduct problems.

Miranda et al. (2007) investigated the interaction between a community sample of children and their mothers (ADHD group, and ADHD and CD group). They found mothers of children with ADHD and CD made significantly more negative personal comments about their children and showed less physical and verbal warmth towards them. However, they stated that without longitudinal studies it was not possible to determine what came first in the causative relations between parental warmth and conduct problems: parents could withdraw because of their child's behaviour; but making negative comments in the absence of positive comments may also contribute to the child eventually developing conduct problems. Burke, Pardini, and Loeber (2008) examined the influence of parenting behaviour on changes in ADHD, ODD and CD, as well as the influence of those child behaviours on parenting practices via a longitudinal study. Boys who met criteria for ODD, CD and ADHD were followed until the age of 17. The results found that child disruptive behaviour symptoms had a greater influence on parenting practices, than parenting behaviour did on child symptoms. ADHD symptoms did not predict any measure of parenting, nor did parenting predict ADHD symptoms. Oppositional symptoms predicted decreases in parental involvement and poorer communication across development. They also found a reciprocal relationship between oppositional behaviour and timid discipline over time, such that parents reported an unwillingness to engage in disciplinary practices for fear of escalating oppositional behaviours. Conduct problems predicted decreases in parental supervision, but did not predict changes in discipline, involvement, or communication.

Chronis et al. (2007) investigated the role of observed parent–child interactions, on the development of conduct problems in children with ADHD over early childhood. When child behaviour and demographic variables were controlled, both maternal Depression and parenting during early childhood (for example, observed praise and positive affect) were unique predictors of the developmental course of conduct problems. They attempted to minimize the effect of childhood behaviour problems on parenting to strengthen their inferences regarding the role of parenting in contributing to individual differences in future conduct problems (by controlling for child behaviour that emerged during observations of maternal parenting behaviour). They concluded

that positive parenting acts as a protective factor against conduct problems in the developmental stage of early childhood.

These studies suggest that the psychosocial risk factors associated with ADHD may be exacerbated in children who develop oppositional and conduct problems. However, the direction of these effects is not yet clear and more longitudinal studies are required. Research is also lacking in the area of which factors may be protective for young people and their parents. Chronis et al. (2007) reported that positive parenting of children with ADHD at 4 to 7 years of age predicted fewer future conduct problems (2 to 8 years later). This emphasizes the need to look at parenting behaviour in parents of children with comorbid ADHD and ODD.

Research has also looked at the relationship between parental psychopathology and parenting practices with respect to behavioural symptoms (Kashdan et al., 2004; Pfiffner et al., 2005). They found parental psychopathology and parenting practices were associated with oppositional and conduct problems. Johnston, Murray, Hinshaw, William, and Hoza (2002) found that maternal responsiveness was negatively related to both child conduct problems and to maternal Depressive symptoms, but not to ADHD symptoms. In a follow-up study, mothers of boys with ADHD fell in the mid-range between parents of typically developing boys and those with oppositional symptoms, suggesting a continuum of disturbance involving over-reactive discipline and less responsive parenting (Seipp & Johnston, 2005). These findings emphasize that the interaction between several factors needs to be considered in future longitudinal research designs.

#### *3.4.3.5 Parental Psychopathology and Comorbid ADHD and ODD*

Studies have looked at the relationship between parental psychopathology with respect to children's behavioural symptoms. Pfiffner et al. (2005) found higher rates of paternal CD, parental ADHD, parental Antisocial Personality Disorder and parental Depression among children with comorbid ADHD and conduct problems, which was consistent with several other studies (Chronis et al., 2003; Faraone, Biederman, & Monuteaux, 2000; Harvey, Friedman-Weieneth, Goldstein, & Sherman, 2007; Pfiffner et al., 1999; Smalley et al., 2000; Steinhausen et al., 2013; Tandon, Si, & Luby, 2011).

Very few studies have looked at ADHD and ODD independently from CD. The studies looking at comorbid ADHD and ODD/CD have found mixed results. A couple of studies examined parental psychopathology and comorbid ADHD and ODD failing to find an association with family histories of CD and Antisocial Personality Disorder (Faraone et al., 2000). However, Frick et al. (1992) did find an association. Some research found parental ADHD predicted ADHD and ODD in their offspring (Freitag et al., 2012; Noordermeer et al., 2017).

Numerous researchers have questioned if parental psychopathology has a greater association with comorbid ADHD and oppositional and conduct problems, than with ADHD alone (Chronis et al., 2003; Pfiffner et al., 2005). Chronis et al. (2003) found parental psychopathology (maternal and paternal childhood ADHD) was more strongly associated with ODD or CD than the ADHD symptoms. Chronis et al. (2007) found that maternal Depression predicted conduct problems 2-8 years following an initial assessment, suggesting that maternal Depression was a risk factor for the developmental course of conduct problems among children with ADHD.

Child psychopathology has also been found to increase the risk of parental psychopathology. Chronis et al. (2003) looked at the relationship between maternal reports of parental psychopathology and children's behavioural symptoms in children with ADHD, comorbid ADHD and ODD/CD and typically developing comparison children. They found that mothers of children in the comorbid ADHD and ODD group were more likely to be at greater risk of psychological problems, reporting an increase in symptoms of Mood Disorders (current Mood Disorder, 5.61 times higher; lifetime Mood Disorder, 5.35 times higher), Anxiety disorders (life-time Social Phobia, 4.35 times more likely; Obsessive Compulsive Disorder, 4.39 times more likely), stimulant and cocaine dependence, and drinking problems.

This research does demonstrate evidence that parental psychopathology (including ADHD, ODD, CD, Antisocial Personality Disorder and Depression) may be an important determinant of the emergence of comorbid ADHD and ODD. However, further systematic longitudinal research is required focussed on comorbid ADHD and ODD specifically. Similarly, the impact of the child's comorbid presentation on parental psychopathology also needs further exploration.

#### *3.4.3.6 Summary*

ADHD severity and rates of comorbid ODD or CD are influenced by psychosocial risk factors (Blanz, Schmidt, & Esser, 1991; Copeland et al., 2009). However, there are very few studies that have investigated the risk factors for comorbid ODD (separately from CD) in young people with ADHD. Given how frequently comorbid ADHD and ODD occur and the negative prognosis of comorbid ADHD and ODD, it is important to research the factors that precede the development of ODD in children with ADHD (Whittinger et al., 2007). It appears that several psychosocial risk factors together compound the risk of comorbid ADHD and ODD developing, which in turn may negatively impact the functioning of families, parental relationships, parenting practices, and parental psychopathology. Poor family functioning (increased conflicts, increased negative interactions, poor affective responsiveness), parental relationship dysfunction (more frequent and unresolved conflict between parents and increased divorce rates), increased aversive parenting (lack of supervision, aversive constraints and negative, inconsistent and detached parenting) and worse parental mental health all being reported. Future systematic longitudinal research is required to look at both the risk and protective psychosocial factors associated with comorbid ADHD and ODD due to most research collapsing ODD into an overall disruptive behaviour disorders category.

## 3.5 KEY COMORBID CONDITIONS WITH ADHD AND ODD

### 3.5.1 Introduction

There appears to be very few studies that have investigated the point prevalence of three or more comorbid disorders. Kessler et al. (1994) reported more than half of all lifetime disorders occurred in 14% of the population who had a history of three of more comorbid disorders. The Ontario Child Health Study (Offord et al., 1987) found comorbidity to be high, with 68% of the sample having one or more additional disorders in children and adolescents. The Puerto Rico Child Psychiatry Epidemiologic Study (Bird et al., 1988) reported comorbidity among disorders was found in 46.1% of the sample for children and adolescents. The Christchurch Health and Development Survey (Fergusson et al., 1993) reported 41% of children met criteria for at least two diagnoses, with more than 10% meeting criteria for three or more diagnoses. Langsford, Houghton,

Douglas, and Whiting (2001) studied Western Australian primary and secondary school students and found extensive comorbidity, seeing it as "the rule rather than the exception": of the 338 students assessed, 156 (46.15%) reported one diagnosis and 182 (53.85%) reported two or more diagnoses. Given these high rates of comorbidity found for children and adolescents it is interesting that there has not been more systematic research in this area. This section will present the known literature for key comorbid conditions with ADHD and ODD.

### 3.5.2 Conduct Disorder with Comorbid ADHD and ODD

In studies of community and clinic samples a large percentage of youth with CD or ADHD (45-70%) also meet criteria for the another disorder (Fergusson et al., 1991; Offord et al., 1991). Hamilton and Armando (2008) found that in a group of young people with ADHD, about 40% were also diagnosed with ODD, and about 30% of these ODD cases also met criteria for CD. These results are consistent with other studies that have found that childhood ODD is associated with adolescent CD in children with ADHD at follow up (August et al., 1999; Biederman, Faraone, Milberger, Jetton, et al., 1996; Mannuzza et al., 2004; Whittinger et al., 2007).

Biederman, Faraone, Milberger, Jetton, et al. (1996) examined 140 children with ADHD and 120 typically developing control participants in a follow up study. At baseline, 65% of the ADHD children had comorbid ODD and 22% had CD. Among those with ODD, 32% had comorbid CD. Children with comorbid ADHD, ODD and CD had more severe symptoms of ODD, more comorbid psychiatric disorders, and lower Global Assessment of Functioning Scales scores. ADHD and ODD, without CD, at baseline assessment in childhood did not increase the risk for CD in the 4-year follow-up. They reported that they identified two subtypes of ODD associated with ADHD: one that is prodromal to CD; and another that is not likely to progress into CD in later years. They reported that these ODD subtypes may have different correlates, course, and outcome. This work was expanded upon by Biederman, Petty, Dolan, et al. (2008) finding that ODD subjects with CD could be distinguished from ODD without CD by their more severe CBCL profile. Also, that ODD subjects without CD differed from ADHD subjects without ODD on the CBCL Aggressive Behaviour scale.

### 3.5.3 Depression and Comorbid ADHD and ODD

Given that both ADHD and ODD are commonly associated with Depressive Disorders there is a lack of research in the area of Depression with comorbid ADHD and ODD. Biederman, Petty, Dolan, et al. (2008) found that young people with ADHD and ODD were more likely to develop Major Depression (measured at 4 years and 10 years follow up in a controlled 10-year prospective longitudinal follow-up study).

### 3.5.4 Autism Spectrum Disorder and Comorbid ADHD and ODD

Co-occurring problems are common in individuals with ASD but their relevance for impairment is largely unexplored (Posserud, Hysing, Helland, Gillberg, & Lundervold, 2018). In the longitudinal Bergen Child Study (population 6237 school children; 226 with ASD) researchers found that the high impairment scores for young people with ASD were largely accounted for by co-occurring problems and predicted more contact with treatment services (Posserud et al., 2018). Of those with ASD 21.2% had co-occurring diagnoses of ADHD and ODD. Lecavalier et al. (2019) assessed 658 young people with ASD and found for those who met criteria for ADHD 50% also met criteria for ODD. Children with ASD and ADHD also having ODD were 2.7 times higher than children without ADHD. Future longitudinal studies are required to help explore the consequences for impairment of young people with ADHD, ODD and ASD, along with the implications for effective treatment.

### 3.5.5 Summary

There is a distinct dearth of research in disorders that may also be present with comorbid ADHD and ODD. This is surprising given extensive comorbidity has been seen as “the rule rather than exception”. Often when three or more disorders are present young people present with more severe symptoms and lower global functioning abilities. Future systematic longitudinal research is required to understand the risk and protective factors for developing further comorbid disorders and understanding how to treat these comorbidities effectively.

## 3.6 NATURAL HISTORY OF COMORBID ADHD AND ODD

ADHD and ODD often emerge during the preschool years (Lavigne et al., 2009; Riddle et al., 2013). August et al. (1999) looked at the persistence and desistance of ODD in a community sample of children with ADHD using a 4-year follow-up design. A sample of disruptive children was compared to a sample of non-disruptive children. Few differences distinguished the groups at baseline. Of the 43 children with baseline diagnoses of ADHD and ODD, only 1 (2.3%) was found to have developed CD at follow-up; they found that over time there was a 57% rate of ODD persistence and a 43% rate of ODD desistance.

There are two well-known broad models to understand comorbidity between ADHD and ODD: *the correlated risk-factor model* (Rhee, Willcutt, Hartman, Pennington, & DeFries, 2008), which proposes that both these disorders share risk factors (for example, genes and environment), and *the developmental precursor model* (Barkley, 2006; Beauchaine, Hinshaw, & Pang, 2010; Johnston & Jassy, 2007), which suggests symptoms of ADHD (for example, disrupted family functioning, negative parenting and parental psychopathology) lead to ODD (Ghosh et al., 2017). These two models predict specific patterns of the early development of ADHD and ODD symptoms. The *developmental precursor model* suggests that early ADHD symptoms should predict later ODD symptoms, but not vice versa (Harvey et al., 2016). The *correlated risk factors model* suggests that comorbidity between ADHD and ODD symptoms should emerge at an early age as a function of correlated risk factors (Harvey et al., 2016).

Harvey et al. (2016) annually assessed 199 children as part of a longitudinal study of pre-schoolers with behaviour problems from ages three to six years of age. They found that ADHD symptoms predicted later argumentative/defiant symptoms, supporting the *developmental precursor model*. They also found that family histories of ADHD and ODD/CD symptoms were correlated risk factors that uniquely predicted ADHD and anger/irritable symptoms, supporting the *correlated risk factors model*. These results suggest that the *correlated risk factors model* may best explain the development of comorbidity between symptoms of ADHD and anger/irritability, whereas the *developmental precursor model* may better explain the development of comorbidity between symptoms of ADHD and argumentative/defiance (Harvey et al., 2016). Their

findings that both the *correlated risk factors* and *developmental precursor models* were supported lends further evidence to the notion that multiple mechanisms are involved in the development of comorbidity between ADHD and ODD.

### 3.6.1 Summary

ADHD and ODD often emerge during preschool years, with around half of these young people having persistent ODD over time. Both the *correlated risk factor model* and the *developmental precursor model* are helpful in understanding the multiple mechanisms involved in the comorbidity between ADHD and ODD. However, more systematic longitudinal research is required to further understand the interaction of the multiple mechanisms and their involvement in the development of comorbid ADHD and ODD.

## 3.7 KEY TREATMENT APPROACHES FOR COMORBID ADHD AND ODD

### 3.7.1 Introduction

Given that the rate of comorbidity between ADHD and ODD is high, and persistence of ADHD and ODD in adolescence is linked with an increased risk of a poor prognosis (delinquent behaviour, substance dependence, Anxiety and Depression) it is important to review the literature on effective treatment approaches. However, there is little agreement among researchers regarding the best management practices for these comorbid conditions. Generally, a team approach by a psychologist, the school system and medical support (paediatrician and/or psychiatrist) is recommended with the development of an individualized approach for each young person and their family. Interventions, both pharmacological and non- pharmacological (family, child, and multi-system), will be discussed in this section.

### 3.7.2 Pharmacological Treatments for Comorbid ADHD and ODD

Hazell (2010) conducted a practitioner review of comorbid ADHD and ODD and found that clinicians reported it was important to manage the core symptoms of ADHD as well as the ODD symptoms. He noted that mild cases may respond to behaviour

management alone, or monotherapy with stimulant medication or atomoxetine. Moderate to severe cases usually required a combination of pharmacotherapy, which may include clonidine, and behaviour management. Severe or refractory cases may require the introduction of an atypical antipsychotic medication such as risperidone.

Limited research has looked at comorbid ADHD and ODD (separately from CD). Pringsheim et al. (2015) performed a systematic review and meta-analysis of stimulant medication, clonidine/guanfacine and atomoxetine for ODD behaviours, CD, and aggression in youth with ADHD, ODD and CD. They found there is high-quality evidence that stimulant medications have a moderate-to-large effect on oppositional behaviour, conduct problems, and aggression in youth with ADHD, with and without ODD or CD. There is very-low-quality evidence that clonidine has a small effect on oppositional behaviour and conduct problems in youth with ADHD, with and without ODD or CD. There is moderate-quality evidence that guanfacine has a small-to-moderate effect on oppositional behaviour in youth with ADHD, with and without ODD. There is high-quality evidence that atomoxetine has a small effect on oppositional behaviour in youth with ADHD, with and without ODD or CD. They concluded that evidence indicates that stimulant medication, clonidine/guanfacine, and atomoxetine can be beneficial for disruptive and aggressive behaviours in addition to core ADHD symptoms, while stimulant medications generally provide the most benefit.

Pringsheim et al. (2015) also performed a systematic review and meta-analysis of randomized controlled trials of antipsychotic medications, lithium, and anticonvulsant medications for aggression and conduct problems in youth with ADHD, ODD, and CD. They found that there is moderate-quality evidence that risperidone has a moderate-to-large effect on conduct problems and aggression in youth with subaverage IQ and ODD, CD, or disruptive behaviour disorder not otherwise specified, with and without ADHD. There was also high-quality evidence that risperidone has a moderate effect on disruptive and aggressive behaviour in youth with average IQ and ODD or CD, with and without ADHD. They found the evidence supporting the use of haloperidol, thioridazine, quetiapine, and lithium in aggressive youth with CD is of low or very-low quality, and evidence supporting the use of sodium valproate in aggressive youth with ODD or CD is of low quality. There is very-low-quality evidence that carbamazepine is no different from placebo for the management of aggression in youth with CD. They concluded that

except for risperidone, the evidence to support the use of antipsychotic medications and mood stabilizers is of low quality.

### 3.7.3 Limitations of Pharmacological Treatments for Comorbid ADHD and ODD

Stimulant medication is generally helpful in the treatment of ADHD, but is not always sufficient alone to manage ODD symptoms in comorbid ADHD and ODD (Jensen et al., 2005), and may not be effective for 20% to 30% of children with ADHD (Swanson, McBurnett, Christian, & Wigal, 1995). Many parents are reluctant to use medication in young children due to concerns about adverse effects on developing brain structures (Henderson & Fischer, 1995) and research has found that parents of children who are prescribed stimulant medications generally only use them for one to two months (Sherman & Hertzig, 1991). There is also little evidence to show that medication improves academic achievement long term (Swanson et al., 1995) or prevents the escalation of ADHD and ODD to CD in adolescence (Hinshaw, 1994; Pelham, Jr. et al., 1998).

### 3.7.4 Non-Pharmacological Treatments for Comorbid ADHD and ODD

Battagliese et al. (2015) completed a meta-analysis of Cognitive Behavioural Therapy (CBT) treatment effectiveness for ADHD and ODD. They focused on CBT's efficacy to reduce externalizing symptoms, improve social competence, increase positive parenting, reduce internalizing behaviours, improve parent stress, and reduce maternal depressive symptoms. The results showed that the biggest improvement after CBT was in ODD symptoms, followed by parental stress, externalizing symptoms, parenting skills, social competence, and ADHD symptoms. They found CBT was also associated with improved attention, aggressive behaviours, internalizing symptoms, and maternal depressive symptoms. They reported that CBT is an effective treatment option for ADHD and ODD and is also associated with reduced parental distress and maternal depressive symptoms. Multimodal treatments targeting both children and caregivers' symptoms (for example, maternal depressive symptoms) appear important to produce sustained and generalized benefits for these young people and their families.

Forehand et al. (2016) compared two behaviour parent training programs (New Forest Parenting Program, designed for children with ADHD; Helping the Noncompliant

Child, designed to treat children with ODD) in a sample of 130 pre-schoolers with comorbid ADHD and ODD. They found that Helping the Noncompliant Child was more effective with disruptive behaviours than the New Forest Parenting Program for children with a comorbid diagnosis.

### 3.7.5 Combined treatment for comorbid ADHD and ODD

The Multimodal Treatment Study of Children with ADHD (MTA Cooperative Group, 1999a, 1999b) is probably one of the most well-known studies investigating treatments for children with ADHD and associated behavioural difficulties. They compared medication management; intensive behavioural treatment (parent, school, and child components); medication and intensive behavioural treatment; and standard community care (by community providers). They found that all four groups showed reductions in symptoms over time. For most ADHD symptoms, children in the combined treatment and medication management groups showed significantly greater improvement than those given intensive behavioural treatment and community care. The combined and medication management treatments did not differ significantly for ADHD symptoms. However, combined treatment was superior for oppositional/aggressive symptoms, internalizing symptoms, teacher-rated social skills, parent-child relations, and reading achievement. This study suggested that combined medication and intensive behavioural treatment demonstrated better results for non-ADHD symptoms and positive functioning outcomes, setting the scene for future research for treatment of young people with combined ADHD and ODD.

Combined treatment, including behavioural and pharmacological interventions, is typically recommended to address ADHD and ODD. Swanson et al. (2001) reported that treatment success improved 20% when combined intervention was used, relative to either psychosocial or pharmacological treatment alone. Zonnevylle-Bender, Matthys, van de Wiel, and Lochman (2007) found Cognitive Behavioural Therapy for adolescents, with behavioural parenting training for ODD, and stimulant medication for ADHD was helpful in preventing delinquency and substance use comparable with typically developing peers.

Connor (2015) reviewed the literature on the characteristics and treatment of children with ADHD and ODD. He presented a clinical approach for treatment of ADHD and ODD emphasizing the importance of child and parent psychoeducation (about the

two disorders alone and in combination), the importance of behavioural management therapy approaches (school and academic supports), and the decision to use medication (depending on symptom severity combined with the child and parental wishes). Greene and Ablon (2001) argue that a broad range of medication and psychosocial interventions need to be considered for this group of young people, carefully selected, matched, and tailored to the individual needs of each child and their family with ADHD and ODD, and implemented and monitored over the longer term for optimal outcomes.

### 3.7.6 Summary

Combined treatment, including both behavioural and pharmacological interventions, is typically recommended to address comorbid ADHD and ODD. A meta-analysis of pharmacological treatment for comorbid ADHD and ODD demonstrated evidence that stimulant medications, clonidine/guanfacine, and atomoxetine can be beneficial for disruptive and aggressive behaviours in addition to core ADHD symptoms with stimulant medications generally providing the most benefit. Except for risperidone, the evidence to support the use of antipsychotic medications and mood stabilizers is of low quality for the treatment of comorbid ADHD and ODD. Multimodal treatment, including CBT with a focus on reducing externalizing symptoms, improving social competence, increasing positive parenting, reducing internalizing behaviours, improving parent stress, and reducing maternal Depression, is important to produce sustained and generalized benefits and is recommended.

## 3.8 Chapter Conclusion for Comorbid ADHD and ODD and Future Research Directions

ADHD is consistently shown to have a greater than chance occurrence with a number of other mental health disorders, especially ODD. High rates of comorbidity between ADHD and ODD have been noted. Children with combined ADHD and Disruptive Behaviour Disorders manifest more severe ADHD symptoms, greater social dysfunction, greater academic underachievement, more behavioural problems, and

greater general impairment in daily functioning. It is hard to get a clear picture of the difficulties associated with ADHD and comorbid ODD, as externalizing disorders (ODD and CD) are often reported together in the literature. Although, ADHD usually first presents in childhood and persists into adulthood, ODD can emerge during different developmental stages and may or may not persist over time: prevalence rates and prognosis depend upon the developmental period that ODD develops.

Assessing for comorbidities can be complex; however, an accurate assessment is important because the co-occurring ADHD and ODD can lead to greater risk for poor outcomes and have implications for treatment responsiveness. Both ADHD and ODD are diagnosed after a comprehensive assessment of gathering information from multiple informants by a qualified professional. Typically, in Australia we use the diagnostic criteria set out in DSM-5 for both disorders. Diagnosing comorbid ADHD and ODD can be complicated due to the overlap in symptomatic behaviour of the two disorders and understanding young people's behaviour during different developmental stages. Clinicians need to avoid mis-diagnosing these comorbid conditions due to a halo effect.

The high rates of comorbid ADHD and ODD may be due to each disorder having similar aetiological risk factors. Child functional factors such as temperament, cognitive functioning, Working Memory, academic achievement, emotional regulation ability and social competency are all important to explore in comorbid ADHD and ODD. In addition, key psychosocial factors such as family functioning, parental relationship functioning, parenting skill, and parental psychopathology play a role in the development and maintenance of ADHD and ODD. There is limited research in the area of comorbid ADHD and ODD due to ODD and CD generally being reported together. Moreover, systematic longitudinal studies are crucial to determine the bidirectional relationship between these disorders and their risk factors across developmental stages. Further clarification of the specific child and psychosocial factors predicting the development of ODD, or ongoing ODD, in young people with ADHD and what factors are protective against ODD are needed. Indeed, investigating the possible interaction between multiple risk factors needs to be considered in future longitudinal research designs.

To date, few studies have investigated the prevalence of three or more comorbid disorders including ADHD and ODD. Further research is required in this area to help

understand the risk and protective factors for comorbidity and the effective treatment of these comorbidities.

There are two well-known models for understand comorbidity between ADHD and ODD that have been noted: the *correlated risk factor model* and the *developmental precursor model*. Researchers suggest both models have validity: that the *correlated risk factors* model may best explain the development of comorbidity between symptoms of ADHD and anger/irritability; whereas the *developmental precursors model* may better explain the development of comorbidity between symptoms of ADHD and argumentative/defiance.

Given that the rate of comorbidity between ADHD and ODD is high, and persistence of ADHD and ODD is linked with an increased risk of a poor prognosis it is important to ensure clinicians use evidence based practice approaches for treatment. Pharmacological treatments are beneficial for ADHD and for aggressive behaviours in addition to core ADHD symptoms. However, combination (pharmacological and non-pharmacological) interventions by a team of professionals (psychologist, school system and paediatrician/psychiatrist), using a multi-systemic framework provide the best outcomes for comorbid ADHD and ODD.

### 3.8.1 Rationale for the Current Study

Many prior studies have used cross sectional correlational designs, which compare the patterns of association between parent and child variables, but are unable to delineate the risk factor trajectories associated with the onset or development of comorbid conditions (Deault, 2010). Longitudinal studies that evaluate whether symptoms of one disorder or risk factor predict later symptoms of a second disorder can help to understand the effects of one disorder/risk factor on the development of another disorder (Deault, 2010; Harvey et al., 2016). A more complete understanding of the transactional relationship between ADHD and ODD symptoms can be examined by looking at how ADHD and ODD predict one another over multiple time points and how other risk factors also play a role in the development.

In Australia, two longitudinal studies have looked at risk factors for adolescent antisocial behaviour: The Australian Temperament Project (2000) (Prior, Smart, Sanson, & Oberklaid, 2000); and The Mater University Study of Pregnancy (1981-1995) (Lawlor

et al., 2004). From these studies, it seems that the trajectories for children with ADHD with regards to developing ODD remain somewhat unclear. Three longitudinal studies have looked at the role of different factors in the development of ODD/CD in children with ADHD (Burke et al., 2008; Chronis et al., 2007; Latimer et al., 2003). These studies emphasise the importance of examining both individual child characteristics and environmental factors when understanding the risk factors for developing or persisting ODD/CD in young people with ADHD. No longitudinal study has looked at the development of ODD (independently of CD) for young people with ADHD. To date, an inconsistent and limited picture remains about the clinical characteristics that contribute to young people with ADHD developing ODD. More systematic longitudinal research is needed to investigate the different child, parent and family domains that can either be a risk factor or a protective factor for developing ODD in young people with ADHD.

### 3.8.2 Aims and Hypotheses

A three-year blinded follow-up evaluation of children with ADHD will be undertaken, investigating those with and without follow up ODD. Four studies will be completed:

#### *3.8.2.1 Study One*

The aim of study one is to examine the key clinical risk and protective *child* associated factors (gender, IQ, temperament, Working Memory, academic ability, emotional regulation, and social problems) for young people with ADHD having follow-up ODD. The hypothesis to be tested is that young people with follow-up ODD and ADHD compared to those diagnosed with ADHD without ODD will manifest differential key child characteristics and have: increased levels of temperament activity; decreased level of task persistence and attention span; lower verbal IQ; worse Spatial Working Memory performance; academic underachievement; have poorer emotional regulation abilities; increased levels of aggression; and increased difficulties in keeping friends and over all social difficulties.

#### *3.8.2.2 Study Two*

The aim of study two is to examine the risk and protective *psychosocial* factors (parental level of education, family functioning, parental relationship functioning, parenting skill, and parental psychopathology) of young people with ADHD having

follow-up ODD. The hypothesis to be tested is that young people with ADHD and follow up ODD compared with those diagnosed with ADHD without ODD will manifest differential key psychosocial characteristics including: decreased parental educational attainment; decreased family functioning; decreased parental relationship functioning; decreased parenting skill; and increased maternal Depression.

#### *3.8.2.3 Study Three*

The aim of study three is to examine the risk and protective *comorbid* conditions (ODD, CD, Depression (MDD and PDD), Anxiety and ASD) and the ADHD subtypes (ADHD-C, ADHD-I, ADHD-HI) of young people with ADHD having follow-up ODD. The hypothesis to be tested is that young people with ADHD having follow-up ODD compared with those diagnosed with ADHD without ODD will manifest differential comorbid conditions, namely: increased ODD; increased CD; and increased ADHD-C.

#### *3.8.3.4 Study Four*

The aim of study four is to help understand which of the significant factors found in study one (child characteristic factors), study two (psychosocial factors) and study three (ADHD subtype and comorbid conditions) better explain the risk factors for young people with ADHD developing or maintaining ODD.

# CHAPTER 4: Methodology

## 4.1 METHODOLOGY

This chapter outlines the Methodology used to systematically examine key child functional characteristics (gender, IQ, temperament, Working Memory, academic ability, emotional regulation skills, and social competence), psychosocial factors (family functioning, parental relationship functioning, parental skill, and parental psychopathology), ADHD subtype (ADHD-C, ADHD-I, ADHD-HI) and comorbid conditions (ODD, CD, MDD, PDD, Anxiety and ASD) risk and protective factors in children with ADHD with follow-up ODD and ADHD without follow-up ODD.

## 4.2 STUDY DESIGN

A three-year blinded longitudinal evaluation of children with ADHD was conducted. Relevant demographic, clinical, and functional (biological, psychological, and social) information was systematically collected from clinically referred participants with ADHD at baseline. The assessors at follow up were blind to the status of each participant at baseline. After the initial assessment, each participant remained involved with the clinic through primary and/or secondary consultation with their primary health care team. Through this process, standardised, incremental ADHD stimulant/non-stimulant medication protocols were used, according to the American Academy of Child and Adolescent Psychiatry (Connolly & Bernstein, 2007). Clinical responder status of at least a 40% improvement in ADHD core symptoms with medication was achieved for all cases as emphasised by Weiss et al. (2019). However, individual and group Cognitive Behaviour Therapy (CBT) treatment elements were not controlled in the Study Design as for the community treatment arm of the MTA Cooperative Group (1999a) study.

## 4.3 RECRUITMENT

Young people with ADHD were recruited to the study following their referral to a specialist ADHD clinic based in the Academic Child Psychiatry Unit (ACPU) and/or

Developmental Neuropsychiatry Program (DNP) at the Royal Children's Hospital (RCH) in Melbourne, Australia. The majority (N= 323, 77%) of the children were referred by school teachers and/or school-based psychologists working in 53 metropolitan primary schools in the city of Melbourne. They were referred for the initial assessment of behavioural problems – academically and socially – in the school and home environments suggesting that they may fulfil the criteria for ADHD. All were medication naïve. The remainder (N=96, 23%) were young people with a pre-existing diagnosis of ADHD referred by local paediatricians, child psychiatrists or clinical psychologists for clarification of their current clinical diagnosis to better target future psychosocial and/or medication treatments. 77 (80%) of this group were treated with stimulant medication or atomoxetine.

## 4.4 PARTICIPANTS

Children diagnosed with ADHD (N=419) were identified from consecutive referrals (N=600), referred to a specialist ADHD clinic based at the RCH Melbourne, Australia. Parent, child, and teacher standardised categorical (semi-structured clinical interview) and dimensional (questionnaire) reports were obtained to establish a diagnosis of ADHD and associated disorders. In addition, two consultant child and adolescent psychiatrists reviewed all diagnoses made after separate clinical interviews. Inter-rater reliability was ascertained between the two consultant psychiatrists completing the diagnostic process for each diagnosis made, including ADHD subtype ($k > 0.90$). After the baseline assessment all participants were followed up three years later. Given that 25% of the sample were not able to be re-assessed (N=104), 150 young people were recruited with ADHD and follow up ODD (120 males; 30 females) and 60 young people were recruited with ADHD alone [ODD absent] (43 males; 17 females). Inclusion criteria were those participants with follow up data for ADHD and separately ODD; a baseline DSM-IV diagnosis of ADHD (all presentations); age range between 7 to 12 years; and a Full Scale IQ score 70 or over. Exclusion criteria, for the two follow-up groups, were young people who had some subclinical ODD features that precluded them from the study (this will be defined later in this chapter) (N=105); full scale IQ less than 70; and children with a primary diagnosis of a Psychotic or Bipolar Disorder.

Participant details for the two groups were as follows: The ADHD without follow up ODD group had a mean age at baseline of 9.33 (2.98) years and the ADHD and follow up ODD group 8.69 (1.56) years, and the two groups did not differ in this regard [$t$(208)=1.58, $p$=.12]. ADHD without follow up ODD group: methylphenidate N%=10; dexamphetamine N%=2; Ritalin LA N%=15; Concerta N%=40; Vyvanse N%=8; Atomoxetine N%=14; no treatment N%=11. ADHD with follow up ODD group: methylphenidate N%=9; dexamphetamine N%=3; Ritalin LA N%=14; Concerta N%=38; Vyvanse N%=9; Atomoxetine N%=17; no treatment N%=10. The two groups did not differ with respect to medication used [$t$(186)=0.15, $p$=.88], final dose titrated to and used [$t$(186)=0.02, $p$=.99] or duration of medication treatment [$t$(186)=0.52, $p$=.61]. All medicated participants were clinical responders and the two groups did not differ [$t$(186)=0.04, $p$=.97]: ADHD without follow up ODD group: 43.00 (1.3) % ADHD with follow up ODD group: 43.00 (1.7) % improvement in ADHD core symptoms (Weiss et al., 2019). The baseline characteristics of the 25% of the sample not able to be re-assessed did not differ from those included in the study.

## 4.5 MEASURES

### 4.5.1 Diagnostic Assessments

#### *4.5.1.1 Anxiety Disorders Interview for Children (A-DISC)*

The A-DISC (Silverman & Albano, 1996) is a semi-structured diagnostic interview schedule with child and parent versions. It is based on DSM-IV criteria and yields a categorical presence or absence of given disorders. In both the parent and child versions, symptoms and associated impairment in social, academic, and family domains are rated separately. It is used frequently to diagnose a range of disorders, including *Anxiety Disorders (Separation Anxiety Disorder, Social Phobia, Specific Phobia, Panic Disorder, Agoraphobia, Generalised Anxiety Disorder), Obsessive Compulsive Disorder, Post-Traumatic Stress Disorder, Depressive Disorders (Major Depressive Disorder, Persistent Depressive Disorder (Dysthymia)), Attention Deficit Hyperactivity Disorder (Inattentive type, Hyperactivity-Impulsive type, Combined type), Oppositional Defiant Disorder, Conduct Disorder, Selective Mutism, Enuresis, Sleep Terror Disorder and Schizophrenia.* The A-DISC was selected for use in this study as it is specific for the

assessment of psychiatric disorders in both children and adolescent populations, rather than adolescent and adult populations. Each disorder was scored in accordance with the Scoring Logic Manual which outlines which questions in the interview endorse specific criteria from DSM-IV. In order to score responses, the A-DISC user would need to identify combinations of corresponding questions for the criteria/diagnoses they wish to identify.The A-DISC has been shown to be a reliable instrument to derive diagnosis in children (kappa co-efficients from .63-.80 for the child interview, and .65-.88 for the parent interview) (Silverman & Berman, 2001). There is support for the concurrent validity of the ADISC-IV ADHD module based on parent interviews (Jarrett, Wolff, & Ollendick, 2007). Research findings also support the clinical utility and validity of the A-DISC.

#### *4.5.1.2 Child Behaviour Checklist (CBCL)*

The CBCL (Achenbach & Rescorla, 2001) is a general measure of emotional and behavioural problems in young people aged 6-18 years. It has a parent, teacher (Teacher Report Form: TRF) and child form (Youth Self Report: YSR). It consists of 118 behaviour problem items with a 3-point response format (0 = not true, 1 = somewhat or sometimes true, 2 = very true or often true). It is based on the young person's behaviour over the last six months. The CBCL provides information on the following syndromes: *anxious/depressed; withdrawn/depressed; somatic complaints; social problems; thought problems; attention problems rule-breaking behaviour; and aggressive behaviour*. It also comprises items identified by experts consistent with the DSM categories of: *depressive problems; anxiety problems; somatic problems; attention deficit/hyperactivity problems, oppositional defiant problems; and conduct problems*. There are two broad band scales that combine several of the syndrome scales: *internalising problems* sums the anxious/depressed, withdrawn-depressed, and somatic complaints scores; *externalizing problems* combines rule-breaking and aggressive behaviour. There also is a *total problems* score, which is the sum of the scores of all the problem items. The CBCL also uses a normative sample to create standard scores. These compare the raw score to what would be typical compared responses for young people of the same gender and similar age. The standard scores are scaled so that 50 is average for the young person's age and gender, with a standard deviation of 10 points. Higher scores indicate greater problems. For each internalizing and externalizing problem scales, and total score, scores can be

interpreted as falling in the normal, borderline, or clinical behaviour range. Any score that falls on the 93rd percentile is considered normal, scores between the 93-97th percentile are borderline clinical behaviour, and any score above the 97th percentile are in the clinical range. Norms take into account both age and gender. There are also separate norms for girls and boys, and separate norms for ages 6-11 and ages 12-18. The CBCL is a reliable tool. Internal consistencies for the externalising, internalising, and total problems ranged from .88 to .96. It also has good concurrent validity as it correlates well with related measures, and has strong discriminant validity (Achenback, 1991).

#### *4.5.1.3 The Autism Spectrum Quotient: Children's Version (AQ-Child)*

The AQ-Child (Auyeung, Baron-Cohen, Wheelwright, & Allison, 2008) is a 50-item parent-report questionnaire that aims to quantify autistic traits in children 4-11 years old. The AQ-Child consists of a series of descriptive statements designed to assess five areas associated with autism and the broader phenotype: social skills, attention switching, attention to detail, communication, and imagination, each represented by ten items. Higher scores correspond to more 'autistic-like' behaviour. AQ-Child items consist of statements that are answered on a Likert scale (definitely agree, slightly agree, slightly disagree, and definitely disagree). The range of scores is 0-150. Total AQ scores are represented by the sum of each item score. The minimum AQ score (0) indicates no autistic traits; the maximum score (150) suggests full endorsement on all autistic items. Using a cut-off score of 76 the AQ-Child has high sensitivity (95%) and specificity (95%). The AQ-Child has good test-retest reliability ($r=0.85$, $p<0.001$) and high internal consistency (Cronbach's coefficient = 0.97) (Auyeung et al., 2008).

#### *4.5.1.4 The Adolescent Autism Spectrum Quotient (AQ-Adol)*

The AQ-Adol (Baron-Cohen, Hoekstra, Knickmeyer, & Wheelwright, 2006) is a parent-report questionnaire that aims to quantify autistic traits in adolescents 12-15 years. The AQ-Adol is divided into five different domains or categories of autistic traits: social skills, attention to detail, attention switching, communication and imagination. Each domain is assessed by ten questions. AQ-Adol items consist of statements that are answered in a Likert scale (definitely agree, slightly agree, slightly disagree and definitely disagree). AQ-Adol items are counterbalanced to avoid a response bias, so that half of

the ‘agree’ responses and half of the ‘disagree’ responses endorse the autistic trait. The AQ-Adol includes questions about both ability and preference. The minimum score 0 and the maximum score is 50. The AQ-Adol total score and its five subscale scores are normally distributed and have demonstrated good test-retest reliability and good internal consistency (Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001). The measure has high sensitivity and specificity: at cut-off score of 26, 83% of patients were correctly identified (sensitivity 0.95, specificity 0.52, positive predictive value 0.84, negative predictive value 0.78), while a cut-off score of 32 correctly identified 76% of patients (sensitivity 0.77, specificity 0.74) (Austin, 2005).

### 4.5.2 Collection of Demographic Information

#### *4.5.2.1 Parental Account of Childhood Symptoms, demographic and developmental sections (PACS)*

The PACS (Taylor et al., 1986) is a semi-structured clinical interview developed as an instrument for the measurement of children’s behaviour problems as experienced at home. It is administered by a trained interviewer. Parents provide detailed descriptions of their child’s behaviour in specified situations over the previous week and over the preceding 12-month period. It also yields demographic data and developmental data. A *Social Adversity Scale* (SAS) is developed based on a summary of family income level (scored 1-4)(1 “$0-$30,000; 2 “ $30,000-$40,000; 3 “$40,000-$50,000; 4 $50,000 and over”), mother’s educational level (scored 1-7) (1 “never attended school”; 2 “some years of primary school”; 3 “primary school”; 4 “some years of secondary school”; 5 “H.S.C, or equivalent”; 6 “technical, trade or TAFE certificate”; 7 “tertiary qualifications”), single parent status (0-1), sibling size (0- ), and broken home status (1-2). The PACS was used only to collect demographic data, via the SAS. It has good inter-rater reliability, K> .80 and adequate validity (Taylor et al., 1986).

### 4.5.3 Temperament Assessment

#### *4.5.3.1 Infancy Temperament Questionnaire*

Thomas and Chess (1977) developed a nine-dimensional model of temperament including: activity level, rhythmicity, approach-withdrawal, adaptability, responsiveness, intensity of reaction, mood, distractibility, and attention span. *Activity*: refers to the

child's physical energy. Is the child constantly moving, or does the child have a relaxing approach? This trait can also refer to mental activity, such as deep thinking or reading activities which become more significant as the person matures. *Regularity:* also known as *rhythmicity*, refers to the level of predictability in a child's biological functions, such as waking, becoming tired, hunger, and bowel movements. *Initial reaction:* also known as *approach or withdrawal*. This refers to how the child responds (whether positively or negatively) to new people or environments. Does the child approach people or things in the environment without hesitation, or does the child shy away? *Adaptability:* refers to how long it takes the child to adjust to change over time (as opposed to an initial reaction). Does the child adjust to the changes in their environment easily, or is the child resistant? *Sensitivity (sometimes called sensory threshold or threshold of responsiveness):* refers to how easily a child is disturbed by changes in the environment. Is the child bothered by external stimuli like noises, textures, or lights, or does the child seem to ignore them? *Intensity:* refers to the energy level of a positive or negative response. Does the child react intensely to a situation, or does the child respond in a calm and quiet manner? *Mood:* refers to the child's general tendency towards a happy or unhappy demeanor. All children have a variety of emotions and reactions, such as cheerful and stormy, happy, and unhappy. Yet each child biologically tends to have a generally positive or negative outlook. *Distractibility:* refers to the child's tendency to be sidetracked by other things going on around them. Does the child get easily distracted by what is happening in the environment, or can the child concentrate despite the interruptions? *Task persistence and attention span:* refer to the child's length of time on a task and ability to stay with the task through frustrations—whether the child stays with an activity for a long period of time or loses interest quickly.

Behaviours for each one of these traits are on a continuum. If a child leans towards the low end of the scale, it could be a cause for concern. This questionnaire is completed retrospectively by parents who are asked "*which was generally true of your child during infancy (babyhood)*" for each dimension. Parents or caregivers are asked to circle on a scale of one (high/irregular/negative/slow/intense/short) to seven (low/regular/positive/quick/mild/long) for each temperament dimension. It has good inter-rater reliability, K> 0.80 and adequate validity (Thomas & Chess, 1977).

### 4.5.4 Intelligence Test

#### *4.5.4.1 Wechsler Intelligence Scales for Children, Fourth Edition (WISC-4)*

The WISC-4 (Wechsler, 2003) is an individually administered paper-and-pencil clinical instrument that provides an estimate of general intellectual ability and is suitable for children aged six to sixteen years. The WISC-4 is the most widely employed standardised measure of general intellectual functioning in young people and has demonstrated sound psychometric properties. The WISC-4 takes from one and half to two hours to complete.

It is a clinician administered test consisting of: three subtests measuring *Verbal Comprehension* abilities, three subtests measuring *Perceptual Reasoning* (non-verbal) abilities; two subtests measuring *Working Memory* performance; and two subtests measuring *Processing Speed* ability. The *Verbal Compression Index (VCI)* measures verbal concept formation, verbal reasoning, and knowledge acquired from one's environment which subtests include: similarities (which measures verbal concepts and reasoning); vocabulary (which measures word knowledge and retrieval); and comprehension (which measures social knowledge and awareness). The *Perceptual Reasoning Index (PRI)* measures perceptual and fluid reasoning, spatial processing and visual-motor integration, which subtests include: block design (which measures visual spatial reasoning and visual-constructional ability); matrix reasoning (which measures nonverbal reasoning and concept formation); and picture concepts (which measures abstract, categorical reasoning). The *Working Memory Index (WMI)* requires working memory processes to manipulate orally presented verbal sequences, or recall orally presented sequential information, which subtests include: digit span forwards (which measures auditory short-term memory); digit span backwards (which measures auditory working memory); letter-number sequencing (which measures shorter string lengths indicating memory, and longer string lengths reflecting auditory processing). The *Processing Speed Index (PSI)* requires visual perception and organisation, visual scanning, and the ability to use hands and eyes together efficiently, which subtests include: coding (which measures speed and accuracy (fine motor control) and incidental learning); and symbol search (which measures mental processing speed and accuracy). A sum of all four of these domains forms a *Full-Scale IQ (FSIQ)* score. Each subscale has a standardized mean and standard deviation of 100 and 15, respectively. The FSIQ

is composed of ten core subtests: the three *Verbal Comprehension* subtests; the three *Perceptual Reasoning* subtests; the two *Working Memory* subtests; and the two *Processing Speed* subtests, all with a standardized mean and standard deviation of 100 and 15. The FSIQ also has a standardized mean and standard deviation of 100 and 15.

The WISC-4 was normed on a representative sample of the United States population, with subsequent publication of Australian norms (Wechsler, 2005), used in the studies undertaken in this thesis. The FSIQ and the four indices, as well as the subtests, have excellent reliability (for example, internal consistency and test-retest) and validity (Wechsler, 2003; Williams, Weiss, & Rolfhus, 2003).

Administration and scoring of the WISC-4 took place according to standard protocols, either by a provisional psychologist (supervised by a registered clinical psychologist) or registered clinical psychologist (Sattler, 2001; Wechsler, 2003).

### 4.5.5 Educational Assessment

#### 4.5.5.1 *Wide Range Achievement Test, Fourth Edition (WRAT-4)*

The WRAT-4 (Wilkinson & Robertson, 2006) measures the basic academic skills: *word reading* (measures word decoding through letter identification and word recognition); *reading comprehension* (measures the ability to identify the meaning of words and to comprehend the ideas and information in a sentence using a modified cloze technique); *spelling* (evaluates an individual's ability to identify sounds and transfer them into written form from dictated letters and words); and *mathematical computation* (measures an individual's ability to count, identify numbers, solve simple oral math problems, and calculate written math problems. Math problems are presented in a range of domains, including arithmetic, algebra, geometry, and advanced operations). It is a pencil and paper test, which provides two equivalent forms (Blue and Green), which enables retesting within short periods of time without potential practice effects and can be completed in fifteen to forty-five minutes depending upon the person's age. It can be administered by those who are have been trained and demonstrate competence in the use of psychological tests.

The WRAT-4 was standardized on a national sample of over 3,000 individuals ranging in age from 5 to 94. Scores were developed for both age and grade referenced

groups. Standard scores (mean of 100 and a standard deviation of 15), percentile ranks, stanines, normal curve equivalent, grade equivalents, and Rasch ability scale scores are provided. Alternate-form immediate retest reliability coefficients ranged from 0.78 to 0.89 for an age-based sample and from 0.86 to 0.90 for grade-based sample. The WRAT-4 has been shown to be highly reliable (0.98 for reading, 0.94 for mathematics, 0.93 for spelling), independent research supports the use of the WRAT-4 for a variety of purposes. Internal validity reported intercorrelations between subtests range from r=0.60 (grade) to 0.63 (age). External validity, correlations with: Wechsler Individual Achievement Test-Third Edition ranging 0.31- 0.92; Woodcock-Johnson-Third Edition, Test of Achievement ranging 0.44 - 0.78; Wechsler Intelligence Scale for Children – fourth edition (Full Scale IQ) ranging 0.57 - 0.81; Stanford Binet Intelligence Scale (Full Scale IQ) ranging 0.67 - 0.78.

Administration and scoring of the WRAT-4 took place according to standard protocols, either by a provisional psychologist (supervised by a registered clinical psychologist) or registered clinical psychologist.

### 4.5.6 Visual Spatial Working Memory Assessment

#### *4.5.7.1 Neuropsychology functioning assessment using tasks from the Cambridge Neuropsychological Test Automated Battery (CANTAB for Windows)*

The CANTAB ("CANTAB for Windows," 1999) was developed at the University of Cambridge and is a behavioural measure that has been validly and reliably linked to functional neuroanatomical and neurophysiological measures in human beings across the life cycle and non-human primates. It is a computerised test battery developed to predominantly assess neuropsychological functioning based on fronto-striatal circuitry and to separate frontal from temporal lobe functions (Fray & Robbins, 1996). The relevant tests used were *Spatial Span (SS) and Spatial Working Memory (SWM)* (incorporating a Strategy score and a *Between Search Errors (BSE)* score of SWM generally). The CANTAB subtests are language free and tasks are graded in difficulty. The CANTAB SS and SWM test batteries were administered in one testing session of approximately forty minutes. The tests were presented on an IBM colour monitor via a

high-resolution, touch sensitive screen. Participants were seated approximately 50 cm from the colour monitor.

The SS task assesses the ability to encode and retrieve a sequence of spatial stimuli. It is a computerised version of the Corsi Block Tapping Test (Milner, 1971) and provides a measure of visuospatial short-term memory capacity. The task assesses the ability of the participant to remember a sequence of squares that change colour in a random order, one square at a time. Participants are required to observe the sequence of squares changing colour, and then to replicate the sequence by touching the boxes on the computer screen. Participants must replicate the correct sequence to progress to the next level of difficulty; an increase in the number of squares that change colour. After an incorrect sequence, the trial remains at the same level of difficulty for a maximum of three attempts. The SS score is calculated as the highest level at which participants successfully replicated at least on sequence of squares. The sequence of squares presented begins at two and increases to a maximum of nine squares. The maximum possible Spatial Span score is nine.

The SWM task of the CANTAB is a self-ordered searching test that measures working memory for spatial stimuli and requires participants to use mnemonic information to work towards a goal. It is based on the Radial Arm Maze developed in animal research (Olton & Wolf, 1981). The WM task requires participants to search for 'blue tokens' hidden in boxes presented on the screen. A box is 'opened' by touching it on the screen. At any one time, there is only one blue token hidden within a box, and participants must search through boxes until it is found. The blue token is then placed in the 'black hole' to the side of the screen. Once a token has been located within a box, that particular box will not contain another blue token, it is 'empty'. Two practice trials with two boxes are initially presented. Four test trials followed at each level of difficulty: two; three; four; six; and eight boxes. Two types of errors are possible during this task. A 'between search' error (BSE), or 'forgetting' occurs when a participant returns to a box that has been emptied of a blue token. A 'within search' error occurs when a participant returns to a box that has already been searched and found to be empty, during the same search. As well as the mnemonic aspect of the SWM task, that is the ability to remember where the blue tokens were in the search task, there is a strategy component to any self-ordered search task. This component measures the ability to plan and organise a sequence of responses (Collins, Roberts, Dias, Everitt, & Robbins, 1998). A 'strategy' score is

estimated during the task, based on performance on the six and eight box levels. It is calculated from the number of times a search was initiated from the same box during a trial. Lower scores represent consistent use of this strategy, whilst high scores represent an inefficient and random search strategy (Owen, Downes, Sahakian, Polkey, & Robbins, 1990; Owen, Doyon, Petrides, & Evans, 1996).

Both the SS and SWM tasks can be applied unchanged to the study of executive function in young children where limited linguistic skills often preclude the assessment of other executive processes. For example, children as young as 4 years can perform the two tasks, although test performance correlates positively with age (Luciana & Nelson, 1998). More detailed descriptions, validity, reliability data and illustrations of the CANTAB tests can be found in Robbins et al. (1994), Fray and Robbins (1996) and at the CANTAB website (https://www.cambridgecognition.com/cantab).

### 4.5.7 Family Functioning

#### *4.5.8.1 McMaster Family Assessment Device (FAD)*

The McMaster Family Assessment Device (FAD) (Epstein et al., 1983) was designed to evaluate families according to the McMaster Model of Family Functioning, a clinically oriented conceptualization of families. It describes structural and organizational properties of the family group and the patterns of transactions among family members which have been found to distinguish between healthy and unhealthy families (Epstein et al., 1983). It is a paper and pencil questionnaire designed to collect information on the various dimensions of the family system as a whole, and to collect this information directly from family members. It can be completed by all family members over the age of twelve. It has 53 items in the questionnaire that are statements a person could make about his or her family. Each family member rates his or her agreement or disagreement with how well an item describes their families by selecting among the four alternative responses: strongly agree, agree, disagree, and strongly disagree. Scores range from 1 to 4, with 1 reflecting healthy functioning and 4 reflecting unhealthy functioning. The questionnaire takes approximately fifteen to twenty minutes to complete.

The FAD is made up of seven scales which measure *Problem Solving, Communication, Roles, Affective Responsiveness, Affective Involvement, Behaviour Control and General Functioning*. *Problem Solving* refers to the family's ability to

resolve problems at a level that maintains effective Family Functioning. *Communication* is defined as the exchange of information among family members (whether the messages are clear and directed to the person they are intended for). *Roles* focuses on whether the family has established patterns of behaviour for handling a set of family functions (provision of resources, providing nurturance and support, supporting personal development, maintaining, and managing the family systems and providing adult sexual gratification). It also measures if tasks are clearly and equitably assigned to family members and whether tasks are carried out responsibly by family members. *Affective Responsiveness* assess the extent to which individual family members are able to experience appropriate affect regarding a range of experiences. *Affective Involvement* assesses which family members are interested in and place value on each other's activities and concerns. *Behaviour Control* assesses the way in which a family expresses and maintain standards for the behaviour of its members. The seventh scale is *General Functioning* which assesses the overall health/pathology of the family.

The FAD provides a more detailed picture of families than do other available scales, because it contains seven different scales, each having acceptable reliability (Epstein et al., 1983). The FAD was developed on the responses of a sample of 503 individuals, from a group of 112 families. The General Functioning scale was developed using the most highly intercorrelated subset of items, which resulted in twelve items (one from Problem Solving, four from Communication, two from Roles, one from Affective Responses, three from Affective Involvement and one from Behaviour Control). Descriptive statistics are available for a variety of patient samples including healthy community dwelling controls. Scale scores categorize family member's responses as either satisfied with functioning in a domain or dissatisfied. The satisfaction cut-off scores (the lower the score the more dissatisfied) for each of the scales are: Problem Solving 2.20; Communication 2.20; Roles 2.30; Affective Responses 2.20; Affective Involvement 2.10; Behaviour Control 1.90; and General Functioning 2.00. Internal consistency, ranges from 0.72 to 0.83 for the subscales, and general functioning is 0.92. Correlations between the subscales range from 0.37 to 0.67. Test-Retest reliability (separated by one week) for forty-five individuals range from 0.66 to 0.76 (Miller, Epstein, Bishop, & Keitner, 1985). To test Concurrent Validity the FAD was compared with the Family Unit Inventory (FUI; an 80-item tool designed to assess a number of family dimensions) where correlations of >.5 were obtained for 6 of the 8 cases where

relationships were predicted between FAD and FUI scales (Miller et al., 1985). The FAD has good construct validity with the ability to discriminate clinical from nonclinical families (76% of the nonclinical group and 64% of the clinical group correctly predicted.

### 4.5.8 Parental Relationship Assessment

#### *4.5.8.1 Dyadic Adjustment Scale (DAS)*

The Dyadic Adjustment Scale (DAS) (Spanier, 1976) assesses parent relationship adjustment: *Dyadic Consensus* (degree to which respondent agrees with their partner); dyadic satisfaction (degree to which respondent fells satisfied with their partner); *Dyadic Cohesion* (degree to which respondent and partner participate in activities together); and *Affectional Expression* (degree to which respondent agrees with partner regarding emotional affection). It is a pencil and paper questionnaire, consisting of seven items. Individuals are asked to indicate the approximate extent of agreement or disagreement with their partner (on a scale: 5=always agree; 4=almost always agree; 3=occasionally disagree; 2=frequently disagree; 1=almost always disagree; 0=always disagree) for: philosophy of life; aims, goals, and things believed important; and amount of time spent together. They are asked to rate how often the following occurs between them and their partner: have a stimulating exchange of ideas; calmly discuss something together; or work together on a project (0=never; 1=less than once a month; 2=once or twice a month; 3=one or twice a week; 4=once a day; 5=more often (than once a day). They are also asked to rate the degree of happiness in their relationship: extremely unhappy, fairly unhappy, a little unhappy, happy, very happy, extremely happy, perfect (middle point "happy" represents the degree of happiness of most relationships). Subscale scores of 2 and below, or a total score of 13 or below, suggest poor functioning in these areas. The DAS reliability coefficient was 0.96 and it has to be shown to validly differentiate between persons who in their perception were happy in their relationship and those who were not.

### 4.5.9 Parental Psychopathology

#### *4.5.9.1 Hopkins Symptom Checklist (HSCL)*

Hopkins Symptom Checklist (HSCL) (Derogatis, Lipman, Rickels, Uhlenhuth, & Covi, 1974) is a self-report symptom inventory, comprised of 58 items which are representative of the symptom configurations commonly observed among outpatients. It

is scored on five underlying symptom dimensions – *Somatization, Obsessive Compulsive, Interpersonal Sensitivity, Anxiety and Depression* – which have been identified in repeated factor analyses. Individuals are asked to rate themselves ("how have you felt during the past seven days including today") on each symptom using a four-point scale of distress, with "not-at-all" being scored 1 and a score of 4 representing "extreme" distress. An average score equal to or greater than 1.75 (raw score 90 or over) is considered a valid cut-off value for prediction of mental distress.

*Obsessive Compulsive* items reflect symptoms that are closely identified with the clinical syndrome of Obsessive Compulsive Disorder (thoughts, impulses, and actions that are experienced as unremitting and irresistible by the individual but are of an ego-alien or unwanted nature, as well as behaviours indicative of a more general cognitive difficulty). *Interpersonal Sensitivity* items focus on feelings of personal inadequacy and inferiority, particularly in comparison to other people (self-deprecation, feelings of uneasiness, and marked discomfort during interpersonal interactions are characteristic manifestations, as are acute self-consciousness and negative expectancies regarding inter-personal communications). *Somatization* items reflects distress arising from perceptions of bodily dysfunction (complaints focused on cardiovascular, gastrointestinal, respiratory, and other systems with marked autonomic medication are included: headaches, pain and discomfort; and other somatic equivalents of anxiety are also represented). *Depression* items reflect a broad range of the symptoms of a clinical Depressive Disorder (symptoms of dysphoric mood and affect are represented as are signs of withdrawal of life interest, lack of motivation, and loss of vital energy, feelings of hopelessness and futility, as well as other cognitive and somatic correlates are also included). *Anxiety* items comprise of a set of symptoms and behaviours associated clinically with high Anxiety (restlessness, nervousness, and tension, as well as additional somatic signs, for example, trembling, free-floating anxiety, and panic attacks).

A series of studies have established the factorial invariance of the primary symptom dimensions, and substantial evidence is given in support of their construct validity. Normative data was used for both discrete symptoms and primary symptom dimensions for 2,500 subjects – 1,800 psychiatric outpatients and 700 normal. Coefficients alpha for each of the dimensions, ranging from 0.84 to 0.87 report high internal consistency. Test-retest coefficients for each dimension, based on a sample of 425 anxious outpatients, performed one week apart show high reliability (ranging

between 0.75 to 0.87). Interrater reliability for the dimensions ranges from 0.64 for Depression to 0.80 for Interpersonal Sensitivity, these coefficients are for a single rater; mean values for pairs of raters were considerably higher. Factorial invariance coefficient for somatization across three comparisons was 0.96, while that for the Obsessive-Compulsive dimension was 0.85. The mean coefficient for Interpersonal Sensitivity was 0.65, the average coefficient for Depression was 0.72 (ranging from 0.52 to 0.86), the Anxiety dimension noted as moderate. A number of studies have indicated extensive validity for the HSCL (Deane, Leathern, & Spicer, 1992; Kaaya et al., 2002).

## 4.6 PROCEDURE

The baseline assessment process involved initially gaining informed consent for participation in the follow up study from each young person's legal guardian (parent) and the young person themselves. Although not legally required, this latter process facilitated engagement of young people in the assessment process. Then the parent and young person were interviewed separately by trained registered clinical psychologists and standardised interviews, questionnaires and psychometric test measures completed. The overall time of assessment ranged from 3-4 hours, with as many rest breaks as necessary to foster engagement and minimise any distress. Most families only took one break for lunch, with the assessment process starting at 10am and finishing by 2-3pm. The parent interview involved administering the semi-structured clinical interview, the Anxiety Disorders Interview Schedule for Children (A-DISC), the Child Behavior Checklist (CBCL), the Parent Account of Childhood Symptoms (PACS) – demographic and developmental sections, the relevant Autism Spectrum Quotient (Children's or Adolescent Version), the Infancy Temperament Questionnaire, the McMaster Family Assessment Devise, the Spanier Dyadic Adjustment Scale and the Hopkins Symptom Checklist. Parents also undertook to ask their child's teacher to complete the Teachers Report Form (TRF) version of the CBCL. The interview with each young person involved administering the semi-structured clinical interview, the Anxiety Disorders Interview Schedule for Children (A-DISC). Then, after a short break Cambridge Neuropsychological Test Automated Battery (CANTAB) SWM and SS tests, and the Wide Range Achievement Test, fourth edition (WRAT 4) were completed. After another

short break, the Wechsler Intelligence Scale for Children, fourth edition (WISC 4) was completed.

On two further occasions, the parent and young person were clinically interviewed separately by two consultant child and adolescent psychiatrists, who also separately reviewed the standardised diagnostic information collected. With the completion of the baseline assessment, the clinical diagnostic appraisal and scored standardised assessment results were fed back to the referring clinician, the young person and parent together. Ongoing secondary consultation and primary consultation clinical support, if needed, was provided by the two consultant child and adolescent psychiatrists.

The clinic contacted all participants after three years had passed since their initial assessment. Those young people and families that provided further informed consent to participate in the re-assessment process were initially interviewed by a consultant child and adolescent psychiatrist. Those participants completed the same standardised assessment protocol and clinical interview with either a provisional psychologist (under the supervision of a Clinical Psychologist) or a registered Clinical Psychologists, as described above.

### 4.6.1 Group Definition: ADHD Diagnosis

An ADHD diagnosis (all presentations) was determined by consultant child and adolescent psychiatrist clinical interview, parent semi-structured clinical interview (A-DISC) and ADHD subscale T scores greater than 69 on both the Child Behaviour Checklist and Teacher's Report Form.

### 4.6.2 Group Definition: ODD Diagnosis

An ODD diagnosis was determined by consultant child and adolescent psychiatrist clinical interview, parent semi-structured clinical interview (A-DISC) and scores on both the parent and teacher forms of the Child Behaviour Checklist and Teacher's Report Form (2001) ODD subscale T scores greater than 70 (well within the clinical range). To determine the exclusion criterion of subclinical ODD features, scores on either of the parent and teacher forms of the Child Behaviour Checklist and Teacher's Report Form ODD subscale T scores lay between 59-70.

### 4.6.3 Group Diagnosis: The Absence of an ODD Diagnosis

The absence of an ODD diagnosis was determined by consultant child and adolescent psychiatrist clinical interview, parent semi-structured clinical interview (A-DISC) and scores on both the parent and teacher forms of the Child Behaviour Checklist and Teacher's Report Form (2001) ODD subscale T scores less than 59 (well within the normal range).

### 4.6.4 Dependant Measures Under Investigations

#### *4.6.4.1 Individual Child Factors*

*Temperament* was assessed by the Infancy Temperament Questionnaire (Thomas & Chess, 1977): activity; rhythmicity; approach-withdrawal; adaptability; responsiveness to new stimuli; intensity of reaction; mood; distractibility; and attention span/task persistence. If a child leans towards the low end of the scale, it could be a cause for concern.

*Intellectual functioning* was assessed by Wechsler Intelligence Scales for Children, Fourth Edition (WISC-4) (Wechsler, 2003): Verbal Comprehension abilities; Perceptual Reasoning (non-verbal) abilities; Working Memory performance; and Processing Speed ability. The three Verbal Comprehension subtests; the three Perceptual Reasoning subtests; the two Working Memory subtests; and the two Processing Speed subtests, all with a standardized mean and standard deviation of 100 and 15. The FSIQ also has a standardized mean and standard deviation of 100 and 15.

*Visual Spatial Working memory* was assessed by the CANTAB ("CANTAB for Windows," 1999): Spatial Span; and Spatial Working Memory. Spatial Span: The span score is calculated at the highest level at which the participant successfully remembers at least one sequence of boxes (range 0-9). Spatial Working Memory –Between Search Errors: are 'forgetting' errors committed when a box previously opened and emptied of a blue token is re-visited during a search with 3,4,6 or 8 boxes (range 0- ). The Strategy score is estimated from the participant's performance on the six and eight box levels. It reflects the number of times a searching sequence is initiated from the same box during a trial. A high strategy scores represents low use of this strategy and lower scores represent

more consistent use of this strategy (range 0- ). Lower scores represent consistent use of this strategy, whilst high scores represent an inefficient and random search strategy.

*Academic ability* (broadly literacy and numeracy) was ascertained using the WRAT 4 (Wilkinson & Robertson, 2006): Word Reading; Sentence Comprehension; Spelling; and Math Computation subtests. All subtests have a mean of 100 and a standard deviation of 15.

*Emotional regulation* was assessed by Child Behaviour Checklist (CBCL) and Teacher Report Form (TRF) (Achenbach & Rescorla, 2001): aggregate scores greater than and equal to 180 on the Anxiety/Depression, Attention and Aggression scales. One approach to identify Deficient Emotional Self-Regulation (DESR) in children with ADHD has been through the *Child Behaviour Checklist (CBCL)* (Achenbach & Rescorla, 2001). The CBCL, as previously outlined, is a general measure of emotional and behavioural problems in young people aged 6-18 years. It has a parent, teacher (Teacher Report Form: TRF) and child form (Youth Self Report: YSR). It consists of 118 behaviour problem items with a 3-point response format (0 = not true, 1 = somewhat or sometimes true, 2 = very true or often true). It is based on the young person's behaviour over the last six months. A particular profile of the CBCL consisting of very abnormal (2SDs) scores on the Anxiety/Depression, Aggression and Attention scales of the CBCL has been suggested to measure severe forms of dysregulated mood and behaviour (Biederman et al., 2009; Faraone, Althoff, Hudziak, Monuteaux, & Biederman, 2005). Because these scales reflect intense emotions (Anxiety/Depression scale), aggression (Aggression scale) and impulsive behaviour (Attention scale), a profile of very high scores in these scales (>210 [2SDs]) has been used as the CBCL-Dysregulation Profile by others (Althoff, Ayer, Rettew, & Hudziak, 2010; Holtmann et al., 2011; Spencer et al., 2011). Spencer et al. (2011) defined DESR if a child had an aggregate cut-off score of >180 but <210 on the Anxiety/Depression, Aggression and Attention scales of the CBCL, stating that they chose this profile due to its conceptual congruence with the clinical concept of Deficient Emotional Self-Regulation and because its extreme (>210) had previously been associated with severe forms of mood and behavioural dysregulation in children with ADHD (Spencer et al., 2011). In this study, an aggregate score of the three scales over and including 180 on the on the Anxiety/Depression, Aggression and Attention scales of the CBCL will be used. This study removed the upper limit cut off of

210 as exclusionary criteria as scores over this number still represent Deficient Emotional Self-Regulation and should be included.

*Aggression* was assessed by the Aggression scale of the Child Behaviour Checklist (CBCL) and Teacher Report Form (TRF) (Achenbach & Rescorla, 2001). Aggression problems were noted when scores were equal to or greater that 70 on the Aggression scale.

*Making and keeping friends* was assessed by the interpersonal relationships section of the Anxiety Disorders Interview for Children (A-DISC) (Silverman & Albano, 1996). Either a score of 0 "no difficulty making/keeping friends" or 1 "difficulty making/keeping friends" was recorded.

*Social problems* were assessed by the Social Problems scale of the Child Behaviour Checklist (CBCL) and Teacher Report Form (TRF) measures (Achenbach & Rescorla, 2001). The Social Problems scale on the CBCL and TRF assesses immature social behaviours as well as difficulties in peer relationships. Examples of items from this scale include: "clings to adults or too dependent," "gets teased," "not liked," "too dependent," "prefers being with younger children," and "lonely". This scale can be broadly understood as representing a negative indicator of social functioning. The large normative sample associated with the CBCL and TRF allowed for the development of "Clinical" and "Borderline" cut-offs for this scale: Scores in the top 2 percentiles (T score above 70) for the Social Problems scale are of clinical concern. Scores above the 93rd percentile (T score above 60) for the Social Problems scale are considered to be within the borderline and clinical range. Reliability and validity of the CBCL and TRF are well established (Achenbach & Rescorla, 2001).

#### *4.6.4.2 Psychosocial Factors*

*Parental level of education* was collected using the Parental Account of Childhood Symptoms, demographic and developmental sections (PACS) (Taylor et al., 1986). Scored from 1 to 7: 1 "never attended school"; 2 "some years of primary school"; 3 "primary school"; 4 "some years of secondary school"; 5 "H.S.C, or equivalent"; 6 "technical, trade or TAFE certificate"; 7 "tertiary qualifications".

*Family functioning* was assessed using the McMaster Family Assessment Device (FAD) (Epstein et al., 1983). Scale scores categorize family member's responses as either satisfied with functioning in a domain or dissatisfied. Satisfaction cut-off scores (the lower the score the more dissatisfied) for each of the scales: Problem Solving 2.20; Communication 2.20; Roles 2.30; Affective Responses 2.20; Affective Involvement 2.10; Behaviour Control 1.90; and General Functioning 2.00.

*Parental relationship* was ascertained from The Dyadic Adjustment Scale (DAS) (Spanier, 1976). Subscale scores of 2 and below, or a total score of 13 or below, suggest poor functioning in these areas.

*Parenting skill* was assessed using the McMaster Family Assessment Device (FAD) (Epstein et al., 1983): *Behaviour Control* subtest. One of the seven scales of the McMaster Family Assessment Device (FAD) (Epstein et al., 1983), *Behaviour Control* assesses the way in which a family expresses and maintains standards for the behaviour of its members. It measures behaviour in situations of different sorts (dangerous, psychological, and social), as well as different patterns of control (flexible, rigid, laissez-faire and chaotic). There are nine questions in this scale that includes questions such as: "you can easily get away with breaking the rules"; "we have rules about hitting people"; "we don't hold any rules or standards"; "if the rules are broken, we don't know what to expect"; "anything goes in our family"; and "there are rules about dangerous situations". The family member rates his or her agreement or disagreement with how well an item describes their families by selecting among the four alternative responses: strongly agree, agree, disagree, and strongly disagree. This scale was used to measure parenting skill for this study. Scale scores categorize family member's responses as either satisfied with functioning in a domain or dissatisfied. Satisfaction cut-off scores (the lower the score the more dissatisfied) for Behaviour Control subtest below 1.90 is considered problematic.

*Parental psychopathology* was assessed by the Hopkins Symptom Checklist (HSCL) (Derogatis et al., 1974): Obsessive Compulsive; Interpersonal Sensitivity; Anxiety; and Depression subtests. An average score equal to or greater than 1.75 (raw score 90 and over) is considered a valid cut-off value for prediction of mental distress.

#### *4.6.4.3 Comorbid Conditions*

*CD diagnosis; Depressive Disorder diagnoses; and Anxiety Disorder diagnoses* were determined by the parent semi-structured clinical interview (A-DISC). The consultant child and adolescent psychiatrist, using the scoring manual, determined if DSM criteria had been met: 0 “not met criteria”; 1 “met criteria”.

*ASD* diagnosis was determined by either the child or adolescent Autism Spectrum Quotient (Auyeung et al., 2008; Simon Baron-Cohen et al., 2006). The consultant child and adolescent psychiatrist using a cut off score above 76 on the AQ-Child and 32 on the AQ-Adolescent were used to determine ASD diagnosis.

## 4.7 STATISTICAL ANALYSIS

Data analyses were performed using the *Statistical Package for the Social Sciences (SPSS/PC)*. Descriptive statistics of the group of children with ADHD and follow-up ODD and the group of children with ADHD alone at follow up are presented. Mean (Standard Deviation - SD) values of dependent and independent variables are noted, once the effects of outliers had been removed. All these variables were normally distributed. Statistical significance for group differences were set at $p < 0.05$ level, Bonferroni corrections were applied to specific multiple comparisons of related independent variables when they arose from the same source of assessment (for example, subscales of a given test). They were not applied when type II errors for given independent variables (for example, mental health disorder diagnoses) were an unacceptable risk, given the clinical and research significance of the related independent variables. Separate logistic regression analyses were conducted for all categorical and continuous dependent variables examined. Variables that significantly predicted group membership of the ADHD and follow-up ODD or ADHD without follow-up ODD groups are in bold type. Clinical significance is noted by (C) and borderline/at risk variables noted by an *. Backwards stepwise logistical regressions were completed for all significant [1] child characteristic factors, [2] psychosocial factors, and [3] ADHD combined type and significant comorbidities to identify the variables that made the best predicted membership of the ADHD and follow-up ODD group.

## 4.8 ETHICAL ISSUES

The Human Research and Ethics Committee, Royal Children's Hospital (RCH), approved all the studies presented in this thesis, as part of a larger ongoing clinical research program: HREC REF. No: 35068 A. Parent(s) of each child in the study and each child themselves provided "informed consent" after verbal discussion and signed written consent as approved by Ethics Committee. The rights of each child and their parent(s) to withdraw from the assessment process at any time without explanation and without any risk of interference in their condition's management were emphasised. "Confidentiality" was also assured. The information collected from each child and their parent(s) was not reported in any way that could identify them and individual children's details will not be outlined in any written and verbal reports that result from these studies.

# CHAPTER 5: Key Child Characteristics of ADHD and Follow Up ODD (Study One)

## 5.1 INTRODUCTION

Children with ADHD often have difficulties with exercising self-control, struggle to comply with adults' instructions and are often labelled as "problem children" and "rule breakers" (Milich & Roberts, 2020; Mischel, Shoda, & Rodriguez, 1989). It is important to be able to distinguish between typical behaviour of children with ADHD and when these behaviours start to become a significant issue requiring further assessment, diagnosis and treatment for Disruptive Behaviour Disorders, such as ODD. As seen in the ODD literature, prevention, and early intervention lead to better outcomes for these children. Therefore, the importance of being able to prevent ODD from developing in children with ADHD is desired. The identification of specific child characteristics that could be risk and protective factors for young people with ADHD developing ODD may be helpful in assisting to target these young people for specific early intervention. This chapter begins with a summary of the predominant clinical child characteristics associated with ODD at follow up in children diagnosed with ADHD: gender; temperament; intelligence; Visual Spatial Working Memory; academic ability; emotional regulation; and social problems. This study will look at these child factors in young people with ADHD alone and ADHD with ODD at follow up to help us explore the risk and protective factors in developing ODD. Results from study one, looking at the key child characteristics of ADHD and follow up ODD will be reported, and the clinical relevance, limitations and future research directions of these findings discussed.

There is a known greater than chance association of ADHD and ODD (Angold et al., 1999; Biederman, Faraone, Milberger, Jetton, et al., 1996). Children and adolescents who have ADHD and ODD are typically more aggressive, have more negative behavioural symptoms of ODD, underachieve more severely in the academic domain, and have greater social difficulties (AACAP, 2009; Carlson et al., 1997; Steiner et al., 2007). While ADHD usually first presents in childhood and persists into adulthood (Biederman & Faraone, 2005), ODD can emerge during different developmental stages and may or may not persist over time (Nock et al., 2007). This is relevant to the development of comorbid ADHD and ODD, as which developmental stage it emerges in may have a different pattern of symptom course, related impairment, treatment response and prognosis (Hudec & Mikami, 2018). This study will investigate children with ADHD followed-up after three years.

### 5.1.1 Gender

Research for both ADHD and ODD consistently report they are typically more prevalent in boys than girls (Angold & Costello, 1996; Quy & Stringaris, 2012; Steiner et al., 2007; Zoccolillo, 1993). However, some studies have shown no gender differences (Lahey et al., 2000; Nock et al., 2007). Most studies looking at comorbid ADHD and ODD have focused on boys; only a few studies have examined this comorbidity in girls, who have been found to have more severe and persistent behavioural problems and are more likely to have additional co-morbid mood disorders and substance abuse (Connor et al., 2010; Steiner et al., 2007). In relation to comorbid ADHD and ODD, different point prevalence rates have consistently been noted between boys and girls (Biederman, Petty, Monuteaux, et al., 2008; Carlson et al., 1997; Munkvold et al., 2011; Trepat & Ezpeleta, 2011). This follow-up study will examine boys and girls with ADHD.

### 5.1.2 Socio-Economic Status

Literature reviews have well documented the relationship between poverty and low SES to a range of negative child outcomes (McLloyd, 1998; McLoyd, 1989). Lower SES has been linked with higher levels of emotional and behavioural difficulties, including social problems, Disruptive Behaviour Disorders and ADHD among young people (Russell, Ford, Williams, & Russell, 2016; Santiago, Wadsworth, & Stump, 2011). Researchers have argued that social factors such as poverty and income inequality are fundamental risk factors for mental health disorders because they limit access to important health-promoting resources (Drukker, Kaplan, Feron, & van Os, 2003). The aspects of SES commonly looked at are: parental educational attainment; family income; relative deprivation; subjective social status; and community level of inequality. However, low SES has been defined in numerous ways making it difficult to compare results across indicators and making it impossible to know which of the several components of SES account for the overall association between low SES and mental health disorders (McLaughlin, Costello, Leblanc, Sampson, & Kessler, 2012). Socio economic status will be examined in this study using the following factors: family income level; mother's educational level; single parent status; sibling size; and broken home status (the Social Adversity Scale (SAS), PACS) (Taylor et al., 1996).

### 5.1.3 Temperament

Temperament and psychopathology have been shown to correlate consistently across childhood (De Pauw & Mervielde, 2010; Nigg, 2006). Certain temperament traits, in particular the anger component of negative emotionality (broad temperamental factor including fear, anxiety and sadness, as well as a tendency to become emotionally upset in the face of frustration), as well as effortful control (the focusing and shifting of attention, inhibitory control, perceptual sensitivity, and a low threshold for pleasure) are associated with ADHD (De Pauw & Mervielde, 2011; Eisenberg et al., 2005; Foley et al., 2008; Martel & Nigg, 2006). ODD has been reported to have temperamental profiles including: high negative emotionality and activity (anger/frustration); intensive and reactive responding; inflexibility; high novelty seeking; low persistence, and low effortful control (Lavigne et al., 2012; Melegari et al., 2015; Posne & Rothbart, 2000; Rabinovitz et al., 2016; Rothbart, 2007; Sanson & Prior, 1999; Stringaris et al., 2010). Very few studies have looked at the temperament of young people with comorbid ADHD and ODD. Three temperament characteristics have been found common in young people with comorbid ADHD and ODD: high levels of activity; decreased levels of persistence; and decreased levels of self-directedness (Stringaris et al., 2010). Further longitudinal research is required in the area of temperament difficulties as it may be a risk factor for developing ODD in young people with ADHD. This will be undertaken in this study.

### 5.1.4 Intelligence

Intelligence is potentially one of a range of significant characteristics that could predict individuals having increased social and/or behavioural difficulties in the future. Understanding the cognitive profiles of young people with ADHD and ODD is difficult as there are limited studies in this area, and many studies have combined ODD and CD into a single entity. When compared to typically developing control participants, research has found ADHD, CD/ODD, and ADHD+CD/ODD had lower FSIQ scores, suggesting that those with comorbid ADHD and ODD/CD did not have greater intellectual impairments than those with ADHD or ODD/CD alone (Dolan & Lennox, 2013; Jensen et al., 2001; Matthys et al., 1999). Some researchers argue that if a young person with comorbid ADHD and ODD has intellectual deficits, they are better explained

by ADHD. Speltz et al. (1999) studied a group of preschool clinic referred boys with ADHD and ODD finding significant deficits in verbal IQ compared to a matched comparison group, highlighting the importance to look at the different subtests of intelligence separately. This study will do this.

### 5.1.5 Visual Spatial Working Memory

Working Memory is one component of Executive Functioning that is important for learning and academic performance. Studies have revealed that Working Memory in children with ODD may be compromised, contributing to their antisocial and aggressive behaviour. Several studies have found that boys with ODD showed impairments in Working Memory (Séguin et al., 2007; Syngelaki et al., 2009; Xu et al., 2017). Although Working Memory deficits in young people with ODD have been found, to what extent they exist independently of ADHD comorbidity is controversial (Dolan & Lennox, 2013; Morgan & Lilienfeld, 2000; Pennington & Ozonoff, 1996). Some studies have found that impairments are more pronounced in those with ADHD and comorbid ODD than ADHD alone (Dolan & Lennox, 2013). Some studies have controlled for ADHD and not found impairments in Working Memory for young people with ODD (Dolan & Lennox, 2013; Fairchild et al., 2009; Oosterlaan et al., 2005; Thorell & Wåhlstedt, 2006), while others have found impairments (Saarinen et al., 2015; Schoorl et al., 2018; Syngelaki et al., 2009). Lin and Gau (2017) found young people with ADHD and ODD had deficits in verbal memory and response inhibition, compared to those with ADHD only. Rhodes et al. (2012) found that boys with ODD demonstrated impaired memory performance in the areas of: verbal memory tasks; spatial memory tasks; on the storage and central executive working memory factors; and the long-term memory factor. They suggested that verbal memory difficulties were more closely associated with ODD than ADHD symptoms. Further longitudinal research is required in this area as Visual Spatial Working Memory deficits maybe a risk factor for ODD in young people with ADHD. This will be undertaken in this study.

### 5.1.6 Academic Abilities

Academic achievement is an important predictor of SES later in life, and wellbeing and health in adulthood (Huisman et al., 2005). It is known that the severity of

ADHD symptoms is correlated with a reduced level of academic attainment and increased rates of school rejection. Children with ADHD have a greater than chance association with spelling, reading and mathematics learning difficulties. Research also indicates that young people with school difficulties, including low academic achievement and frequent truancy, are at very high risk for Disruptive Behaviour Disorders (Janosz et al., 2000). The literature in ADHD and ODD is limited, most studies historically reporting on Disruptive Behaviour Disorders together (ODD and CD). The impact of comorbid ADHD and ODD on academic achievement has shown varied results in the literature: some studies suggest comorbidity is associated with worse academic outcomes; some suggest that ADHD only is associated with worse academic achievement, while others demonstrate no difference. Further research is required to understand the impact of a young person's academic performance for comorbid developing ADHD and ODD. This will be examined in this study.

### 5.1.7 Emotional Regulation and Aggression

Emotional regulation is an important characteristic for human beings to develop, as it is crucial for long-term wellbeing, good personal relationships, and successful performance at work. Research has found a strong association between ADHD alone, ODD alone, and emotion dysregulation (Becker et al., 2006; Cole et al., 1994; Gilliom et al., 2002; Sjöwall et al., 2013; Stringaris & Goodman, 2009). The long term prognosis of people with ADHD and emotional regulation difficulties is poor and predicts worse psychosocial functioning and associated clinically significant impairments (Diler et al., 2007; Faraone et al., 2019; Peyre, Speranza, Cortese, Wohl, & Purper-Ouakil, 2015). Biederman, Spencer, et al. (2012) found that emotional dysregulation in children with ADHD was associated four years later with more psychiatric comorbidities, greater social impairment and ADHD persistence, compared to individuals with ADHD without emotion dysregulation. There is limited longitudinal research investigating emotional regulation deficits in comorbid ADHD and ODD (without CD). Some research suggests that emotion regulation problems may be more linked to ODD than ADHD symptoms (Schoorl et al., 2016; Sobanski et al., 2010). Bunford et al. (2018) found that young people with ADHD did have emotion dysregulation difficulties, and that it did not differ as a function of ADHD subtype or comorbid ODD. Further longitudinal research in ADHD and ODD is required to understand the impact of the comorbidity on emotional regulation

of children and adolescents: emotional regulation deficits in young people with ADHD may be a risk factor for developing ODD. This will be undertaken in this study.

Children with disruptive behaviour disorders have also been found to be more: hostile towards peers; to interpret others' behaviours as more hostile (for example, people are out to get me, people will treat me badly); hypervigilant to cues of threat or hostility; and to miss out on benign or reconciliatory cues than typically developing children (Crick & Dodge, 1994; Muris, 2006; Schniering & Rapee, 2004). Disorders with increased aggressive behaviour carry an increased risk for long-lasting negative impact on well-being and are one of the commonest reasons for parents to seek child and adolescent psychiatric treatment (Koelch, Döpfner, Freitag, Dulz, & Rösler, 2019). Hawes, Kimonis, Mendoza Diaz, Frick, and Dadds (2020) found an association between teacher reported aggression and ODD/CD. Aggression will be included as a factor in this longitudinal study as it may be an early predictor for the development of ODD and there is limited research in the area of ODD separate to CD.

### 5.1.8 Social Problems

Better social competence is related to peer acceptance, emotional health, school readiness, successful interpersonal relationships, and social adjustment. Deficits in social competence lead to negative social interactions that can impact on relationships, lead to early school leaving and vocational problems in adulthood. Children with deficits in social competence have been found to have reduced empathy for others, are less cooperative, have poorer conflict resolution skills, and display negative behaviour and problems in their social interaction skills (Chen & Jiang, 2002; Gouley et al., 2008). Young people with ADHD have a greater risk for social competence deficits compared to typically developing children (Barkley, DuPaul, et al., 1990; Biederman, Fried, et al., 2012; Hoza & McQuade, 2015). In general, most researchers have found that children with Disruptive Behaviour Disorders also have poorer social relationships than non-disruptive peers, seen in conflicting interactions and frequent peer rejection, which continue into their adolescence (Campbell, 1990; Emond et al., 2007; Oliver et al., 2011; Pope et al., 1989; Vitaro et al., 2001; Yoon et al., 2000). When ADHD is comorbid with ODD it has been reported to be associated with greater social impairment than ADHD alone. However, Becker, Langberg, et al. (2012), after undertaking a literature review

looking at ADHD and co-occurring externalizing symptoms, concluded that the problems in peer relations, friendship and social skill/competence domains among youth with ADHD are not wholly attributable to co-occurring behaviour problems**,** with results differing depending upon the rater (parent verses teacher). Assessing both peer functioning (making and keeping friends) and broad level social problems are important to successful social functioning (Ladd, 2005; Parker & Asher, 1993) and both will be investigated in this study. It has been difficult to gain a clear picture of the risk factors for comorbid ADHD and ODD and the impact of the comorbidity on young people's social problems due to limited research in the area, specifically looking at ODD separate from CD, highlighting the importance of this study.

## 5.2 AIMS AND HYPOTHESES

The aim of study one is to examine the key clinical risk and protective *child* associated factors (gender, temperament, IQ, Visual Spatial Working Memory, academic ability, emotional regulation, and social problems) for young people with ADHD and follow-up ODD compared to ADHD alone. The hypothesis to be tested is that young people with follow-up ODD and ADHD compared to those diagnosed with ADHD without ODD will manifest differential key child characteristics including: have increased boys; have increased levels of temperament activity; have a decreased level of task persistence and attention span; lower verbal IQ; worse Visual Spatial Working Memory performance; experience academic underachievement; have poorer emotional regulation abilities; have increased levels of aggression; and increased difficulties in keeping friends and over all social difficulties.

## 5.3 METHOD

The majority of the methods for this Study (one) are described in Chapter four.

Data analyses were performed using the *Statistical Package for the Social Sciences (SPSS/PC)*. Descriptive statistics of the group of children with ADHD and follow-up ODD and the group of children with ADHD alone at follow up are presented.

Mean (Standard Deviation - SD) values of dependent and independent variables are noted, once the effects of outliers had been removed. Statistical significance for group differences were set at $p < 0.05$ level, with Bonferroni corrections applied for multiple comparisons, when appropriate. Separate logistic regression analyses were conducted for all categorical and continuous dependent variables examined. Variables that significantly predicted group membership of the ADHD and follow-up ODD or ADHD without follow-up ODD groups are in bold type. Variables that met clinical significance is identified by (C) and those in that "at risk" range identified by a *. In addition, performance on the Visual Spatial Working Memory task between search errors score was compared with a repeated measures Analysis of Variance (ANOVA) using a two-factor design which included a between-subjects factor (group) and a within subjects factor (for example, difficulty level). To protect against violation of the homogeneity of covariance assumption, analysis of within-group effects or interactions with degrees of freedom greater than one were examined using a repeated measures design within MANOVA (Wilks' Multivariate test of significance). Performance on the Spatial Span task and the Spatial Working Memory strategy measure were compared between groups using one-way ANOVA. To investigate the relationship between short-term memory (span) and Spatial Working Memory, Pearson's product moment correlation coefficients were employed.

## 5.4 RESULTS

### 5.4.1 Demographic Characteristics

Table 1 presents the age (Mean (Standard deviation)), gender (number) and Social Adversity Status (Mean (Standard deviation)) for ADHD with and without follow up ODD groups. The two groups did not differ for age, gender, or Social Adversity Status at baseline. The two groups did not differ for ADHD severity. Table 1 also presents the age at follow up (Mean (Standard deviation)), which did differ at follow up, with the ADHD and follow up ODD group being younger in comparison to the ADHD alone group. This was a small effect, decreased age being more likely to predict ADHD and follow-up group membership.

**Table 1: Age (baseline and follow up), gender and Social Adversity Status in the ADHD and follow-up ODD and ADHD alone groups**

| | +ODD | -ODD | WALD | Df | P | OR | (95%CI) |
|---|---|---|---|---|---|---|---|
| **Age at base line** | 8.69 (2.56) | 9.33 (2.98) | 2.45 | 1 | 0.12 | 0.92 | (0.82-1.02) |
| **Age follow up** | **11.1 (2.54)** | **12.5 (3.32)** | **9.19** | **1** | **0.002** | **1.19** | **(1.06-1.33)** |
| **Gender** | Male 120 Female 30 | Male 43 Female 17 | 1.71 | 1 | 0.19 | 1.58 | (0.79-3.15) |
| **SAS** | 8.48 (2.14) | 7.89 (1.94) | 2.99 | 1 | 0.08 | 1.16 | (0.98-1.36) |

Note: +ODD: ADHD with follow up ODD group; -ODD: ADHD without ODD group; SAS: Social Adversity Status

### 5.4.2 Temperament

The ADHD and follow up ODD group demonstrated an increased temperament activity level compared to the ADHD without ODD group (see Table 2). There were no differences between the two groups in rhythmicity, approach-withdrawal, adaptability, responsiveness, intensity of reaction, mood, distractibility and task persistence and attention span. Table 2 presents the temperament factors (Mean (Standard Deviation)) for the ADHD and ODD and ADHD alone groups at follow up. This was a clinically significant effect, increased temperament activity being more likely to predict ADHD and follow-up ODD group membership compared to ADHD without follow up ODD.

**Table 2: Temperament factors in the ADHD and follow up ODD group membership (n=150) compared to ADHD alone group at follow up (n=60)**

| Temperament dimension | +ODD | -ODD | Wald | Df | p | OR | (95%CI) |
|---|---|---|---|---|---|---|---|
| **Activity level** | **1.05(.66)** | **1.47(.59)** | **11.47** | **1** | **0.001** | **2.97** | **(1.58-5.56)** |
| **Rhythmicity** | 0.98(.72) | 1.06(.77) | 0.32 | 1 | 0.57 | 1.15 | (0.714-1.88) |
| **Approach-withdrawal** | 1.00(.69) | 0.79(.73) | 2.43 | 1 | 0.12 | 0.66 | (0.40-1.11) |
| **Adaptability** | 1.10(.73) | 0.92(.78) | 1.46 | 1 | 0.23 | 0.74 | (0.46-1.21) |
| **Responsiveness** | 0.40(.63) | 0.48(.67) | 0.35 | 1 | 0.55 | 1.19 | (0.67-2.12) |
| **Intensity of reaction** | 1.31(.60) | 1.20(.71) | 0.76 | 1 | 0.39 | 0.78 | (0.45-1.36) |
| **Mood** | 0.45(.50) | 0.66(.70) | 2.86 | 1 | 0.09 | 1.68 | (0.92-3.08) |
| **Distractibility** | 0.74(.67) | 1.03(.75) | 4.52 | 1 | 0.03 | 1.76 | (0.15-2.97) |
| **Persistence Attention span** | 1.29(.71) | 1.51(.63) | 3.18 | 1 | 0.08 | 1.66 | (0.95-2.88) |

Note: +ODD: ADHD with follow up ODD group; -ODD: ADHD without ODD group

### 5.4.3 Intelligence

Table 3 presents the baseline intelligence subtests and full-scale scores (Mean (Standard deviation)) for the ADHD and ODD and ADHD alone groups at follow up. Only Verbal Comprehension differed in the ADHD and ODD group at follow up, with this group showing a significantly lower verbal comprehension ability compared to the ADHD alone group. There were no differences between the two groups in Perceptual Reasoning, Working Memory, Processing Speed or Full-Scale IQ. This was a very small effect; lower Verbal Comprehension index score only being more likely to predict ADHD and follow-up ODD group membership compared to ADHD alone group.

**Table 3: Intelligence subtests and full-scale IQ predicting ADHD and follow up ODD group membership (n=150) compared to ADHD alone group at follow up (n=60)**

| IQ index | +ODD | -ODD Mean | Wald | Df | p | OR | (95%CI) |
|---|---|---|---|---|---|---|---|
| **VCI** | **89.83 (15.83)** | **96.24 (14.92)** | **6.11** | **1** | **0.01** | **1.03** | **(1.01-1.05)** |
| **PRI** | 92.91 (14.39) | 94.13 (14.89) | 0.27 | 1 | 0.60 | 0.99 | (0.97-1.02) |
| **WMI** | 89.32 (16.54) | 88.85 (14.22) | 0.02 | 1 | 0.88 | 1.00 | (0.98-1.03) |
| **PSI** | 86.9 (13.93) | 86.9 (17.26) | 0.00 | 1 | 0.99 | 1.00 | (0.98-1.03) |
| **FSIQ** | 88.52 (14.81) | 92.79 (15.01) | 3.02 | 1 | 0.08 | 0.98 | (0.96-1.00) |

Note: +ODD: ADHD with follow up ODD group; -ODD: ADHD without ODD group; VCI: Verbal Comprehension Index (WISC); PRI: Perceptual Reasoning Index (WISC); WMI: Working Memory Index (WISC); PSI: Processing Speed Index (WISC); FSIQ: Full Scale IQ (WISC)

### 5.4.4 Academic Abilities

Baseline academic achievement abilities (reading, spelling, and mathematics) did not differ between the ADHD and ODD group and ADHD without ODD group at follow up (See Table 4). Table 4 presents the academic factors (Mean (Standard deviation)) between the ADHD and ODD and ADHD alone groups at follow up.

**Table 4: Academic abilities in the ADHD and follow up ODD group membership (n-150) compared to ADHD alone group at follow up (n=60)**

| Academic ability | +ODD | -ODD | Wald | df | p | OR | (95%CI) |
|---|---|---|---|---|---|---|---|
| **Reading** | 95.17 (18.51) | 96.38 (16.89) | 0.13 | 1 | 0.72 | 0.99 | (0.98-1.02) |
| **Spelling** | 91.34 (15.15) | 94.18 (15.15) | 1.02 | 1 | 0.31 | 0.99 | (0.97-1.01) |
| **Mathematics** | 83.13 (17.10) | 87.49 (17.47) | 2.35 | 1 | 0.13 | 0.99 | (0.97-1.01) |

Note: +ODD: ADHD with follow up ODD group; -ODD: ADHD without ODD group

### 5.4.5 Visual Spatial Working Memory

Table 5 presents the key baseline Visual Spatial Working Memory factors (Mean (Standard Deviation)) between the ADHD and ODD and ADHD alone groups at follow up.

**Table 5: Visual spatial working memory in the ADHD and follow up ODD group membership (n=150) compared to ADHD alone group at follow up (n=60)**

| Visual Spatial Working Memory | +ODD | -ODD | Wald | df | p | OR | (95%CI) |
|---|---|---|---|---|---|---|---|
| **Spatial Span** | 4.35 (1.36) | 4.76 (1.45) | 2.96 | 1 | 0.09 | 1.23 | (0.97-1.57) |
| **Strategy** | 36.92 (4.56) | 36.64 (3.89) | 0.15 | 1 | 0.70 | 1.01 | (0.94-1.09) |
| **Between search errors** | **56.52 (18.28)** | **50.20 (19.74)** | **4.03** | **1** | **0.05** | **1.02** | **(1.00-1.04)** |

Note: +ODD: ADHD with follow up ODD group; -ODD: ADHD without ODD group

First, the Visual Spatial Working Memory Between Search Errors (BSE) total score was significantly lower in the ADHD alone group compared to the ADHD and follow up ODD group. This finding suggests that the ADHD and follow up ODD group has a worse Visual Spatial Working Memory performance overall than the ADHD alone group. This was a small effect, worse BSE being more likely to predict ADHD and follow-up ODD group membership compared to ADHD alone group.

Second the Visual Spatial Working Memory BSE Strategy score was not significantly different in the ADHD and follow up ODD group compared to the ADHD alone group. This result implies that the two groups did not differ with respect their efficient, planned, and organised approach to the Visual Spatial Working Memory search strategy.

Third the Spatial Span score did not differ between the two groups, nor did the total time taken to complete the Visual Spatial Working Memory task, although there was a non-significant trend for Spatial Span to be better in the ADHD alone group. Pearson product moment correlation coefficients revealed that the Visual Spatial Working Memory BSE total score had a large positive correlation with the Visual Spatial Working Memory BSE Strategy score ($r$=0.63, $p$ <.0005) and a large negative correlation with the Spatial Span score ($r$=-0.61, $p$ <.0005). Also, the Visual Spatial Working Memory BSE Strategy score had a medium strength negative correlation with the Spatial Span score ($r$=-0.37, $p$ <.0005).

These findings suggested that Visual Spatial Working Memory overall performance is similarly dependent on both the Strategy (organising) and Span (capacitance) components of Working Memory. In addition, these results imply that the better overall Visual Spatial Working Memory performance in the ADHD alone group may be more related to a better Spatial Span performance, given that Strategy (organising planning) did not differ between the two groups and the non-significant trend for Spatial Span to be better in the ADHD alone group. Consistent with this, Figure 1 shows the mean between-search errors for each group according to the difficulty level of the task. There was a significant effect of group [F (1,182) = 4.54, $p$ = .03] and task difficulty [Wilks' $\lambda$ = 0.12, F (3,180) = 450.23, $p$ < .0005], but no interaction between the group and difficulty factors [Wilks' $\lambda$= 0.98, F (3,180) = 1.54, $p$ = .21]. Investigation of the group effect showed that, when arranged across level of difficulty, the ADHD and follow up ODD group made significantly more BSE than the ADHD alone group.

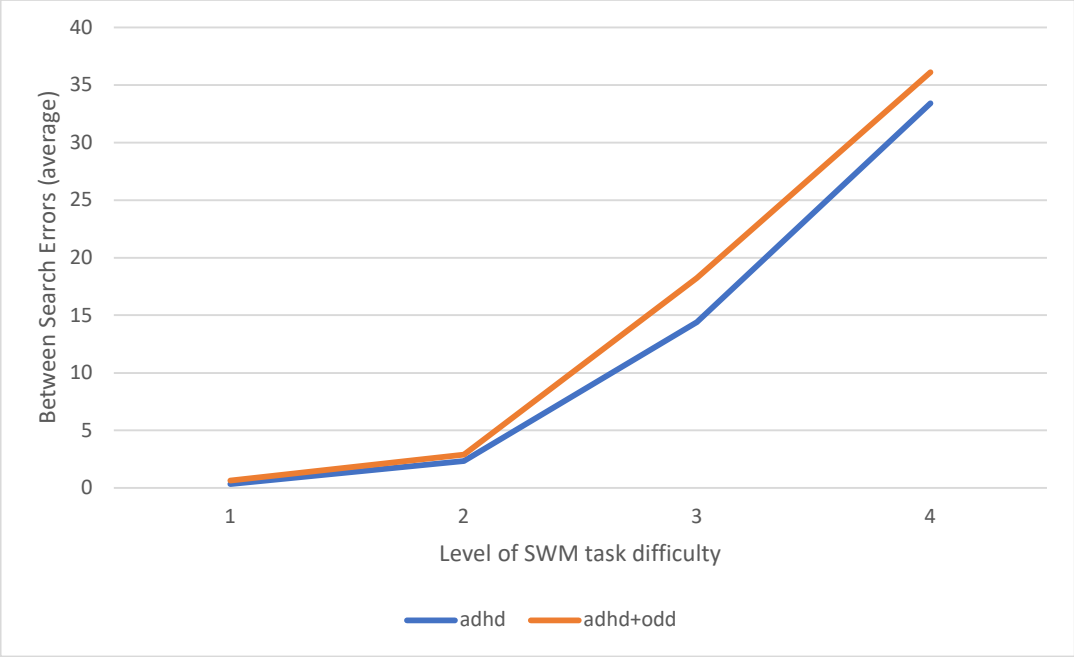


Figure 1. *Mean number of 'between-search' errors at each level of difficulty on the visual spatial working memory task: 1=BSE 3 boxes, 2=BSE 4 boxes, 3=BSE 6 boxes, 4=BSE 8 boxes*

### 5.4.6 Emotional Regulation and Aggression

The ADHD and ODD at follow up group demonstrated worse Emotional Regulation Difficulties (DESR) than the ADHD alone group as reported by parents (see Table 6). There were no differences between the groups for emotional regulation difficulties as reported by teachers (TRF DESR). Table 6 presents DESR (Mean (Standard Deviation)) for the ADHD and ODD and ADHD alone groups at follow up as reported by parents and teachers. Only parent reported DESR were found to be more likely to predict ADHD and follow-up ODD group membership compared to ADHD without follow up ODD. However clinically: parents of children in both groups identified that the majority of children had DESR problems (97% in the ADHD and ODD at follow up group and 80% in the ADHD alone group); teachers reporting about half of the children in both groups having DESR problems (62% in the ADHD and ODD at follow up group and 46% in the ADHD alone group).

The ADHD and follow up ODD group demonstrated increased aggression compared to the ADHD alone group as reported by both parents and teachers (see Table 6). This was a small effect, more aggression (both teacher and parent reported) being more likely to predict ADHD and follow-up ODD group membership compared to ADHD without follow up ODD. Clinically, both parents and teachers reported aggression difficulties for the ADHD and ODD group (parents identifying difficulties in the clinical range and teachers identifying difficulties in the “at risk” range). For the ADHD alone group, only parents reported aggressive difficulties in the “at risk” range.

**Table 6: Deficient Emotional Self-Regulation (DESR) and Aggression in the ADHD and follow up ODD group membership (n=150) compared to ADHD alone group at follow up**

| Emotional regulation | +ODD | -ODD | Wald | df | P | OR | (95%CI) |
|---|---|---|---|---|---|---|---|
| **CBCL DESR** | **97% difficulties** | **80% difficulties** | **13.44** | **1** | **<0.0005** | **9.06** | **(2.79-29.46)** |
| **TRF DESR** | 62% difficulties | 46% difficulties | 2.88 | 1 | 0.09 | 1.94 | (0.90-4.17) |
| **CBCL aggressive scale** | **80.26 (10.48) (C)** | **66.41 (11.96) *** | **39.13** | **1** | **<0.0005** | **1.12** | **(1.08-1.15)** |
| **TRF aggressive scale** | **69.57 (14.35) *** | **59.24 (8.73)** | **13.01** | **1** | **<0.0005** | **1.08** | **(1.03-1.12)** |

Note: DESR: Deficient Emotional Self Regulation; +ODD: ADHD with follow up ODD group; -ODD: ADHD without ODD group; difficulties: score including and over 180 DESR; CBCL: Child Behavior Checklist; TRF: Teacher Report Form; (C): Clinical range for aggression; *At-risk range for aggression

### 5.4.7 Social Problems

The ADHD and follow up ODD group demonstrated worse social competence as reported by both parents and teachers on the social problems scale. This was a small effect, worse social problems (both parent and teacher reported) being more likely to predict ADHD and follow-up ODD group membership compared to ADHD without follow up ODD. There were no differences between the two groups in making friends and keeping friends.

Clinically, both parents and teachers, identified that the children in the ADHD with ODD group at follow up displayed social problems in the "at risk" range of difficulties. Parents of the children in the ADHD alone group rated these children also in the "at risk" range of difficulties.

Table 7 presents the social competence subscales (Mean (Standard Deviation)) for the ADHD and ODD and ADHD alone groups at follow up.

**Table 7: Social problems in the ADHD and follow up ODD group membership (n=150) compared to ADHD alone group at follow up (n=60)**

| Social problems | +ODD | -ODD | Wald | Df | p | OR | (95%CI) |
|---|---|---|---|---|---|---|---|
| **Making friends – parent** | 19% difficulties | 37% difficulties | 2.79 | 1 | 0.09 | 2.48 | (0.85-7.19) |
| **Keeping friends - parent** | 48% difficulties | 27% difficulties | 3.28 | 1 | 0.07 | 1.62 | (0.96-2.71) |
| **CBCL social problems** | **69.62 (10.41) *** | **66.25 (11.32) *** | **4.04** | **1** | **0.04** | **1.03** | **(1.00-1.06)** |
| **TRF social problems** | **64.76 (9.43) *** | **59.92 (7.77)** | **7.05** | **1** | **0.008** | **1.07** | **(1.02-1.12)** |

Note: difficulties: making and keeping friends difficulties reported; +ODD: ADHD with follow up ODD group; -ODD: ADHD without ODD group; CBCL: Child Behavior Checklist; TRF: Teacher Report Form; *At-risk range for social problems

## 5.5 DISCUSSION

### 5.5.1 Age

Age did not differ at base line between the ADHD and follow up ODD and the ADHD alone groups, and therefore age was not subsequently controlled when examining the different factors analysed in this study. Nevertheless, given that ODD can emerge during different developmental stages and may or may not persist over time (Nock et al., 2007), it remains a limitation for this study. The majority of the young people in the study were in the childhood developmental stage and this needs to be taken into consideration when interpreting the findings: different developmental stages may have a different pattern of symptom course, related impairment, and prognosis (Hudec & Mikami, 2018). It is important for future longitudinal studies to examine adolescents who develop ODD as a "late starter" (little oppositional behaviour during childhood, but the behaviours become more observable during adolescence) as the risk and protective factors may differ for this group.

### 5.5.2 Gender

Gender did not significantly differ between the ADHD and follow up ODD and ADHD without ODD groups, and therefore gender was not controlled in this study. Interestingly, there was a higher number of males compared to females in the study which may reflect the higher prevalence rate for boys (62 to 68.7%) compared to girls (28.6 to 71%), noted in the literature (Biederman, Petty, Monuteaux, et al., 2008; Carlson et al., 1997; Munkvold et al., 2011; Trepat & Ezpeleta, 2011). Some researchers propose that different ODD criteria should be used to assess girls. They point out that before adolescence, girls may exhibit aggression in ways that are not captured by the current definitions (for example, they tend to be less overtly aggressive and more covertly aggressive) (Steiner et al., 2007; Zoccolillo, 1993). Given most of the young people in this study were pre-adolescence, this is a limitation for this study. Costello et al. (2003) found a much stronger link between ODD and ADHD among girls than boys and the longitudinal link of ADHD predicting the onset of ODD was found only in females. Future longitudinal research is required over different developmental stages to investigate the association of gender to ADHD and ODD.

### 5.5.3 Socio-Economic Status

There was no significant difference found on the Social Adversity Scale (SAS) between the ADHD and follow up ODD and ADHD without ODD groups. Therefore, SAS was not controlled in this study. This is consistent with the literature that has linked lower SES with higher levels of both Disruptive Behaviour Disorders and ADHD (Russell et al., 2016; Santiago et al., 2011). SAS was not hypothesized to significantly differ between the ADHD and follow up ODD and ADHD without ODD groups. One limitation for this study could be using the SAS as the definition of SES. Although parental factors and perception are typically used to measure SES, McLaughlin et al. (2012) found that adolescent's subjective social status was most consistently associated with mental disorders, which was not one of the measures used in this study. Future research needs to examine how status dimensions other than parental income and education, such as adolescent's perceived social status, affect mental health outcomes. McLaughlin et al. (2012) examined this by asking adolescents "where you think you stand relative to other young people in your school, neighbourhood, or community". In addition, Sweet (2010)

suggests including memberships of social groups and possession of desirable objects to assess adolescent's perception of their social status.

### 5.5.4 Temperament

Temperament activity level differed between the ADHD and follow up ODD compared to the ADHD alone group, consistent with the published literature (Stringaris et al., 2010) and confirming my hypothesis that an increase in temperament activity level would predict ADHD and follow up ODD. A key methodological limitation constraining the interpretation of these results are the different instruments used to ascertain temperament in the published literature, which may measure different constructs making it difficult to compare findings. From a clinical point of view, temperament activity not only refers to a child's physical energy (constantly moving) but also their mental activity (such as, constant thinking or being able to read activities). Increased temperament activity suggests that these young people may have less awareness of those around them and more difficulties analysing different physiological, cognitive and social cues that aid in the identification of emotional states in themselves and others (Chess & Thomas, 1987, 1999) and be less able to problem solve as issues arise. Therefore, teaching specific skills regarding slowing down, reading the environment, being able to think more deeply, and problem solve might be beneficial to add to treatment plans for young people with ADHD to help prevent the development of ODD. Temperament is malleable and interventions targeting affective, attentional, and behavioural regulatory components of temperament may reduce future disruptive behaviours in children (Wichstrøm et al., 2018). Good parent management skills (non-harsh discipline, high maternal positive guidance) and treating parental psychopathology have also been found to effectively moderate the relationship between children's temperament and Disruptive Behaviour Disorders (Antúnez et al., 2016; Guo & Mrug, 2018; Rubin et al., 2003). Future longitudinal research investigating temperament, parent management skills and parental psychopathology is required to help us understand how these factors together impact on young people with ADHD developing ODD.

### 5.5.5 Intelligence

The Verbal Comprehension Index was lower in the ADHD and follow up ODD compared to the ADHD alone group, consistent with the findings of Speltz et al. (1999) and my hypothesis. Verbal Comprehension involves the capacity to access and find words, the ability to retrieve information, vocabulary, and verbal reasoning and problem-solving skills. Lower scores suggest that these individuals will have trouble understanding oral language or verbal expression and therefore have difficulty finding words to communicate their emotions and needs. Young people with lower Verbal Comprehension abilities have difficulty: understanding instructions that are given to them; understanding compound and complex sentences and colloquialisms; following multi-step directions; communicating how they are feeling in a situation; and difficulty reasoning aloud. They may also: feel confused and look disinterested in the classroom; get frustrated easily due to not understanding or keeping up in the classroom or with their peers; and have trouble solving conflicts or problems that arise. Therefore, assessing if a young person has deficits in their different intelligence abilities is important and indeed Verbal Comprehension difficulties maybe a risk factor for developing future behavioural difficulties. Early detection of Verbal Comprehension difficulties in young people with ADHD can lead to accommodations being made by parents and teachers (to use brief, clear, concise language when giving instructions, to check they have understood what is expected of them and use visual tools to aid in understanding and memory) which may reduce future behavioural difficulties and the development of ODD. Skills training for young people with ADHD in verbal reasoning and problem solving may also assist these young people to better resolve conflicts and problems with family and peers. Future longitudinal research is required to aid our understanding of the different and varied components of Intelligence and ADHD and ODD (separately from CD).

### 5.5.6 Visual Spatial Working Memory

Investigation of the key components of Visual Spatial Working Memory revealed important differences between the ADHD and follow-up ODD and ADHD without ODD groups. A better overall Visual Spatial Working Memory performance was evident in the ADHD with no ODD group, especially as the tasks got harder. This finding was consistent with my hypothesis that ADHD and follow up ODD would have a worse Visual Spatial Working Memory performance. Moreover, these results implied that the better

overall Visual Spatial Working Memory performance in the ADHD alone group may be more related to a better Spatial Span performance, given the non-significant trend for Spatial Span to be better in the ADHD alone group and that Strategy (organising planning) did not differ between the two groups . Visual Spatial Working Memory is the temporary storage memory that enables one to complete or work on complex tasks while being able to keep this information in mind and inhibit distracting information. Deficits in this area will impact on a young person's school performance and communicating with others. Young people exhibiting poor performance on Visual Spatial Working Memory tasks also tend to have worse problem-solving skills. Early detection of Visual Spatial Working Memory difficulties can allow parents and teachers to assist young people with ADHD by teaching compensatory strategies, recall strategies and using memory aids that may prevent the development of future ODD. Parents and teachers of young people with ADHD can be mindful to: reduce the number of elements that can interfere with Working Memory; repeat information; make connections to other concepts; present concepts in a variety of different ways; provide memory aids and visual supports (posters, graphic organizers, lists of procedures); and to teach and practice problem-solving strategies. Future longitudinal research is required to aid our understanding of the different and varied components of Working Memory and ADHD and ODD (separately from CD).

### 5.5.7 Academic Ability

The finding that academic ability did not differ between the ADHD with and without follow up ODD groups failed to support the proposed hypothesis that academic ability would be more impaired in the group of young people with ADHD and ODD at follow up. Clinically the means and standard deviations for reading and spelling in both groups did not differ significantly from the general population (mean 100, standard deviation 15), while mathematics was about one standard deviation lower than the general population for both groups.

These findings are consistent with previous literature, as it has been questioned if the difficulties associated with negative school outcomes are due to comorbid ADHD, rather than ODD. Burke et al. (2014) found that ODD symptoms were not predictive of low educational attainment. Liu et al. (2017) found that young people with ADHD had poorer performance across different domains of school functioning and youth with

ADHD and ODD/CD had more behavioural problems, but similar academic performance than those with ADHD only. They revealed that ADHD impaired academic performance, while ODD/CD aggravated behavioural problems, suggesting that the ODD/CD may specifically contribute to social difficulties in these young people rather than academic difficulties.

Although widely used in Australia in educational settings, the WRAT-4 has some important methodological limitations that affect any conclusions drawn (Wilkinson & Robertson, 2006): for example, there are few attempts given to each participant to solve mathematics problems and the reading test does not cover "phonemic awareness, knowledge of phonics or alphabetic principals, and vocabulary". Indeed, Wilkinson and Robertson (2006) recommend more detailed further educational assessment to guide educational interventions and this is important for future longitudinal research.

Both groups showed small deficits in mathematics ability (low average range of functioning), which is important to note. Monitoring and preventive work in this area may reduce future difficulties in mathematics for young people with ADHD. Although reading and spelling did not show any clinical impairment for both the ADHD alone and ADHD and follow up ODD groups, it is important to note that the majority of the young people in this study were in primary school. Once in secondary school, where the work load increases in quantity and complexity, a diagnosis of ADHD may interfere with academic achievement via problems sustaining attention, difficulties focusing on task instructions, having disorganised notes and written reports, and higher rates of off-task physical activity (motor overactivity of hands and feet) that may all result in worse test performance and suboptimal development of academic skills (DuPaul & Stoner, 2014). Hence, future longitudinal research should investigate academic abilities in more detail in youth with ADHD and ODD.

Academic achievement has a complex and dynamic interrelationship with cognition, behavioural, emotional dysfunction and environmental factors (parenting skills) that all need to be considered when looking at academic achievement outcomes for individuals with ADHD (Deault, 2010). Academic outcome is not only affected by the child's factors (IQ, Working Memory, motivation, academic aptitude, emotional regulation) but parent factors (for example, parenting skill and parental psychopathology) have also been found to influence children's success as they progress through school.

Future research is needed in the area of academic success and family variables (for example, the extent to which families encourage and motivate their children to do well academically, or the amount of support and structure that is provided at home with respect to homework). Narrowing in on the types of parenting skills that contribute to children's academic success could help define specific goals in parent intervention programs for young people with ADHD (Deault, 2010).

### 5.5.8 Emotional Regulation and Aggression

Parent reported emotional regulation difficulties were significantly worse for the ADHD and follow up ODD group than the ADHD alone group. This is consistent with the literature that children with ODD have been found to have poorer emotion regulation skills compared to children without ODD (Schoorl et al., 2016; Sobanski et al., 2010) and my hypothesis. Teacher reported emotional regulation difficulties did not differ significantly between the two groups, although there was a non-significant trend for the ADHD and follow up ODD group to have more teacher reported emotional regulation problems. Clinically, parents reported both groups (ADHD and ODD at follow up group 97%; ADHD alone group 80%) had significant DESR difficulties. For about half of the young people in each group teachers reported DESR difficulties (ADHD and ODD at follow up group 62%; ADHD alone group 46%). Emotional dysregulation is characterized by mood instability, severe irritability, aggression, temper outbursts, and hyper-arousal (Tonacci et al., 2019). Emotional regulation difficulties have been said to be one of the most psychosocially impairing and cost intensive mental health issues (Döpfner et al., 2019), so if identified and treated early in children with ADHD it may prevent the development of future ODD, as well as other difficulties. Treatment that assists young people with ADHD to learn self-regulation, such as meditation, mindfulness and stress management can help them gain control over negative emotions and their response to emotional situations. Dialectical Behaviour Therapy [a variety of cognitive behavioural therapy (young people are taught to pinpoint and adjust their core beliefs) combined with meditation] has been shown to be effective for the treatment of emotional dysregulation with Disruptive Behaviour Disorders (Burmeister et al., 2014; Marco, García-Palacios, & Botella, 2013; Stadler, Manetsch, & Vriends, 2016). Future longitudinal research should focus on more targeted and specific aspects of parent,

teacher, and self-reported emotion regulation difficulties association with ADHD and ODD.

Parent and teacher reported aggression was significantly increased in the ADHD and follow up ODD group compared to the ADHD alone group, consistent with my hypothesis and the extant literature (Biederman, Petty, Dolan, et al., 2008). Parent reported aggression was in the clinical range for the ADHD and follow-up ODD group and in the "at risk" range for the ADHD alone group. Teacher reported aggression was in the "at risk" range for the ADHD and follow-up ODD group while in the normal range for the ADHD alone group. Aggressive behaviour can cause physical and/or emotional harm to others, leading to breakdowns in relationships. It often occurs when a young person has trouble coping with their emotions (especially frustration), acts impulsively or lacks interpersonal understanding. However, it can also occur as a response to obtaining something that the young person wants. Young people with ADHD taught skills in conflict resolution, problem solving, emotional regulation and assertiveness maybe able to prevent the development of future ODD. Parents with good emotion coaching skills have been found to show positive effects on young people's emotion regulation and externalizing problems, suggesting that ODD and emotion regulation difficulties can be shaped through positive socialization processes (Calkins et al., 1998; Dunsmore et al., 2013; Dunsmore et al., 2016; Shortt et al., 2010). This research emphasizes the importance of assessing and including teaching parents' emotion coaching skills as part of managing young people with ADHD. Psychosocial treatment (parent management training, school-based training, functional family therapy/brief strategic family therapy, and Cognitive Behaviour Therapy) have all been found effective in treating aggression, although, if there is severe aggression, pharmacotherapy may also be indicated (Ghosh et al., 2017). Turgay (2004) suggests that the integration of medication, individual and family counselling, educational and psychosocial interventions including with the school and community, may increase the effectiveness of interventions in treating aggression. For young people with ADHD and ODD, stimulant medications including new generation long-acting medications and non-stimulant medications are considered the drugs of choice for managing aggressive behaviours and Disruptive Behaviour Disorders (Turgay, 2004). A current meta-analysis also reported that physical exercise in children with ADHD significantly improved aggressive behaviours, and therefore could also be incorporated in the daily lives of children with ADHD (Zang, 2019). Future longitudinal

research should systematically examine more targeted and specific aspects of parent, teacher, and self-reported aggression's association with ADHD and ODD.

### 5.5.9 Social Problems

Parent reported ability to make and keep friends did not differ between the ADHD and follow up ODD and ADHD alone groups. This finding was not consistent with the hypothesis that the ADHD and follow up ODD group would have more difficulties with keeping friends. However, it is consistent with the finding from the literature review undertaken by Becker, Luebbe, et al. (2012) concluding that the problems in peer relations among youth with ADHD cannot be wholly attributable to co-occurring behaviour problems. Under some circumstances, aggressive behaviour has been positively associated with peer acceptance (Vaughn, Vollenweider, Bost, Azria-Evans, & Snider, 2003). Both groups in this study were clinically impaired (with ADHD), putting them at risk of having significant peer relationship difficulties; so not seeing a significant difference between the groups could be due to two clinically impaired groups being compared, rather than to a typically developing healthy control group (Livingston, Dykman, & Ackerman, 1990; Pliszka, 1989). Interestingly, there was a non-significant trend seen for the ADHD and follow up ODD group to have difficulties keeping friends ($p = 0.07$). In this study only the parent view was obtained and results may have differed if teacher reports were ascertained (Becker, Luebbe, et al., 2012). This is an important area for future research as most research has found that peer acceptance in early childhood is a predictor of later peer relationship difficulties (Hay, 2005) with many researchers finding that early peer relationships have a significant correlation with aggressive, delinquent, oppositional, and illegal behaviours (Bagwell, Newcomb, & Bukowski, 1998; Coie, Lochman, Terry, & Hyman, 1992; Kupersmidt & Coie, 1990). Further research looking at the different developmental stages of the young person is important (specifically adolescents), especially given research showing young people with Disruptive Behaviour Disorders typically have associations with deviant peers (Dodge et al., 2007) and that affiliation with like peers can lead to the initiation of delinquent behaviour and further impact on their behaviour difficulties (Burke et al., 2002; Elliott & Menard, 1996; Keenan et al., 1995).

The findings for overall social problems, as reported by both parents and teachers, did differ between the ADHD and follow up ODD group compared to the ADHD alone group, which is consistent with the hypothesis that the ADHD and follow up ODD group would have more broad social problems. This is consistent with the literature, that when ADHD is comorbid with ODD, it has been reported to be associated with greater social impairment than ADHD alone on both parent and teacher ratings (Becker, Langberg, et al., 2012; Kuhne et al., 1997). Problems in this area can lead to negative social interactions that can impact on relationships, mental health, lead to early school leaving and vocational problems in adulthood (Semrud-Clikeman, 2007). Therefore, identifying social problems in young people with ADHD and intervening early is important for the prevention of future behavioural difficulties and the development of ODD. Universal social skills programs targeted at pre-schoolers are important as many models of development suggest that early intervention, compared to intervention at older ages, holds special promise because developmental trajectories are most malleable early in life (National Research Council (US), Institute of Medicine (US), & Committee on Integrating the Science of Early Childhood Development, 2000). More targeted intervention programs are also required for young people with ADHD with poor peer relationships. Bierman and Erath (2006) suggest that social-cognitive, emotional and behavioural skills are necessary for successful social interaction, and that these skills can be taught, including: social skills; emotional regulation; problem-solving skills; conflict management skills; sharing / cooperation skills; and improving parenting skills. Future research is required in this area as social problems have been found to be impacted upon by other risk factors, such as: low intelligence (Hyde et al., 2010); limited Working Memory capacities (Yaghoub Zadeh et al., 2007); and being exposed to adverse social experiences (for example, peer rejection, harsh parental discipline, community violence) that can lead to deviant social-cognitive dispositions which predicts disruptive behaviour problems (Dodge & Pettit, 2003; Troop-Gordon & Ladd, 2005). Longitudinal systematic research is required to look more closely at the interactions of these different risk factors and their contribution to social problems in young people with ADHD and ODD in order to develop more targeted interventions.

### 5.5.10 Overall Limitations

Three overall major limitations further constrain the interpretation of the results presented. First, the use of a clinical sample rather than an epidemiological sample may lead to divergent findings that are not generalisable. In particular, the severity and chronicity of functional impairments at home and school, for clinically referred young people, are known to drive referral to services (Mannuzza et al., 2004; Ogden & Hagen, 2018). In addition, rates of ODD are higher in clinically referred samples, as is the severity of all presentations of ADHD (Mannuzza et al., 2004; Ogden & Hagen, 2018). This study used strict inclusion criteria and therefore may represent a subsample of relatively homogeneous individuals with ADHD alone and ADHD and follow-up ODD.

Second, three factors (emotional regulation, aggression, and social problems) were measured using the same instrument that were used to define the ADHD and follow up ODD groups (the CBCL). This is especially true for the aggression factors given that ODD DSM Scale uses some of the items (argues, disobedient at home, disobedient at school, stubborn and has temper tantrums) used to define the Aggressive Behaviour subscale. This issue was considered when selecting measurement instruments for this study. However, it was thought to add additional questionnaires for both parents and teachers to complete would risk an excessive amount of questionnaires to complete, that could have resulted in poor completion rates, reduced motivation and accuracy of reporting (McColl et al., 2001). Interestingly, when looking at the mean and standard deviation for each of these factors the scores reflect a similar range to the general population (from the normal range, through to a clinical range), suggesting that in this case they could be appropriate measures.

Third, it is unknown if baseline psychosocial treatment interventions were offered to the young people in this study by other community professionals as these were not controlled. This variation may lead to divergent outcomes over time, given the known varied response of ADHD and comorbid disorders to comprehensive management when implemented (Waxmonsky, 2003).

Nevertheless, the clinical sample investigated evinced a number of characteristics that partially addressed these methodological limitations: the large number of referring schools, the predominantly treatment naïve cases referred by school professionals, and the standardized medication algorithms used post baseline assessment with all the cases

clear responders according to criteria outlined by Weiss et al. (2019). The two follow-up groups did not differ with respect to baseline type of medication used, final dose titrated to, or duration of baseline medication treatment. So, the associations reported are most likely due to changes over the 3-year follow-up period rather than the baseline medication treatment offered. However, in future, these limitations would be addressed via careful longitudinal examination of ADHD and follow up ODD compared to ADHD alone in epidemiologically derived samples with cases receiving standardized medication and psychological interventions in the community (Sciberras et al., 2013). In addition, outcomes for emotional regulation, aggression and social difficulties can be ascertained by instruments other than the CBCL.

## 5.6 SUMMARY

This study highlights important child factors to include in assessments of young people with ADHD. If early intervention is offered in these areas, it may prevent the development or persistence of future ODD. Significant child factors were: lower verbal comprehension; increased temperament activity level; Visual Spatial Working Memory difficulties; emotional regulation difficulties; aggression; and social problems. Although the differences for a number of factors were small in their effects, these small effects are to be expected given both groups are clinically impaired rather than comparing a clinically impaired group to a typically developing healthy control group (Livingston et al., 1990; Pliszka, 1989) and remain clinically important and significant. In addition, the known heterogeneity of ADHD and ODD, separately and when comorbid and the sheer multiplicity of small effect size risk factors that can lead to ADHD and ODD, whether alone or comorbid, can also explain these small effects (Barkley, 1997; Castellanos & Tannock, 2002; Ferrin & Vance, 2014; Hervey, Epstein, & Curry, 2004; Rapport et al., 2001). It is difficult for clinicians to identifying early predictors of outcomes for children with ADHD. However, these results provide some clinical guidance in identifying indicators for young people with ADHD developing or experiencing persistence of ODD, which can be helpful in informing preventive and therapeutic practices. Future longitudinal research is required to aid our understanding of these factors for young people with ADHD at different developmental stages, adolescents who develop ODD as

a “late starter” and the interactions of different risk and protective factors in order to develop more targeted interventions.

# CHAPTER 6: Key Psychological Factors of ADHD and Follow Up ODD (Study Two)

## 6.1 INTRODUCTION

Mental illness and psychological well-being are influenced by social factors (Marmot et al., 2010). Children and adolescents are not only influenced by broader community characteristics, but also their family environment that accounts for a big proportion of their life experience. Several studies have well documented the significant linkage between family context and child psychological development (Hetherington & Martin, 1979). Family functioning, parental relationship functioning, parenting skill, and parental psychopathology are environmental aspects that can influence the development of mental health disorders in young people. Most research shows that ADHD is associated with problematic family functioning including: greater stress within the family; lower levels of marital satisfaction; use of less effective parenting strategies; higher rates of parental psychopathology, especially maternal Depression; and negative / conflicted parent-child relationships (Anastopoulos et al., 2009; Deault, 2010; Johnston & Mash, 2001). Family factors are also known to be risk factors for developing Disruptive Behaviour Disorders - factors such as: poor family functioning; low parental relationship quality; poor parenting practices; and familial psychopathology (Burke et al., 2002; DeLisi & Vaughn, 2014; Greene et al., 2002; Marmorstein et al., 2009; Matthys & Lochman, 2016; Pardini & Frick, 2013). So, it is not unexpected that studies of families of children with ADHD and comorbid conduct problems have found associations between disturbances in family, disrupted parent-child relationships, increased levels of parenting dysfunction and parental psychopathology (Johnston & Mash, 2001). The identification of specific psychosocial characteristics that may be risk and protective factors for young people with ADHD developing ODD would be helpful in assisting to target these young people and their families for specific early intervention. This chapter begins with a brief summary of the predominant psychosocial factors associated with children diagnosed with ADHD and ODD at follow up including: parents' level of education; family functioning; parental relationship functioning; parenting skill; and parental psychopathology. This study (two) will examine these psychosocial factors in young people with ADHD alone and ADHD and follow up ODD to identify the risk and protective factors in developing ODD. Results from study two will be reported and their clinical relevance, limitations and future research directions explored.

### 6.1.1 Parental Level of Education

Parental education level is a significant predictor of a child's educational achievements and behavioural outcomes (Davis-Kean, 2005; Duncan, Brooks-Gunn, & Klebanov, 1994; Nagin & Tremblay, 2001). Parental education, specifically maternal education, has been found to be the strongest of the family SES predictors of young people's educational and vocational achievement in adulthood (Dubow, Boxer, & Huesmann, 2009; Eccles & Harold, 1993; Frome & Eccles, 1998). McLaughlin et al. (2012) and Sonego, Llácer, Galán, and Simón (2013) found adolescents whose parents had completed a degree at university were less likely to have a mental health disorder. Eccles and Harold (1993) suggested that parents socialize their children towards higher levels of educational achievement and occupational success, by modelling achievement-related behaviours and fostering positive expectations for academic performance. Parental education level has also been found to be an important predictor of children's behavioural outcomes (Davis-Kean, 2005; Nagin & Tremblay, 2001). Parental education level, both mothers and fathers, will be examined in this study.

### 6.1.2 Family Functioning

Family functioning is an important factor in a child's life that impacts on all their relationships and future wellbeing (Al Ubaidi, 2017). Many studies have associated poor family functioning with a child having ADHD alone (Cussen et al., 2012; Johnston & Mash, 2001; Mohammad pour & Kasaei, 2013) and ODD alone (Greene et al., 2002; Harvey et al., 2011; Lavigne et al., 2015; Lucia & Breslau, 2006). Families with children who have ADHD have been found to have difficulties at the level of "unhealthy functioning" in the Problem Solving, Roles, Affective Involvement, General Functioning, and behaviour control subscales on the McMaster Family Assessment Device (FAD) for family functioning.

Generally, studies have looked at ADHD with comorbid ODD and CD collapsed into one Disruptive Behaviour Disorder construct. Kiliç and Sener (2005) investigated family functioning in children with comorbid ADHD and ODD/CD, finding these families scored high at the level of unhealthy functioning in the Roles and Behaviour Control subscales of the FAD for family functioning. İmren et al. (2013), also using the FAD, found all areas of family functioning were scored high at the level of unhealthy

functioning for young people with ADHD. So, overall, the families of children and adolescents with ADHD comorbid with ODD or CD, had poorer family functioning in most of the subscales of the FAD.

There are very few studies that have investigated the relationship with comorbid ADHD and ODD (without CD) and overall family functioning. Barkley et al. (1992) compared two groups of clinic-referred adolescents (one with ADHD, one with ADHD and ODD) to a community control group. They found that adolescents with ADHD and ODD reported more conflicts at home, while their mothers reported greater negative interactions, greater personal distress and less satisfaction in their marriages than in the control group.

Some researchers have argued that the family functioning problems of young people with ADHD appear to mostly originate from Disruptive Behaviour Disorder features such as rule-breaking, aggressive and antisocial behaviours (Barkley et al., 1992; Fletcher, Fischer, Barkley, & Smallish, 1996). They report that most of the negativity in family interactions related to ADHD is due to comorbid disruptive behaviours in the children (Barkley, 1998; Barkley et al., 1992). Thus, comorbidity of ADHD with Disruptive Behaviour Disorders may increase the levels and types of family-related impairments of psychosocial functioning. Others argue that there is evidence that ADHD may place children at risk for developing ODD symptoms by disrupting family functioning (Harvey et al., 2011).

The majority of research designs that explore the relationship between family psychosocial variables and oppositional or conduct problems in children with ADHD use cross-sectional designs to contrast the patterns of associations among families (Deault, 2010), which are generally less useful in separating cohort effects and studying causation (Kelsey et al., 1998). Longitudinal research designs are more powerful in being able to achieve these outcomes. More longitudinal research of young people and ADHD with comorbid ODD is required to fully understand family functioning deficits in this area.

### 6.1.3 Parental Relationship

Although there is a lot of literature that has shown that family structure is linked with child wellbeing, less is known about the extent to which positive mother-father relationship quality is linked with children's outcomes (Goldberg & Carlson, 2014).

Several studies have found marital conflict was associated with heightened rates of children with Disruptive Behaviour Disorders (Bornovalova et al., 2013; Erath & Bierman, 2006). Several behavioural problems during childhood have been liked to marital conflict and discord: lower completion of high school and attending university; greater risk of unemployment after leaving school; increased mental health problems and criminal behaviour (Fergusson & Horwood, 1998; Fergusson et al., 2005; McLeod & Kaiser, 2004; Needlman et al., 1991). Wymbs, Pelham, Gnagy, et al. (2008) studied adolescents with comorbid ADHD and ODD also reporting a higher prevalence of frequent and unresolved conflict between their parents, than in families of adolescents with ADHD only.

Some researchers have argued that poorer marital satisfaction in families was not a symptom of ADHD, but was more associated with child behavioural problems instead (Johnston & Mash, 2001; Zarei et al., 2010). They hypothesized that the stressful and demanding nature of ADHD symptoms may elicit more marital miscommunications in parents. Studies have shown a link between the severity of child behaviour and interparental discord/interparental conflict and report greater discord among parents of youths with ADHD and comorbid ODD/CD than among parents of youths with ADHD alone or without ADHD (Barkley et al., 1992; Wymbs, Pelham, Gnagy, et al., 2008). Zarei et al. (2010) also found that the education level of the parent was an important protective factor in marital satisfaction in parents of children with ADHD, suggesting that the inclusion of other psychosocial factors is essential to further our understanding of the development of ODD. Therefore, further longitudinal research in understanding how parental relationship functioning is linked to young people with ADHD developing ODD (separately from CD) is needed.

### 6.1.4 Parenting Skill

Understanding the ways in which parents, through effective parenting, can help their children to regulate their emotions and behaviour, to facilitate friendships with peers and social networks, and to achieve academically is critical in understanding the pathways that shape different developmental trajectories for children with ADHD (Deault, 2010). Maladaptive parenting strategies (including: less warmth and sensitivity; and negative controlling parenting) and high levels of parental stress have been linked to children with

ADHD (Cussen et al., 2012; Johnston & Mash, 2001; Keown, 2012; Modesto-Lowe et al., 2008). Decreased parenting confidence has also been associated with ADHD (Cussen et al., 2012; Gordon & Hinshaw, 2017; Johnston & Mash, 2001).

Some studies suggest that ODD symptoms, not ADHD symptoms, predicted less positive parenting, including a lack of warmth and positive parental involvement, as well as reports of decreased parental involvement and responsiveness, poorer communication, more negative discipline strategies and parental intrusiveness (Burke et al., 2008; Evans et al., 2009; Johnston, Chen, & Ohan, 2006; Johnston & Mash, 2001; Johnston et al., 2002; Kashdan et al., 2004; Pfiffner et al., 2005).

There may also be a bi-directional relationship between parenting behaviour and child behaviour. Allmann (2018) found that: children's symptoms of psychopathology (ADHD, ODD, Depression and Anxiety) compounded over time due to decreased exposure to adaptive, and increase exposure to a maladaptive parenting styles; and maladaptive parenting continued to increase over time due to the persistence of child symptoms. Researchers suggest that children are at higher risk of developing Disruptive Behaviour Disorders due to a social learning and reciprocal process: over time children learn that oppositional and aggressive behaviours are effective ways to avoid undesired activities (for example, going to bed, doing chores), and parents become increasingly disengaged from attempting to control their child's behaviour or increasingly using timid discipline over time (Burke et al., 2008; Danforth, Anderson, Barkley, & Stokes, 1991; Dishion, Bullock, & Granic, 2002; McCart & Sheidow, 2016; McKee, Harvey, Danforth, Ulaszek, & Friedman, 2004; Shaw et al., 2001; Snyder & Patterson, 1995). This suggests that child behaviour can modify parenting behaviours in maladaptive ways over time (Deater-Deckard, 2000). Without further longitudinal studies it is not possible to determine what came first in the causative relationship between decreased parental warmth and conduct problems.

Chronis et al. (2007) found that positive parenting acts as a protective factor against conduct problems in the developmental stage of early childhood, suggesting that the interaction between several factors needs to be considered in future longitudinal research designs.

### 6.1.5 Parental Psychopathology

Parental psychopathology is an important risk factor for children's functioning (Chronis et al., 2003). Theoretical models suggest that parent psychopathology may place children at risk for behavioural, emotional, and social difficulties through several processes including: shared genetics; less available or poor parenting; less energy to fulfil their parenting role; exposure to parents' maladaptive cognitions, affect, and behaviour; exposure to stressful environments; and learned helplessness as a result of parents inability to change their children's behaviour (Dodge, 1990; Goodman & Gotlib, 1999; Harrison & Sofronoff, 2002; McCarty & McMahon, 2003). There is extensive research linking parent psychopathology to child functioning (Breaux et al., 2014).

Studies have reported that parental psychopathology is a risk factor for more severe and persistent child and adolescent ADHD symptoms (Biederman et al., 2011; Morgan et al., 2016). Several studies have noted positive associations between levels of maternal depressive symptoms and mother's ratings of child behavioural problems, usually rating their child with more externalizing and internalizing problems and lower social skills than children of parents without Depression (Boyle & Pickles, 1997; Breaux et al., 2014; Chi & Hinshaw, 2002; Chilcoat & Breslau, 1997; Fleck et al., 2015; Mineka et al., 1998; Najman et al., 2000). In addition, parental Depression has also been shown to predict family discord and behaviour problems in children with ADHD (Biederman et al., 2011; Cunningham et al., 1988). Maternal Depression has been found to predict conduct problems 2-8 years following the initial assessment for ADHD and the persistence of ODD (August et al., 1999; Blatt-Eisengart et al., 2009; Chronis et al., 2003).

Some studies suggest that parental psychopathology is more likely to be associated with oppositional and conduct problems than with ADHD alone (Chronis et al., 2003; Frick et al., 1992; Pfiffner et al., 2005). However, studies looking at comorbid ADHD and ODD have found mixed results. A couple of studies examined parental psychopathology and comorbid ADHD and ODD and failed to find an association with family histories of Conduct Disorder and Antisocial Personality Disorder (Faraone et al., 2000; Petty et al., 2009). Further longitudinal research in the area of ADHD and ODD (separately from CD) is required.

Although maternal Depression is commonly linked to ODD in children and adolescents (Barry et al., 2018), ODD also tends to occur in families with a history of parental ADHD, parental disruptive Behaviour Disorders, Substance Use Disorders, Depressive Disorders, Anxiety Disorders, Borderline Personality Disorder and Antisocial Personality Disorders, suggesting that a vulnerability to develop ODD may also be partly inherited (Biederman et al., 1992; Bornovalova et al., 2014; Bountress & Chassin, 2015; Breaux et al., 2014; Chronis et al., 2003; Hirshfeld-Becker et al., 2008; Shaw et al., 1994). Hirshfeld-Becker et al. (2008) found parents with comorbid disorders, such as Depression and a Disruptive Behaviour Disorder, are even more likely to have a child with a Disruptive Behaviour Disorder.

Most research assumes parental psychopathology plays a causal role in the development of ODD. However, child behaviour problems and parental psychopathology have shown a bidirectional relationship, with higher levels of parental distress being linked to more child behavioural problems, and more frequent problematic child behaviours being associated with increased parental distress over time (Neece et al., 2012). Parental psychopathology, therefore, may also serve to maintain ODD once established through a reciprocal process (Kazdin & Wassell, 1999; McKee et al., 2008).

Parental psychopathology has been found to not only be a predictor, but also a risk factor, interacting with other predictors (difficult temperament and poor parenting skill/harsh discipline) to increase the risk of developing ODD and the severity of ODD (McKee et al., 2008). Research has found it difficult to separate parenting behaviour from parental psychopathology in their contribution to the development of Disruptive Behaviour Disorders in children (Burke et al., 2002; Kaplan & Liu, 1999). Kaplan and Liu (1999) suggest while both poor parenting and parental psychopathology contribute to the development of these disorders, parental psychopathology may be a stronger determinant of disruptive behaviours in children than parenting behaviour. Smeekens et al. (2007) also recommend that parental personality (for example, ego-resiliency: resourceful adaptation to changing circumstances, including problem-solving strategies and the ability to perform under stress) needs further exploration as a potential predictor of ODD in children, rather than a strict parental diagnosis.

Parental psychopathology has also been found to be a barrier to accessing treatment and treatment outcomes for young people with ADHD (Kazdin & Wassell,

2000; Owens et al., 2002). These studies highlight the importance of future longitudinal research investigating the varied aspects of parental psychopathology and the management of parental psychopathology.

### 6.1.6 Summary

Research has shown that ADHD alone and ODD alone are associated with problematic lower levels of parental education, family functioning, lower levels of marital satisfaction, use of less effective parenting strategies and higher rates of parental psychopathology (Anastopoulos et al., 2009; Burke et al., 2002; Deault, 2010; DeLisi & Vaughn, 2014; Greene et al., 2002; Johnston & Mash, 2001; Marmorstein et al., 2009; Matthys & Lochman, 2016; Pardini & Frick, 2013). So, it is not surprising that studies of families of children with ADHD and comorbid conduct problems have found associations between disturbances in these areas as well. However, there is still a lot unknown about these factors for young people with ADHD who develop ODD, and limited studies looking at ODD separate from CD. Therefore, more systematic, and targeted longitudinal studies are required.

## 6.2 AIMS AND HYPOTHESES

The aim of study two is to examine the risk and protective *psychosocial* factors (parental level of education, family functioning, parental relationship functioning, parenting skill, and parental psychopathology) of young people with ADHD and follow-up ODD compared to ADHD alone. The hypothesis to be tested is that young people with ADHD and follow up ODD compared with those diagnosed with ADHD without follow-up ODD will manifest differential key psychosocial characteristics: decreased parental educational attainment; decreased family functioning; decrease parental relationship functioning; decreased parenting skill; and increase maternal Depression.

## 6.3 METHODS

All methods for this Study (two) are described in Chapter four.

## 6.4 RESULTS

The two groups did not differ for age, gender, social adversity status or ADHD severity at baseline (See Chapter 5, study one).

### 6.4.1 Parental Level of Education

The ADHD and ODD at follow up group demonstrated both mothers and fathers had significantly less education than the ADHD alone group (at baseline). These were small effects (See Table 8).

**Table 8: Parental level of education predicting ADHD and follow up ODD group membership (n=150) compared to ADHD alone group at follow up (n=60)**

| Level of schooling (PACS) | +ODD | -ODD | Wald | df | p | OR | (95%CI) |
|---|---|---|---|---|---|---|---|
| Mothers level of schooling | 4.94 (1.15) | 5.53 (1.27) | 9.71 | 1 | 0.002 | 1.51 | (1.16-1.95) |
| Fathers level of schooling | 4.89 (1.89) | 5.47 (1.30) | 8.63 | 1 | 0.003 | 1.50 | (1.14-1.97) |

### 6.4.2 Family Functioning and Parenting Skill

Baseline family functioning did not differ between the ADHD and follow-up ODD and ADHD alone groups for any of the subtests or for overall general functioning (See Table 9). Similarly, parenting skill, defined by the Behaviour Control subscale (FAD), did not differ between the two groups (See Table 9). Clinically, both groups

reported significant difficulties in Problem Solving, Communication, Affective Responsiveness, Behaviour Control, and overall general functioning (See Table 9). In relation to parenting skill, both groups reported clinically significant difficulties in the Behaviour Control subtest (See Table 9).

**Table 9: Family functioning predicting ADHD and follow up ODD group membership (n=150) compared to ADHD alone group at follow up (n=60)**

| FAD subtests | +ODD | -ODD | Wald | df | P | OR | (95%CI) |
|---|---|---|---|---|---|---|---|
| **Problem solving** | 2.06 (0.39) (C) | 1.99 (0.38) (C) | 0.20 | 1 | 0.66 | 1.03 | (0.90-1.19) |
| **Communication** | 2.16 (0.36) (C) | 2.08 (0.36) (C) | 0.89 | 1 | 0.35 | 1.05 | (0.95-1.16) |
| **Roles** | 2.45 (0.37) | 2.39 (0.40) | 0.27 | 1 | 0.61 | 1.02 | (0.94-1.11) |
| **Affective Responsiveness** | 2.01 (0.05) (C) | 1.89 (0.56) (C) | 0.87 | 1 | 0.35 | 1.06 | (0.94-1.19) |
| **Affective Involvement** | 2.19 (0.38) | 2.11 (0.53) | 0.35 | 1 | 0.56 | 1.03 | (0.93-1.15) |
| **Behavior Control** | 1.83 (0.36) (C) | 1.76 (0.37) (C) | 0.52 | 1 | 0.47 | 1.04 | (0.94-1.15) |
| **General Functioning** | 2.02 (0.43) (C) | 1.92 (0.41) (C) | 0.74 | 1 | 0.39 | 1.03 | (0.97-1.10) |

Note: +ODD: ADHD with follow up ODD group; -ODD: ADHD without ODD group; (C): clinical range

### 6.4.3 Parental Relationship

The two groups did not differ on any of the baseline parental relationship aspects ascertained (See Table 10). Clinically, both groups did not report any difference in the parental relationship functioning compared to the general population.

**Table 10: Parental relationship predicting ADHD and follow up ODD group membership (n=150) compared to ADHD alone group at follow up (N=60)**

| DAS | +ODD | -ODD | Wald | df | p | OR | (95%CI) |
|---|---|---|---|---|---|---|---|
| **Philosophy of life** | 2.48 (1.04) | 2.39 (.95) | 0.21 | 1 | 0.65 | 1.09 | (0.76-1.57) |
| **Aims, goals and things believed important** | 2.37 (1.08) | 2.33 (1.07) | 0.05 | 1 | 0.82 | 1.04 | (0.74-1.47) |
| **Amount of time spend together** | 2.46 (1.2) | 2.36 (0.99) | 0.20 | 1 | 0.65 | 1.08 | (0.78-1.50) |
| **Have a stimulating exchange of ideas** | 3.85 (1.46) | 4.03 (1.29) | 0.43 | 1 | 0.52 | 0.92 | (0.70-1.20) |
| **Calmly discuss something** | 4.34 (1.43) | 4.69 (1.22) | 1.80 | 1 | 0.18 | 0.82 | (0.61-1.10) |
| **Working together on a project** | 4.04 (1.72) | 4.29 (1.49) | 0.63 | 1 | 0.43 | 0.91 | (0.72-1.15) |
| **Happiness in relationship** | 4.28 (1.50) | 4.36 (1.44) | 0.08 | 1 | 0.78 | 0.97 | (0.75-1.24) |
| **Total** | 23.06 (7.37) | 24.21 (6.77) | 0.72 | 1 | 0.40 | 0.98 | (0.93-1.03) |

Note: +ODD: ADHD with follow up ODD group; -ODD: ADHD without ODD group

### 6.4.4 Parental Psychopathology

The two groups did not differ on baseline parental psychopathology for the Depression and Anxiety dimensions, once Bonferroni corrections were applied. In contrast, the Obsessive Compulsive, Interpersonal Sensitivity and Total parental psychopathology scales were significantly higher in the ADHD and ODD follow up group compared to the ADHD alone group. The findings suggest that the ADHD and ODD group at follow up had worse parental mental health functioning overall, and in the Obsessive Compulsive and Interpersonal Sensitivity dimensions (See Table 11). The Total parental psychopathology finding was a small effect, while the Obsessive Compulsive and Interpersonal Sensitivity findings were at a significant level. Clinically, parents of the ADHD and ODD at follow up group reported a number of subscales (Obsessive Compulsive; Depression; Interpersonal Sensitivity) and overall psychopathology to be in the clinically significant range of difficulty, compared to the ADHD alone group parents identifying clinically significant difficulties with overall parental psychopathology and the Depression subscale approaching significance.

**Table 11: Parental psychopathology predicting ADHD and follow up ODD group membership (n=150) compared to ADHD alone group at follow up (n=60)**

| Hopkins | +ODD | -ODD | Wald | df | p | OR | (95%CI) |
|---|---|---|---|---|---|---|---|
| **Obsessive Compulsive** | **1.9 (0.69)** (C) | **1.69 (0.56)** | **8.19** | **1** | **0.003** | **2.25** | **(1.32-3.83)** |
| **Depression** | 1.95 (0.64) (C) | 1.72 (0.53) | 5.35 | 1 | 0.02 | 1.90 | (1.10-3.28) |
| **Anxiety** | 1.61 (0.57) | 1.48 (0.52) | 2.45 | 1 | 0.11 | 1.62 | (0.89-2.95) |
| **Interpersonal Sensitivity** | **2.02 (0.67)** (C) | **1.71 (0.6)** | **8.56** | **1** | **0.003** | **2.24** | **(1.31-3.84)** |
| **Total subscale** | **105.83 (28.76) (C)** | **93.44 (25.28) (C)** | **7.68** | **1** | **0.006** | **1.02** | **(1.01-1.03)** |

Note: Hopkins: Hopkins Symptom Checklist; +ODD: ADHD with follow up ODD group; -ODD: ADHD without ODD group; (C): clinically significant range

## 6.5 DISCUSSION

### 6.5.1 Parental Level of Education

Both parents (mothers and fathers) had significantly less education in the ADHD and follow-up ODD group compared to the ADHD alone group, confirming my hypothesis. These findings are also consistent with previous literature where parental education level had been found to be an important predictor of children's behavioural outcomes (McLaughlin et al., 2012; Nagin & Tremblay, 2001). McLaughlin et al. (2012) found adolescents whose parents had not completed a university degree were more likely to have a mental health disorder (across all diagnostic groups). Assisting parents with further education maybe a helpful preventative factor for the development or persistence of ODD.

### 6.5.2 Family Functioning

The ADHD and ODD and ADHD alone groups at follow up did not show any difference in family functioning, not confirming my hypothesis that decreased family functioning would predict membership in the ADHD and follow-up ODD group. Generally, studies have looked at ADHD with comorbid ODD and CD collapsed into one Disruptive Behaviour Disorder construct, this group then demonstrating high scores at the level of unhealthy family functioning (İmren et al., 2013; Kiliç & Sener, 2005). There have been very few studies that have investigated the relationship with comorbid ADHD and ODD (without CD) and overall family functioning. This study suggests that for young people with ADHD the development or persistence of ODD (compared to CD) may not be associated with family functioning difficulties, due to finding no differences between the groups. This differs from the researchers who have argued that the family functioning problems of young people with ADHD appear to mostly originate from Disruptive Behaviour Disorder features such as rule-breaking, aggressive and antisocial behaviours (Barkley, 1998; Barkley et al., 1992; Fletcher et al., 1996).

Importantly, for the ADHD and ODD at follow up group and ADHD alone group, most subtest scales and the general overall functioning score were in the clinically significant range, indicating that these families did have significant overall family functioning difficulties, specifically in the areas: of Problem Solving; Communication; Roles; Affective Involvement; and Behaviour Control. The ADHD with no follow-up ODD group also manifested clinically significant scores supporting literature reporting poor family functioning is also associated with a child having ADHD alone (Cussen et al., 2012; Johnston & Mash, 2001; Mohammad pour & Kasaei, 2013). Clinically these findings signify the importance of assessing and treating family functioning difficulties for young people with ADHD and/or ODD. Effective family functioning relies on the establishment of clear roles within the family, good communication skills, the ability to relate and maintain relationships, and the ability to make decisions and solve problems (Epstein, Bishop, Ryan, Miller, & Keitner, 1993; Silburn et al., 2006; Zubrick, Williams, Silburn, & Vimpani, 2000). Therefore, teaching families who have young people with ADHD skills such as role identification and accountability, financial management, conflict resolution, assertive communication, and problem-solving can assist families with young people with both ADHD and ODD (Epstein et al., 1993; Silburn et al., 2006;

Zubrick et al., 2005). More longitudinal research of young people and ADHD with comorbid ODD is required to fully understand family functioning deficits in this area.

### 6.5.3 Parental Relationship

The ADHD and ODD and ADHD without ODD groups at follow up did not demonstrate any parental relationship functioning differences, not confirming my hypothesis. It is also not consistent with previous studies finding that marital conflict was associated with heightened rates of Disruptive Behaviour Disorders (Bornovalova et al., 2013; Erath & Bierman, 2006) or that poorer marital satisfaction in families was not a symptom of ADHD but more associated with disruptive behaviour problems (Johnston & Mash, 2001; Wymbs, Pelham, Gnagy, et al., 2008; Zarei et al., 2010).

Clinically, both groups manifest no difficulties overall in their parental relationships with parents reporting being happy in their relationships. However, both groups reported having differences of opinion in their philosophy of life, aims, goals, things they believed important and the amount of time they spend together compared to normative data from the DAS, suggesting some difficulties are emerging in their parental relationships. Several studies have found that the impact of parental conflict during childhood leads to difficulties in adulthood: lower completion of high school and attending university; greater risk of unemployment after leaving school; increased mental health problems and criminal behaviour (Fergusson & Horwood, 1998; Fergusson et al., 2005; McLeod & Kaiser, 2004; Needlman et al., 1991). It is also important to note that the nature of the association between the quality of the parents' relationship and their children's behaviour can be bidirectional (Cui et al., 2007; Goldberg & Carlson, 2014; Schermerhorn et al., 2007). Given the young age of the participants in this study it would be important to continue to follow these families over time to assess parental relationship difficulties and long-term outcomes. Other factors could also be a protective in the parental relationship functioning for parents of children with ADHD, such as the level of parental education (Zarei et al., 2010) and supportive co-parenting (Parkes et al., 2019) and need to be considered. Future longitudinal research will help us understand in greater detail the impact of ADHD, and separately ODD, on key further detailed aspects of parental relationship functioning and parental conflict, for developing ODD (separately from CD) in young people with ADHD.

### 6.5.4 Parenting Skill

The ADHD and ODD and ADHD without ODD groups at follow up did not differ in parenting skill, not confirming my hypothesis that poorer parenting skills would predict membership in the ADHD and follow up ODD group. It is also not consistent with some studies suggesting that ODD symptoms, not ADHD symptoms predicted poorer parent management skills (Burke et al., 2008; Evans et al., 2009; Johnston et al., 2006; Johnston & Mash, 2001; Kashdan et al., 2004; Pfiffner et al., 2005). This finding does indicate and support those studies that report maladaptive parenting strategies and high levels of parental stress are linked to children with ADHD without Disruptive Behavioural Disorders (Cussen et al., 2012; Johnston & Mash, 2001; Keown, 2012; Modesto-Lowe et al., 2008). Clinically, both groups noted significant difficulties in this area, suggesting this is an area for further investigation and treatment of young people with ADHD.

Although the Behaviour Control subtest of the FAD assesses the way in which a family expresses and maintains standards for the behaviour of its members (including, different patterns of control: flexible; rigid; laissez-faire; and chaotic parenting styles are assessed) it may not have been comprehensive enough or sensitive enough to pick up the differences in parent management styles for these two clinical populations. Miller et al. (1985) has questioned if the separate dimensions are sufficiently independent scales compared to traditional psychometric tests for each scale. This factor was considered when selecting measurement instruments for this study. However, I and my supervisory team, thought adding additional questionnaires for both parents and teachers to complete would be excessive, and possibly result in poor completion rates, reduced motivation, and accuracy of reporting. Future studies should use measures that assess specific factors that have been found to correlate with children's disruptive or delinquent behaviour (lower degree of involvement; poor parent-child relationship; worse parent-child conflict management; low parental warmth; poor monitoring; and harsh and inconsistent discipline) (Burt et al., 2005; Jaffee et al., 2004; Masten & Garmezy, 1985; Stormshak et al., 2000; Tang et al., 2017; Waschbusch, 2002).

The bidirectional relationship between parent management skills and the influence of the young person's ADHD on the quality of parenting, needs to be acknowledged. Children with ADHD typically present with high activity, short attention span, impulsiveness, increased aggression and poor social skills (Johnston & Mash, 2001). Many researchers have noted the effects of the child's ADHD symptoms on

parental behaviour (Allmann, 2018; Danforth et al., 1991; McKee et al., 2004), suggesting that child behaviour can modify parenting behaviours in maladaptive ways over time (Deater-Deckard, 2000). This, together with the clinical reality that a substantial number of children with ADHD do not receive pharmacological treatment (due to cost, it not being acceptable to parents, or the child refusing to take it) and for those who do use medication some typical medications wear off in the evening, the use of other interventions for young people with ADHD, including parent management strategies, are important (Foy & Earls, 2005; Modesto-Lowe et al., 2008). The effectiveness of training parents of children with ADHD in behavioural interventions is supported by several studies, which show these parents have high levels of stress, as well as a lack of confidence in their parenting skills, when compared with typically developing control participants (August et al., 1999; Cunningham, 2007; Danforth, Harvey, Ulaszek, & McKee, 2006; Hartman, Stage, & Webster-Stratton, 2003; Hutchings et al., 2007; Johnston & Mash, 2001). Evidence based parenting training includes: an overview of ADHD and its impact on parent-child interactions; strategies to attend to and improve child compliance; and strategies to reduce disruptive behaviour (Modesto-Lowe et al., 2008). Early treatment, positive parenting, and the absence of comorbidity may all optimize functioning and likely improve the course of young people with ADHD (Modesto-Lowe et al., 2008).

Although parenting *per se* may not alter the development of ADHD, parents play an important role delivering medication and behavioural interventions for children and adolescents with ADHD as they are responsible for administering medication and implementing behaviour management strategies, which can prevent worsening of the severity of ADHD and behavioural difficulties (Modesto-Lowe et al., 2008). Low parenting self-esteem and efficacy were associated with a poorer response to behavioural, pharmacological, and combined treatments in the Multi-Modal Treatment Study for ADHD (Hoza et al., 2000), suggesting the importance of further research in this area. In contrast, positive parenting has been found to predict fewer future conduct problems and was a protective factor for the developmental course of conduct problems in children with ADHD (Chronis-Tuscano et al., 2011), suggesting further longitudinal research looking at risk and protective factors is needed.

### 6.5.5 Parental Psychopathology

The ADHD and follow-up ODD manifest increased Obsessive Compulsiveness, Interpersonal Sensitivity and overall Total parental psychopathology compared to the ADHD alone group. These findings do not support the hypothesis that maternal Depression would predict the membership of young people in the ADHD and ODD group at follow up. Although, the ADHD and ODD group parents did report clinically significant levels of depressive symptoms and the ADHD alone group were approaching clinical significance, suggesting both groups of parents are manifesting depressive symptoms. It is also not consistent with studies that have reported positive associations between levels of maternal depressive symptoms and mother's ratings of more externalizing, internalizing and conduct problems than children of parents without depressive symptoms (August et al., 1999; Blatt-Eisengart et al., 2009; Boyle & Pickles, 1997; Breaux et al., 2014; Chi & Hinshaw, 2002; Chilcoat & Breslau, 1997; Chronis et al., 2003; Fleck et al., 2015; Mineka et al., 1998). However, the findings are consistent with the studies that suggest that overall parental psychopathology is more likely to be associated with oppositional and conduct problems than with ADHD alone (Chronis et al., 2003; Frick et al., 1992; Pfiffner et al., 2005).

Parental psychopathology has been found to have negative effects on parent management practices. Interpersonal Sensitivity and Obsessive Compulsive difficulties impact differently. Interpersonal Sensitivity is an undue and excessive awareness of and sensitivity to the behaviour and feelings of others (Boyce & Parker, 1989), that can lead to feelings of personal inadequacy and inferiority and discomfort during interpersonal interactions (Derogatis et al., 1974). It has been found to correlate with attachment insecurity, Depression and Anxiety disorders (Boyce, Parker, Barnett, Cooney, & Smith, 1991; Harb, Heimberg, Fresco, Schneier, & Liebowitz, 2002; Otani et al., 2014). It can impact on: the parent's ability to create a secure attachment in a child; can result in a core belief in the child that they are unworthy of love and support; and increasing their dependence on others acceptance to maintain a positive self-regard; and/or a core belief that others are unreliable and rejecting, leading to avoidance of closeness and relationships due to the fear of interpersonal rejection (Bartholomew, 1990). Obsessive compulsive behaviour in parents can cause unwanted, persistent and intrusive thoughts and can lead to more general cognitive difficulty and Anxiety (Derogatis et al., 1974). This can impact on: parenting by causing negative parenting practices (causing parents to

be overprotective (repeatedly checking on the child)); negative interpersonal styles; role modelling anxious behaviour (rituals of cleaning, dressing routines, worries about health and safety); and increase the risk of social, emotional and behavioural disorders in young people (Black, Gaffney, Schlosser, & Gabel, 2003; Kashdan et al., 2004).

It is important to note that both groups showed clinically significantly over all parental psychopathology difficulties suggesting that parents of young people with both disorders had substantially worse mental health difficulties. This is significant given the literature showing that parental psychopathology is a risk factor for children's functioning and more severe and persistent ADHD symptoms (Biederman et al., 2011; Breaux et al., 2014; Chronis et al., 2003; Dodge, 1990; Goodman & Gotlib, 1999; Harrison & Sofronoff, 2002; McCarty & McMahon, 2003; Morgan et al., 2016).

Most research assumes parental psychopathology plays a causal role in the development of ODD. However, child behaviour problems and parental psychopathology have demonstrated a bidirectional relationship. Therefore, parental psychopathology may also serve to maintain ODD once established through a reciprocal process (Chronis et al., 2003; Kazdin & Wassell, 1999; McKee et al., 2008; Neece et al., 2012). Parental psychopathology has also been found to not only be a predictor, but also a risk factor, interacting with other predictors (difficult temperament and poor parenting skill/harsh discipline) to increase the risk of developing ODD and the severity of ODD (McKee et al., 2008). Research has found it difficult to separate parenting behaviour from parental psychopathology in their contributions to the development of Disruptive Behaviour Disorders in children (Burke et al., 2002; Kaplan & Liu, 1999). Future longitudinal research is needed to help us understand the complex interaction between parental psychopathology, parenting skill and young people with ADHD developing ODD.

In addition, parental psychopathology has been found to be a barrier to accessing treatment, and to predict poorer compliance with and response to parent training and forgetting to administer medication for young people with ADHD (Griest & Forehand, 1983; Kazdin & Wassell, 2000; McMahon et al., 1981; Owens et al., 2002; Sonuga-Barke et al., 2002; Weiss et al., 2000). This further highlights the importance and clinical relevance of treating parental psychopathology as part of the management package for young people with ADHD.

Two particular limitations for this study of parental psychopathology exist so the data should be interpreted carefully. First, the parents in this study were not clinically diagnosed, and all psychopathology measures were based on parents' self-reports of symptoms. Although using dimensional measures provides several advantages, these findings may not generalize to parents with a clinical diagnosis (Noordermeer et al., 2017). Second, the use of one questionnaire to collect the information of parental psychopathology did not cover all possible diagnoses. Previous studies have found parental ADHD, parental Disruptive Behavioural Disorders, parental Anti-social Personality Disorder and parental substance abuse have been risk factors for children Developing Behavioural Disorders (Biederman et al., 1992; Bornovalova et al., 2014; Bountress & Chassin, 2015; Breaux et al., 2014; Chronis et al., 2003; Hirshfeld-Becker et al., 2008; Shaw et al., 1994). This information was not collected through this questionnaire and should be included in future longitudinal studies.

### 6.5.6 Overall Limitations

Two overall major limitations further constrain the interpretation of the results presented. First, the use of a clinical sample rather than an epidemiological sample may lead to divergent findings. In particular, the severity and chronicity of functional impairments at home and school for clinically referred young people are known to drive referral to services (Mannuzza et al., 2004; Ogden & Hagen, 2018). In addition, rates of ODD are higher in clinically referred samples as is the severity of all presentations of ADHD (Mannuzza et al., 2004; Ogden & Hagen, 2018). This study used strict inclusion criteria and therefore may represent a subsample of individuals with ADHD alone and ADHD and follow-up ODD. Second, it is unknown if psychosocial treatment interventions were offered to the young people in this study by other community professionals as these were not controlled. This variation may lead to divergent outcomes over time depending on varied response of comorbid disorders to the comprehensive management offered (Waxmonsky, 2003).

Nevertheless, the clinical sample investigated evinced a number of characteristics that partially addressed these methodological limitations: the large number of referring schools, the predominantly treatment naïve cases referred by school professionals, and the standardized medication algorithms used post baseline assessment with all the cases

clear responders according to criteria outlined by Weiss et al. (2019). The two follow-up groups did not differ with respect to baseline type of medication used, final dose titrated to, or duration of baseline medication treatment. So, the associations reported are most likely due to changes over the 3-year follow-up period rather than the baseline medication treatment offered. However, in future, these limitations would be addressed via careful longitudinal examination of ADHD and follow up ODD compared to ADHD alone in epidemiologically derived samples with cases receiving standardized medication and psychological interventions in the community (Sciberras et al., 2013).

## 6.6 SUMMARY

This study highlights important psychosocial factors to consider for young people with ADHD: if early intervention is offered in these areas it may prevent the development or persistence of future ODD. The significant factors were: both mothers and fathers' lower level of education; and increased overall parental psychopathology, Obsessive Compulsive symptoms and Interpersonal Sensitivity. It is important to note, that the ADHD and follow-up ODD group manifest most subscales for family functioning (Problem Solving, Communication, Roles and Affective Involvement) and overall family functioning in the clinical range for difficulties, suggesting that these would impact negatively on the day to day lives of these families and young people. Both groups also reported clinically significant difficulties in the Behavior Control subtest suggesting deficits in parenting skills. Also, clinically significant overall parental psychopathology for both ADHD and follow-up ODD and ADHD alone groups was evident. Although significant differences were primarily small in their effects, these are to be expected given both groups are clinically impaired rather than comparing a clinically impaired group to a typically developing healthy control group (Livingston et al., 1990; Pliszka, 1989). Hence, they remain important clinically. Also, the known heterogeneity of ADHD and ODD, separately and when comorbid and the sheer multiplicity of small effect size risk factors that can lead to ADHD and ODD, whether alone or comorbid, can also explain these small effects (Barkley, 1997; Castellanos & Tannock, 2002; Ferrin & Vance, 2014; Hervey et al., 2004; Rapport et al., 2001). These results provide some clinical guidance in identifying psychosocial indicators for young people with ADHD developing or having

persistent ODD, which can be helpful in informing preventive and therapeutic practices. Future longitudinal studies will help us understand the complex (risk and protective factors) and bidirectional nature of psychosocial factors (family functioning, parental relationship functioning, parenting skill and parental psychopathology) for young people with ADHD developing or maintaining ODD.

# CHAPTER 7: Key comorbid conditions and subtypes of ADHD and follow up ODD (Study Three)

## 7.1 INTRODUCTION

Co-occurring mental health problems are the norm, rather than the exception among children and adolescents with ADHD in both community and clinical samples (Barkley, 2006; Becker, Luebbe, et al., 2012). ADHD is consistently shown to have a greater than chance occurrence with a number of other psychiatric conditions including Anxiety Disorders, Depressive Disorders and Disruptive Behaviour Disorders (CD and ODD) (Angold et al., 1999; Becker, Luebbe, et al., 2012; Biederman, Faraone, Milberger, Curtis, et al., 1996). This could suggest that ADHD places young people at risk for the development of other mental health problems (Wilens et al., 2002). Researchers also often report that ODD co-occurs with ADHD, CD, Mood Disorders (MDD and PDD), Anxiety Disorders and ASD (American Psychiatric Association, 2013; Burke & Loeber, 2010; Burke et al., 2005; Ezpeleta et al., 2006; Rowe et al., 2010). The identification of specific comorbid disorders that may be risk and/or protective factors for young people with ADHD developing ODD could be helpful in assisting to target young people for specific early intervention. This study will look at the comorbid disorders, as well as ADHD subtype, in young people with ADHD alone and ADHD and ODD at follow up to help us explore the risk and/or protective factors in developing ODD. Results from study three, looking at ADHD subtype and the comorbidity of ADHD and follow up ODD will be reported and the clinical relevance and limitations to these findings will be discussed.

### 7.1.1 ADHD Subtypes

ADHD is often identified and diagnosed during primary school years as the young person's inattention becomes more impairing once they start school. During childhood excessive motor activity and inattention are the most common symptoms (APA, 2013; Dulcan & Lake, 2012). In most individuals with ADHD, symptoms of hyperactivity become less obvious or subside in adolescence and adulthood, but difficulties of restlessness, inattention, poor planning and impulsivity persist (APA, 2013; Cherkasova et al., 2013; Colomer-Diago et al., 2012; Faraone et al., 2015). Literature on the different subtypes of ADHD (C: Combined type; HI: Hyperactivity Impulsive type; I: Inattentive type) have found variation of difficulties overtime. Individuals with ADHD-C typically

manifest more severe ADHD core symptoms and these symptoms are relatively more stable across developmental stages compared to ADHD-HI and ADHD-I presentations (Lacramioara & Eugene, 2007; Meyer & Sagvolden, 2006; Wilens & Spencer, 2010). There is also a high chance that children with ADHD-HI will be diagnosed with ADHD-C by adolescence (Riley et al., 2008). Children with ADHD-I were found to have more learning difficulties and lower reading, spelling and mathematic scores over an 8-year period than children with ADHD-HI and ADHD-C (Massetti et al., 2008). Freitag et al. (2012) reported a different pattern of risk factors for inattentive and hyperactive-impulsive ADHD symptoms: inattentive symptoms were significantly affected by psychosocial risk factors, while hyperactive-impulsive symptoms were more influenced by biological risk factors. Additionally, hyperactive-impulsive symptoms appeared to be more related to risk factors for ODD/CD in the ADHD population (Freitag et al., 2012). Most young people with ADHD-C have one or more comorbid condition and this is a major contributor to the complexity of optimally managing ADHD-C; (Polanczyk et al., 2007; Wilens & Spencer, 2010). The predominant comorbid externalising and internalising disorders with ADHD-C are ODD and CD (about half of the clinical population with ADHD), Anxiety (about 20-30%), Learning Disorder (about 20-25%) and Depressive Disorders (Biederman, Ball, et al., 2008; Biederman, Faraone, Mick, & Lelon, 1995; Lacramioara & Eugene, 2007; Wilens et al., 2002). This study will look at the role of the ADHD subtypes in developing ODD.

### 7.1.2 Three or More Comorbid Disorders

Although there are published studies that have examined the prevalence of disorders in children and adolescents, there appear to be very few studies which that investigated the prevalence and outcomes of three or more comorbid disorders. When children and adolescents have ADHD comorbid with other disorders they tend to have more severe and enduring psychopathology than either disorder alone (Daviss, 2008; Hoza et al., 2010; Sikora et al., 2012; Tannock, 2009; Vance, 2011; Zablotsky et al., 2020). Children with ADHD and co-occurring comorbidities were more likely to have a poorer quality of life and greater peer and family problems compared to ADHD alone (Armstrong et al., 2015).

### 7.1.3 Oppositional Defiant Disorder

A condition which is frequently comorbid with ADHD is ODD, occurring in up to 70% of young people with ADHD (Burke et al., 2002; Connor & Doerfler, 2008; Fergusson et al., 1991; Offord et al., 1991). Only a few studies have investigated ODD separately from CD, often reporting them together under the heading of Externalizing Disorder or Disruptive Behaviour Disorder. DSM-IV precluded making a diagnosis of ODD in the presence of CD, as the literature was not clear whether ODD and CD could be comorbid (Steiner et al., 2007). Historically, there were questions if they represented two parts of a single continuum or construct (Moffitt et al., 2008). However, there is significant evidence that they are two distinct disorders (Pardini, Frick, & Moffitt, 2010; Rowe et al., 2010) and the majority of children with ODD do not develop CD (Lahey & Loeber, 1994; Loeber et al., 2000; Rowe et al., 2010). ODD has been found to have different socio-environmental and genetic correlates than CD (Dick et al., 2005; Hudziak et al., 2005). Rowe et al. (2010) examined the links between ODD and CD finding key differences: CD largely predicted behavioural outcomes, whereas ODD predicted emotional disorders in early adult life; the irritable and headstrong dimensions in ODD symptoms predicted later behavioural and emotional disorders. It is now accepted and defined in DSM-5 that ODD and CD are different diagnoses and often co-occur in young people. Given the recent clarification around CD and ODD being distinct disorders research in the comorbidity of CD and ODD is only in its early stages.

Young people with ADHD and ODD are the most frequently referred groups to mental health centres (Merikangas et al., 2011), have significant peer and family impairments, academic underachievement (Leadbeater & Ames, 2017) and an increased risk of developing Depression, Anxiety and antisocial behaviour (Copeland et al., 2009; Nock et al., 2007). It is estimated that a young person with ODD or CD will cost society approximately ten times that of a young person without a Disruptive Behaviour Disorder (Foster & Jones, 2005). Therefore, early identification of risk factors for developing ODD in young people with ADHD could aid in targeting specific early intervention to prevent long-term negative outcomes.

### 7.1.4 Depression and Anxiety

Children and adolescents with ADHD have been found to have a greater than chance rate of comorbid Depression (Angold et al., 1999; Biederman, Monuteaux, et al., 2008; Lundervold et al., 2016) and Anxiety (Cox, Jr., 1982; Vance, 1997; Vance et al., 2002). When these disorders co-exist, the associated functional impairments are more severe (Hoza et al., 2010; Tannock, 2009; Vance, 2011). Both Depression and Anxiety are also commonly associated with ODD and can develop as early as preschool age (Nock et al., 2007; Rowe et al., 2010; Steiner et al., 2007). In a more recent study Nock et al. (2007) found at least 50% of participants with ODD had comorbid Anxiety or Depression. There is some evidence to suggest that ODD, and not CD, may best explain the comorbidity between Disruptive Behaviour Disorders and Depression (Burke & Loeber, 2010). It is unclear what role coexisting disorders with ADHD play in the role of the development of ODD in young people.

### 7.1.5 Autism Spectrum Disorder

It is only in the current version of the DSM (APA, 2013) that ADHD can be diagnosed in the presence of ASD, as it was thought that the symptoms of both disorders overlapped significantly. Research in this area is limited, although individuals with co-occurring ASD and ADHD face more challenges than those with one condition only and it increases the likelihood of Disruptive Behaviour Disorder (Sikora et al., 2012; Zablotsky et al., 2020). There is also limited research in the area of ODD and ASD, even though comorbidity between the two disorders is common (Gadow et al., 2008; Guttmann-Steinmetz et al., 2009). Some researchers have questioned whether ODD behaviour among children with ASD reflects the same disorder seen in children without ASD (Kaat & Lecavalier, 2013). Others have reported that comorbid ODD does appear to present differently from other ASD related behavioural difficulties and that ASD symptomotology is not uniquely associated with ODD behaviours, suggesting that co-occurrence of the two disorders is a true comorbid presentation (Gadow et al., 2008; Mandy et al., 2014). Further longitudinal research in the area of comborbid ADHD, ASD and ODD is important given the high prevalence of these comobid conditions.

### 7.1.6 Summary

ADHD is consistently reported to have a greater than chance occurrence with a number of other psychiatric conditions. The identification of specific ADHD subtypes and comorbid disorders that may be risk and/or protective factors for young people with ADHD developing ODD would be helpful in assisting to target young people for specific early intervention. Specific ADHD subtypes and comorbid disorders in young people with ADHD will be explored to help understand their association in developing or the persistence of ODD.

## 7.2 AIMS AND HYPOTHESIS

The aim of study three is to examine the risk and protective *comorbid* conditions (ODD, CD, Depression (MDD and PDD), Anxiety and ASD) and the ADHD subtypes (ADHD-C, ADHD-I, ADHD-HI) of young people with ADHD and follow-up ODD. The hypothesis to be tested is that young people with ADHD and follow-up ODD compared with those diagnosed with ADHD without follow-up ODD will manifest differential comorbid conditions: increased ODD; increased CD; and increased ADHD-C.

## 7.3 METHODS

All methods for this study (three) are described in chapter three.

## 7.4 RESULTS

The two groups did not differ for age, gender, social adversity status or ADHD severity at baseline (see chapter 5, study one).

ADHD baseline subtype (ADHD-C, ADHD-HI, ADHD-I**)** and baseline key high prevalence comorbid conditions were compared between the ADHD and ODD and ADHD without ODD groups at follow up**.** ADHD-C versus ADHD-I was significantly

higher in the ADHD and ODD follow up group compared to the ADHD alone group (see Table 12). This effect was clinically significant. There were no differences between the two groups for ADHD-HI versus ADHD-I and ADHD-C versus ADHD-HI.

**Table 12: ADHD presentation predicting ADHD and follow up ODD group membership (n=150: ADHD-C n=117, ADHD-HI n=11, ADHD-I n=22) compared to ADHD alone group at follow up (n=60: ADHD-C n=33, ADHD-HI n=6, ADHD-I n=21)**

| ADHD type | Wald | Df | p | OR | (95%CI) |
|---|---|---|---|---|---|
| **ADHD-C versus ADHD-I** | **9.37** | **1** | **0.002** | **3.40** | **(1.55-7.46)** |
| **ADHD-HI versus ADHD-I** | 0.92 | 1 | 0.34 | 0.75 | (0.41-1.36) |
| **ADHD-C verses ADHD-HI** | 0.94 | 1 | 0.33 | 1.90 | (0.52-6.97) |

Note: ADHD-C: Attention Deficit Hyperactivity Disorder – combined type; ADHD-HI: Attention Deficit Hyperactivity Disorder – Hyperactivity-Impulsive type; ADHD-I: Attention Deficit Hyperactivity – Inattentive type

Table 13 presents the comorbid conditions (numbers in each group) at baseline assessed for the ADHD and ODD and ADHD alone groups at follow up. Baseline ODD and CD were increased in the ADHD and follow-up ODD group compared to the ADHD alone group. These were both clinically significant effects (see Table 13). Baseline Generalised Anxiety Disorder (GAD) was decreased in the ADHD and ODD group compared to the ADHD alone group at follow up. Again, this was a clinically significant effect. There were no differences between the groups for Panic Disorder, Separation Anxiety Disorder, Social Anxiety Disorder, MDD, PDD (Dysthymia) or ASD.

**Table 13: Comorbid conditions predicting ADHD and follow up ODD group membership (n=150) compared to ADHD alone group at follow up (n=60)**

| | +ODD | -ODD | Wald | df | P | OR | (95%CI) |
|---|---|---|---|---|---|---|---|
| **ODD** | **88%** | **55%** | **25.12** | **1** | **<0.0005** | **6.26** | **(3.05-12.82)** |
| **CD** | **57%** | **27%** | **14.32** | **1** | **0.005** | **3.57** | **(1.85-6.87)** |
| **Panic Disorder** | 10% | 20% | 3.48 | 1 | 0.06 | 2.20 | (0.96-5.04) |
| **Separation Anxiety Disorder** | 30% | 28% | 0.05 | 1 | 0.82 | 0.93 | (0.48-1.80) |
| **Social Anxiety Disorder** | 35% | 38% | 0.21 | 1 | 0.65 | 1.16 | (0.62-2.15) |
| **GAD** | **39%** | **55%** | **4.50** | **1** | **0.03** | **0.52** | **(0.28-0.95)** |
| **MDD** | 19% | 25% | 0.19 | 1 | 0.34 | 0.71 | (0.35-1.44) |
| **PDD (Dysthymia)** | 40% | 45% | 0.42 | 1 | 0.52 | 0.82 | (0.45-1.50) |
| **ASD** | 36% | 21% | 2.05 | 1 | 0.15 | 2.06 | (0.77-5.56) |

Note: ODD: Oppositional Defiant Disorder; CD: Conduct Disorder; MDD: Major Depressive Disorder; PDD: Persistent Depressive Disorder (Dysthymic Disorder); GAD: Generalised Anxiety Disorder; ASD: Autism Spectrum Disorder

## 7.5 DISCUSSION

The aim of study three was to investigate the role of comorbid conditions and ADHD subtype in the development or persistence of comorbid ADHD and ODD. It was hypothesised that ODD, CD and ADHD-C would act as risk factors for the development or persistence of ODD in young people with ADHD. These hypotheses were supported by the findings: a diagnosis of ADHD-C, ODD and CD at baseline significantly predicted a young person would have ADHD and ODD at follow-up. Interestingly, GAD at baseline significantly predicted that a young person would not have comorbid ADHD and ODD at follow-up, suggesting GAD is a protective factor in the development of ODD for young people with ADHD. No group differences were found for Panic Disorder,

Separation Anxiety Disorder, Social Anxiety Disorder, Major Depressive Disorder, Persistent Depressive Disorder or Autism Spectrum Disorder.

### 7.5.1 ADHD Combined Presentation

The finding that young people with ADHD-C (compared to ADHD-I) at baseline were clearly more likely to predict membership of the ADHD and ODD group at follow up is consistent with studies that have found variations of difficulties overtime for the different subtypes of ADHD (Freitag et al., 2012; Lacramioara & Eugene, 2007; Massetti et al., 2008; Meyer & Sagvolden, 2006; Wilens & Spencer, 2010). Freitag et al. (2012) reported hyperactive-impulsive symptoms appeared to be more related to risk factors for ODD/CD in the ADHD population. Other researchers found that individuals with ADHD-C typically manifested more severe ADHD core symptoms and these symptoms were relatively more stable across developmental stages compared to ADHD-HI and ADHD-I presentations (Lacramioara & Eugene, 2007; Meyer & Sagvolden, 2006; Wilens & Spencer, 2010). In contrast, studies have linked inattentive symptoms to higher rates of internalising problems, anxiety/depression, withdrawn/depressed behaviour and executive problems (Dulcan & Lake, 2012; Graetz et al., 2001; Wilens et al., 2002).

This finding is also consistent with the literature reporting that ADHD-C and ODD are commonly comorbid (Biederman & Faraone, 2005; Lacramioara & Eugene, 2007; Wilens & Spencer, 2010). It could also be associated with parents of children with the ADHD-C presentation reporting more stress than mothers of children with the ADHD-I presentation (Lewis, 1992; Miranda et al., 2007). Miranda et al. (2007) found that mothers reported that children with ADHD-C were more likely to be distracted, less amenable to environmental changes, moodier, and more challenging to rear than children with ADHD-I. Parents of children with ADHD-C reported that they felt less reinforced in their parenting role, more isolated, felt less parenting competence, more depressed, less emotionally attached to their children, and more overwhelmed in their parenting role (Miranda et al., 2007).

This study highlights the importance of being able to prevent the development of ODD, as when ODD is comorbid with ADHD-C it is a major contributor to difficulties in optimally managing ADHD-C (Biederman, Faraone, Keenan, Steingard, & Tsuang, 1991; Guilherme Polanczyk et al., 2007; Wilens & Spencer, 2010). The use of

pharmacological interventions has proven to be effective in the treatment of ADHD. However, medications are not always acceptable to parents and young people and are not always associated with functional improvements (for example, social difficulties, learning and behavioural difficulties, the quality of parenting, parenting stress or parental psychopathology). Although, there is not enough evidence to suggest that non-pharmacological treatments (for example, Parent Management Training and Cognitive Behavioural Therapy for young people) for ADHD significantly reduce the core symptoms of ADHD, they can be useful in managing significant secondary functional impairments (parental stress, emotion regulation difficulties, social skill difficulties) associated with ADHD that are not optimally improved by medication and may be helpful in the prevention of ODD. More systematic longitudinal research needs to focus on combined pharmacological and non-pharmacological treatments for ADHD in the prevention of future ODD for young people with ADHD-C.

### 7.5.2 Oppositional Defiant Disorder

It is not surprising that young people with ADHD and ODD at baseline were found to be clearly more likely to have ODD at follow up: as already mentioned, ADHD and ODD are commonly comorbid (Biederman & Faraone, 2005; Lacramioara & Eugene, 2007; Wilens et al., 2002); and there is a growing body of evidence that suggests that prevalence rates of ODD are stable into late adolescence and trajectories of symptoms persist into young adulthood (Boylan et al., 2007; Burke et al., 2014; Leadbeater et al., 2012; Maughan, et al., 2004). In addition, early onset ODD has been found to have a more detrimental trajectory, with a two-to-threefold risk of developing conduct problems in adolescence or an Antisocial Personality Disorder in adulthood (Connor, 2002; Fraser & Wray, 2008; Loeber & Farrington, 2000; Tremblay, 2000).

These results highlight the importance of providing effective treatment for ADHD and ODD. Combined intervention (both pharmacological and psychosocial) is recommended for comorbid ADHD and ODD (Swanson et al., 2001; Zonnevylle-Bender et al., 2007). Swanson et al. (2001) found that treatment success improved 20% when combined intervention was used, relative to either psychosocial or pharmacological treatment alone. Cognitive Behavioural Therapy treatment has been found effective for ADHD and ODD: reducing externalizing symptoms; improving social competence;

increasing positive parenting; reducing internalizing behaviours; improving parent stress; and reducing maternal Depression (Battagliese et al., 2015). Most of the literature recommends intervention taking a whole of systems approach (individual, parents, school, and community) for the treatment of ADHD and ODD (Connor, 2015; Greene & Ablon, 2001; MTA Cooperative Group, 1999b). Multimodal treatments targeting both children (reducing externalizing symptoms, improving social competence) and caregivers' symptoms (for example, increasing positive parenting, reducing maternal depressive symptoms, and improving parent stress) appear important to produce sustained and generalized benefits.

Further research is needed to fully understand which features of ODD predict future difficulties: some researchers suggest that defiance, vindictiveness and aggression are associated with continued externalizing problems; whereas irritability is more associated with internalizing problems (Kolko & Pardini, 2010; Mikolajewski et al., 2017; Rowe et al., 2010; Vidal-Ribas et al., 2016). These studies suggest that underlying subtypes or dimensions of ODD are being recognised, but the actual number are still uncertain (Ghosh et al., 2017) and their relationship to developmental pathways are only just beginning to be explored.

Future research also needs to explore the bi-directional relationship between parenting behaviour and child behaviour. Researchers found that children are at higher risk of developing or maintaining Disruptive Behaviour Disorders due to a social learning and reciprocal process: over time children learn that oppositional and aggressive behaviours are effective ways to avoid undesired activities (for example, going to bed, doing chores), and parents become increasing disengaged from attempting to control their child's behaviour, thus evolving families with a lack of warmth, high rates of conflict, and poor parental monitoring (Dishion, Bullock, et al., 2002; McCart & Sheidow, 2016; Shaw et al., 2001; Snyder & Patterson, 1995). This model suggests that child behaviour can modify parenting behaviours in maladaptive ways over time (Deater-Deckard, 2000). It also highlights the importance of early intervention for young people with ADHD and ODD.

### 7.5.3 Conduct Disorder

Young people with ADHD and CD at baseline were found to be significantly more likely to have ODD at follow up. This is consistent with Rowe et al. (2010) who found

CD predicted future behavioural difficulties. Biederman, Faraone, Milberger, Jetton, et al. (1996) found children with comorbid ADHD, ODD and CD had more severe symptoms of ODD, more comorbid psychiatric disorders, and lower Global Assessment of Functioning Scales scores. Steiner et al. (2007) found comorbid CD and ODD associated with higher rates of Mood Disorders and social impairment. Therefore, effective treatment of young people with ADHD, CD and ODD is essential. The National Institute for Health and Care Excellence (NICE) clinical guidelines for Antisocial Behaviour and Conduct Disorders in children and young people emphasizes integrated and person-centred service delivery with key priority areas: comprehensive assessment; parent training programs; foster care/guardian training programs; child-focused programs; multimodal interventions; pharmacological interventions; and improving access to services (National Collaborating Centre for Mental Health (UK) & Social Care Institute for Excellence (UK), 2013).

Given the recent clarification around CD and ODD being distinct disorders research in the comorbidity of ADHD, CD and ODD is only in its early stages. Further research is required to better explain the processes that lead to co-occurring or developmental associations between ADHD, CD and ODD (Lochman, and Matthys, 2018).

### 7.5.4 Generalised Anxiety Disorder

This study found GAD to be a protective factor for young people with ADHD against the development of ODD, which is consistent with some of the extant literature about the effect of comorbid Anxiety on the natural history and outcome of ADHD (Calhoun & Mayes, 2005; Woolston et al., 1989). However, others have found that Anxiety may lead to more severe, treatment non-responsive symptomatology in young people with ADHD (Eiraldi, Power, & Nezu, 1997; Kashani, Dandoy, & Orvaschel, 1991).

With regards to young people with ADHD developing ODD, Anxiety may act in a similar way it does with “Test Anxiety”: you need an optimal level of arousal to best compete the test (exam, performance or competitive event). However, when the Anxiety (or level or arousal) exceeds that optimum, the result is a decline in performance (Teigen, 1994). “Test Anxiety” mainly occurs because of the fear of negative evaluation (Liebert

& Morris, 1967). A young person with ADHD and an optimal level of Anxiety may fear being negatively evaluated (by peers, teachers and/or family) so they inhibit inappropriate behaviour (defiance, irritability, uncooperativeness and temper outbursts) acting as a protective factor against developing ODD. Tannock (2009) found that young people with ADHD and Anxiety worried about "competency and performance in the areas of academics, athletics and social situations" which may influence their behavioural responses to act more appropriately in situations. This is an important area for future research, especially for the implications of effective treatment of comorbid ADHD and Anxiety.

Future research is required in the area of ADHD and Anxiety to help fully understand the impact of Anxiety on future ODD. Bilgiç et al. (2013) found that the presence of ODD symptoms was a risk factor for the development of future Anxiety symptoms in children and adolescents with ADHD, suggesting the order of the development of disorders may also be important to explore. Some researchers have found that children with ADHD-I were linked to higher rates of internalizing problems, including Anxiety (Dulcan & Lake, 2012; Graetz et al., 2001; Jensen et al., 2001; Tannock, 2009), suggesting that the different ADHD subtypes may be important to development of other future diagnoses also.

### 7.5.5 Limitations

Two overall major limitations constrain the interpretation of the results presented. First, the use of a clinical sample rather than an epidemiological sample may lead to divergent findings. In particular, the severity and chronicity of functional impairments at home and school for clinically referred young people are known to drive referral to services (Mannuzza et al., 2004; Ogden & Hagen, 2018). In addition, rates of ODD are higher in clinically referred samples as is the severity of all presentations of ADHD (Mannuzza et al., 2004; Ogden & Hagen, 2018). This study used strict inclusion criteria therefore may represent a subsample of individuals with ADHD alone and ADHD with follow-up ODD. Second, it is unknown if psychosocial treatment interventions were offered to the young people in this study by other community professionals as these were not controlled. This variation may lead to divergent outcomes over time depending on

varied response of comorbid disorders to the comprehensive management offered (Waxmonsky, 2003).

Nevertheless, the clinical sample investigated evinced a number of characteristics that partially addressed these methodological limitations: the large number of referring schools, the predominantly treatment naïve cases referred by school professionals, and the standardized medication algorithms used post baseline assessment with all the cases clear responders according to criteria outlined by Weiss et al. (2019). The two follow-up groups did not differ with respect to baseline type of medication used, final dose titrated to, or duration of baseline medication treatment. They also did not differ in medication status at follow-up. So, the associations reported are most likely due to changes over the 3-year follow-up period rather than the baseline medication treatment offered. However, in future, these limitations would be addressed via careful longitudinal examination of ADHD and follow up ODD compared to ADHD alone in epidemiologically derived samples with cases receiving standardized medication and psychological interventions in the community (Sciberras et al., 2013).

## 7.6 SUMMARY

This study highlights the importance of identifying the ADHD subtype and assessing for comorbid conditions in young people with ADHD. If early intervention is offered for these disorders it may prevent the development or persistence of future ODD. The ADHD subtype and comorbid disorders at baseline that predicted future or persistent ODD were: ADHD-C, ODD and CD. GAD was found to be a protective factor, with young people with ADHD and GAD less likely to develop ODD. These results provide some clinical guidance in identifying and targeting ADHD-C and comorbid disorders for young people with ADHD to prevent the development or the persistence of ODD, which can be helpful in informing preventive and therapeutic practices. Further research is required for fully understand the protective nature of GAD**.** Future systematic research is required to understand the risk and protective factors for developing further disorders (comorbidity of three or more disorders) and understanding how to treat these comorbidities effectively.

# CHAPTER 8: Significant predictors of ADHD and follow-up ODD (Study Four) and overall summary

## 8.1 INTRODUCTION

Understanding risk factors for young people developing or maintaining mental health disorders is complex. Many researchers have been interested in the interactions between risk factors for a more multidimensional exploration of the relationships between factors. Zimet and Jacob (2001) recommended that multiple factors need to be looked at, because mechanisms are rarely so simple that they are fully explained by a single moderator or mediator. In the previous three studies child characteristics, psychosocial factors, ADHD subtype and comorbidities were explored to help us recognize individual factors that contribute to the development or persistence of ODD in young people with ADHD. This study investigates the factors found significant in the previous three studies (temperament activity level; Verbal Comprehension; Visual Spatial Working Memory; aggression; social problems; parental level of education; overall parental psychopathology, parental Obsessive Compulsive and parental Interpersonal Sensitivity; ADHD-C; ODD; CD; and GAD) to help us understand which ones contribute more to the probability of developing or maintaining ODD in young people with ADHD.

### 8.1.1 Comprehensive Models

Researchers have noted that there is a need for comprehensive models that include both risk and protective factors to explain the relationship between factors and the development of ODD in young people with ADHD (Burke et al., 2008; Kirby Deater-Deckard & Dodge, 1997; Pike et al., 1996). Danforth, Connor, and Doerfler (2016) suggested the development of co-morbid ADHD and ODD reflects the culmination of interactions amongst genetic and environmental factors. Specifically, genetic factors contribute to child behaviours that are challenging (for example, impulsivity), and these behaviours are likely to provoke parenting approaches that negatively impact on the parent-child relationship, creating interactive circumstances that progress to increase the young person's behavioural problems over time (Burke et al., 2008; Danforth et al., 2016). Burke et al. (2008) found that child disruptive behaviour symptoms exerted a greater influence on parenting practices, than parenting behaviour did on child symptoms, focusing on the reciprocal interactions between parents and children being important. ODD symptoms predicted decreases in parental involvement, poorer communication, and timid discipline over time (reluctance to engage in disciplinary practices for fear of

escalating oppositional behaviours) (Burke et al., 2008). Cox and Paley (2003) proposed that families are a dynamic and interactive system consisting of interdependent subsystems, including whole-family factors, parent-child subsystem, co-parenting subsystem, and marital subsystem. Therefore, different associations of multi-level family factors and child symptoms should be explored together. Future research looking at multiple factors is needed. Biological factors, family psychiatric and psychosocial risk factors have rarely been assessed together as risk factors for comorbid ADHD and ODD (Maughan, Taylor, Caspi, & Moffitt, 2004).

### 8.1.2 Longitudinal Studies for Disruptive Behaviour Disorders

Limited longitudinal studies have looked at the interactions among several of the Disruptive Behaviour Disorders (ODD and CD) risk factors. In a community sample, Lavigne et al. (2015) examined the associations of context (stress and family conflict); parent (Depression); parenting (hostility, support, and scaffolding); and child factors (receptive vocabulary; negative affect; effortful control; inhibitory control; attachment; and sensory regulation). They found that higher scores on family risk factors (family conflict, parent hostility in parenting) and child temperament were positively associated with child ODD symptoms. They also looked at both parent and teacher ratings of child ODD symptoms, showing that there was poor agreement between parents and teachers, suggesting that ratings by different raters should not be considered to be equivalent. Similarly, Smeekens et al. (2007) looked at four domains of factors in a community longitudinal study: parent-child interaction and parent-child attachment; child characteristics (temperament and cognitive abilities); parental characteristics (personality); and contextual characteristics (SES, partner support, and stressful life events). They found that multiple family factors (parent-child interaction, parent-child attachment, and various parental, child, and contextual characteristics) served as predictors of child later externalizing behaviour problems. However, it is not known if these are the same for young people when they have ADHD.

### 8.1.3 ADHD and ODD Cross-Sectional Studies

Noordermeer et al. (2017) looked at the risk factors for ADHD and ODD in a cross-sectional study examining: pre and perinatal risk factors (pregnancy duration , birth

weight, maternal smoking during pregnancy); transgenerational factors (parental ADHD, parental warmth and criticism); and postnatal risk factors (parental SES, adverse life events, deviant peer affiliation). For ADHD and ODD they found adverse life events, parental ADHD, parental criticism, deviant peer affiliation, parental SES and higher birth weight acted as risk factors. Lin et al. (2013) studied a group of young people with ADHD using a cross-sectional study method and found family factors at the family unit level were less correlated with ODD symptoms than factors at the dyadic (including, couple dyadic and parent-child dyadic levels) and individual levels (including, parental individual and child individual levels). They recommended that further research using a longitudinal design is needed to examine the direction of the associations between multilevel family factors and child ODD symptoms and potential mediating effects.

### 8.1.4 ADHD and Disruptive Behavioural Disorders Longitudinal Studies

Three longitudinal studies have looked at the role of different factors in the development of Disruptive Behaviour Disorders (ODD/CD) in children with ADHD. These studies found that children who went on to develop the comorbid diagnosis of ADHD and ODD/CD had significantly more deviant scores throughout development on a range of factors including: child characteristics (difficult temperaments, attention problems and more oppositional, aggressive and destructive behaviours); parent characteristics (elevated levels of maternal depressive, aggressive personality symptoms and higher levels of rejecting parenting); and community characteristics (less social support and living in more dangerous neighbourhoods) (Burke et al., 2008; Chronis et al., 2007; Deault, 2010; Latimer et al., 2003). No longitudinal study, to the knowledge of the author, has looked at the development of ODD (independently of CD) for young people with ADHD. Longitudinal research is needed to investigate the different child, parent and family domains that can either be a risk factor or a protective factor for developing ODD in young people with ADHD.

### 8.1.5 Summary

Many previous studies have used correlational designs, which compare the patterns of association between parent and child variables but are unable to delineate the trajectory associated with the onset or development of comorbid conditions (Deault, 2010). Longitudinal studies that evaluate whether symptoms of one disorder predict later

symptoms of a second disorder can help to understand the effects of one disorder on the development of another disorder (Deault, 2010; Harvey et al., 2016). Literature evaluating the relationship between ADHD and later Disruptive Behavioural Disorders has revealed mixed findings and externalising disorders have not been looked at individually. Further clarification using longitudinal designs of the specific child and psychosocial factors predicting the development of ODD, or ongoing ODD, in young people with ADHD, and what factors are protective against ODD is needed. This study's findings may help to inform targeted ADHD treatments, by including targeted treatment of key child characteristics, parental psychopathology, and family functioning domains.

## 8.2 AIMS AND HYPOTHESES

The aim of study four is to help understand which of the significant factors found in study one (child characteristic factors), study two (psychosocial factors) and study three (ADHD subtype and comorbid conditions) better explain the risk factors for young people with ADHD developing or maintaining ODD. The hypothesis to be tested is that young people with ADHD and follow up ODD, compared with those diagnosed with ADHD without ODD, will manifest differential key risk factors: increased aggression (both parent and teacher reported); social problems (both parent and teacher reported); have a mother with decreased educational attainment; increased parental psychopathology; ADHD-C; and ODD.

## 8.3 METHODS

Two changes to the data set were made to ensure not too many variables were used in the logistic regression model to ensure that relevant regressors were entered to increase the precision of the estimated coefficients and predicted values. Firstly, only mothers' level of education (not fathers' level of education) was entered, as there were more data for mothers and mothers have typically been looked at in previous research. Secondly, the categories for mothers' level of education were collapsed from seven categories to four, due to low numbers in the categories for those who only completed

primary school or less. Mother's education was coded in four categories: less than high school graduation; high school graduation; TAFE graduation; university graduation.

All significant Child Characteristic Factors, Psychosocial Factors, ADHD subtype and Comorbid Conditions from study one, two and three were compared separately between the ADHD and ODD and ADHD without ODD groups at follow up. Child Characteristic Factors included: Verbal Comprehension as defined by the WISC-4 (Wechsler Intelligence Scales for Children, Fourth Edition (Wechsler, 2003)); Temperament Activity level as defined by the Infancy Temperament Questionnaire (Thomas & Chess, 1977); Visual Spatial Working Memory, Between Search Errors as measured by the CANTAB using tasks from the Cambridge Neuropsychological Test Automated Battery ("CANTAB for Windows," 1999); DESR difficulties as defined by Anxiety/Depression, Attention and Aggression scales (aggregate scores greater than and equal to 180) of the CBCL (parent) (Achenbach & Rescorla, 2001); Aggression difficulties as defined by the Aggression Scale of the CBCL (parent) and TRF (teacher) (Achenbach & Rescorla, 2001; Spencer et al., 2011); and Social problems as defined by the Social Problems subscale of the CBCL (parent)/TRF (teacher) (Achenbach & Rescorla, 2001). Psychosocial Factors included: mothers level of education as defined by the Parental Account of Childhood Symptoms, demographic and developmental sections (PACS) (Taylor et al., 1986) (modified); and Parental psychopathology defined by the Hopkins Symptom Checklist (Derogatis et al., 1974) for the Obsessive Compulsive subscale, Interpersonal Sensitivity subscale and overall Total psychopathology. Diagnosis was ascertained by the Anxiety Disorders Interview for Children (A-DISC) (Silverman & Albano, 1996) for ADHD-C, ODD, CD and GAD.

Three separate backwards stepwise logistic regressions ($p=<.1$) were completed for [1] all significant child characteristic factors (Verbal Comprehension, Visual Spatial Working Memory (between search errors), DESR difficulties (parent), Aggression (parent and teacher reported), and social problems (parent and teacher reported)), [2] significant psychosocial factors (Mothers education level, Total Parental Psychopathology, Interpersonal Sensitivity subscale, and Obsessive Compulsive Subscale) and [3] ADHD-C and significant comorbidities (ODD, CD and GAD). The cut-off of $p<.1$, instead of the usual $p<.05$, was used as we were identifying potential predictor variables (already found significant) rather than to test a hypothesis, consistent with previous research (Ranganathan, Pramesh, & Aggarwal, 2017).

A backwards stepwise logistical regression (p=<.1) of the nine factors found significant from the logistic regressions listed above was used to determine the variable selection for the inclusion in the final regression model that best predicted membership of the ADHD and follow-up ODD group.

## 8.4 RESULTS

All significant Child Characteristic factors from study one (Verbal Comprehension, Temperament Activity, Visual Spatial Working Memory (Between Search Errors), DESR (parent CBCL), Aggression (both parent CBCL and teacher TRF), and Social Problems (both parent CBCL and teacher TRF) were entered into a backwards stepwise logistic regression analyses to ascertain the strength of their prediction of ADHD and ODD follow up group membership compared to the ADHD without ODD at follow up group (see Table 14 for significant results).

**Table 14: Significant Child Characteristic factors predicting ADHD and follow up ODD group membership (n=150) compared to ADHD alone group at follow up (n=60)**

| Child Characteristics Factors | Wald | Df | P | OR | (95%CI) |
|---|---|---|---|---|---|
| Temperament Activity | 5.89 | 1 | 0.02 | 2.84 | (1.22-6.75) |
| Aggression (Parent CBCL) | 7.37 | 1 | 0.007 | 9.68 | (1.88-49.79) |
| Social Problems (Teacher TRF) | 5.3 | 1 | 0.02 | 1.07 | (1.01-1.14) |

All significant psychosocial factors from study two (mothers education level, Obsessive Compulsive subscale, Interpersonal Sensitivity subscale and overall Total psychopathology) were entered into a backwards stepwise logistic regression analyses to ascertain the strength of their prediction of ADHD and ODD follow up group membership compared to the ADHD without ODD at follow up group (see Table 15 for significant results).

**Table 15: Significant Psychosocial factors predicting ADHD and follow up ODD group membership (n=150) compared to ADHD alone group at follow up (n=60)**

| Psychosocial Factors | Wald | df | P | OR | (95%CI) |
|---|---|---|---|---|---|
| Mothers Education Level | 10.38 | 1 | 0.02 | | |
| Total Parental Psychopathology | 3.86 | 1 | 0.05 | 1.01 | (1.0-1.03) |

All significant ADHD subtype and comorbid condition factors from study three (ADHD-C, ODD, CD and GAD) were entered into a backwards stepwise logistic regression analyses to ascertain the strength of their prediction of ADHD and ODD follow up group membership compared to the ADHD without ODD at follow up group (see Table 16 for significant results).

**Table 16: Significant Comorbid factors predicting ADHD and follow up ODD group membership (n=150) compared to ADHD alone group at follow up (n=60)**

| Comobid Factors | Wald | df | P | OR | (95%CI) |
|---|---|---|---|---|---|
| ADHD-C | 2.87 | 1 | 0.09 | 2.18 | (0.89-5.35) |
| ODD | 6.16 | 1 | 0.01 | 3.50 | (1.3-9.46) |
| CD | 3.40 | 1 | 0.07 | 2.22 | (0.95-5.19) |
| GAD | 8.60 | 1 | 0.003 | 0.30 | (0.14-0.67) |

Note: ADHD-C: Attention Deficit Hyperactivity Disorder – Combined Type; ODD: Oppositional Defiant Disorder; CD: Conduct Disorder; GAD: Generalised Anxiety Disorder

All factors found significant from the three backwards stepwise logistic regressions above: child characteristic factors (temperament activity, parent (CBCL) Aggression and teacher (TRF) social problems); psychosocial factors (mothers education level and Total parental psychopathology); and comorbid conditions factors (ADHD-C, ODD, CD and GAD) were entered into a backwards stepwise logistic regression analyses to ascertain the strength of their prediction of ADHD and ODD follow up group

membership compared to the ADHD without ODD at follow up group (see Table 17 for significant results).

**Table 17: Overall significant factors that best predict ADHD and follow up ODD group membership (n=150) compared to ADHD alone group at follow up (n=60)**

| Factors | Wald | df | P | OR | (95%CI) |
|---|---|---|---|---|---|
| Temperament Activity level | 5.89 | 1 | 0.02 | 2.84 | (1.22-6.75) |
| Aggression (parent report) | 7.37 | 1 | 0.007 | 9.68 | (1.88-47.79) |
| Social problems (teacher report) | 5.3 | 1 | 0.02 | 1.07 | (1.01-1.14) |

Table seventeen presents the overall significant factors assessed for the ADHD and ODD and ADHD alone groups at follow up that best predicted ADHD and follow up ODD group membership. Temperament activity level, aggression (parent report) and social problems (teacher report) were increased in the ADHD and ODD group compared to the ADHD alone group.

## 8.5 DISCUSSION

The aim of study four was to investigate the role of all significant child characteristic factors, psychosocial factors, comorbid conditions, and ADHD subtype together, to find the best predictors in the development or persistence of ODD in young people with ADHD. It was hypothesised that young people with ADHD and follow up ODD, compared with those diagnosed with ADHD without ODD at follow up, will manifest differential key risk factors: increased aggression (both parent and teacher reported); social problems (both parent and teacher reported); have a mother with decreased educational attainment; increased parental psychopathology; ADHD-C; and baseline ODD. The factors found that best predicted membership in the ADHD and ODD follow-up group were temperament activity level, parent reported aggression and teacher reported social problems, supporting half of the proffered hypotheses. The factors of

teacher reported aggression, mothers' level of education, parental psychopathology, ADHD-C, ODD, CD and GAD were eliminated as predictors in the overall analyses.

These findings suggest that child characteristics better explain the risk factors for young people with ADHD developing or maintaining ODD than psychosocial factors, ADHD subtype or comorbidies. These findings are consistent with research by Lin et al. (2013) examining young people with ODD, finding family factors at the whole level were less correlated with ODD symptoms than factors at the dyadic and individual levels, with the factors directly related to children (parent-child relationship and child emotion regulation) showing significant association with child behavioural ODD symptoms. These findings are consistent with literature suggesting that child disruptive behaviour symptoms exert a greater influence on parenting practices and parental psychopathology, than parent behaviour does on child symptoms (Burke et al., 2008; Danforth et al., 2016; Johnston & Mash, 2001; Neece et al., 2012).

These findings highlight that when assessing young people for ADHD wider criteria than listed in DSM-5 and obtaining information from multiple informants should be considered as routine practice. Examining ADHD symptoms of inattention, disorganization, and/or hyperactivity-impulsivity does not enable identification of aggression and social problems. Assessing temperament, aggression and social problems could aid in targeting young people with ADHD with difficulties in these areas receiving early intervention that could prevent the development of ODD. These findings may help to clarify the importance of including Parent Management Training, emotional regulation training, problem solving and social skills development programs into the treatment plans for young people with ADHD (especially ADHD-C). Helping parents to manage their child's activity level, aggression and role model appropriate problem solving and social skills may be important to assist in the prevention of ODD for children with ADHD. In addition, teaching young people emotional regulation skills, problem solving, and social skills may also be beneficial.

This study strengthens the argument of the current policy directions towards earlier intervention and prevention for ODD, addressing early risk factors, before secondary risk factors develop (Webster-Stratton & Reid, 2018). Providing emotional regulation and social skills programs in routine curricula from primary school may not only benefit those young people at risk of developing ODD, but may assist young people

who are at risk of developing other mental health difficulties, by enhancing and strengthening their protective factors.

It is important not to forget that both the ADHD alone and ADHD and ODD at follow up groups showed clinically significant difficulties with family functioning and parent psychopathology (see chapter six, study two). Clinically these findings cannot be ignored as they signify the importance of assessing and treating family functioning difficulties for young people with ADHD and/or ODD. They also emphasize the importance of using a systematic approach for both assessment and treatment of young people with ADHD and their families. Ensuring families have effective family functioning, which includes good communication skills, the ability to maintain relationships and the ability to make decisions and solve problems effectively is essential (Epstein et al., 1993; Silburn et al., 2006; Zubrick et al., 2000). Parental psychopathology must also be assessed and addressed to ensure the most effective treatment plans for young people with ADHD. The effectiveness of training parents of children with ADHD in behavioural interventions is suggested by several studies, which show these parents have high levels of stress as well as a lack of confidence in their parenting skills when compared with parents of typically developing control participants (August et al., 1999; Danforth et al., 2006; Hartman et al., 2003; Johnston & Mash, 2001) and have been found helpful in reducing pre-schoolers ADHD symptoms (Sonuga-Barke et al., 2018). Early treatment, positive parenting, and the absence of parent mental health difficulties may all optimize functioning and likely improve the course of young people with ADHD (Modesto-Lowe et al., 2008).

### 8.5.1 Limitations

Three overall major limitations identified in the previous chapters that constrain the interpretation of the results are relevant when considering them. First, the use of a clinical sample rather than an epidemiological sample may lead to divergent findings. In particular, the severity and chronicity of functional impairments at home and school for clinically referred young people are known to drive referral to services (Mannuzza et al., 2004; Ogden & Hagen, 2018). In addition, rates of ODD are higher in clinically referred samples as is the severity of all presentations of ADHD (Mannuzza et al., 2004; Ogden & Hagen, 2018). This study used strict inclusion criteria and therefore may represent a

subsample of individuals with ADHD alone and ADHD with follow-up ODD. Second, it is unknown if psychosocial treatment interventions were offered to the young people in this study by other community professionals as these were not controlled. This variation may lead to divergent outcomes over time depending on varied response of comorbid disorders to the comprehensive management offered (Waxmonsky, 2003). Third, three factors (emotional regulation, aggression, and social problems) were measured using the same instrument that were used to define the ADHD and follow up ODD groups (the CBCL). This is especially true for the aggression factors given that ODD DSM Scale uses some of the items (argues, disobedient at home, disobedient at school, stubborn and has temper tantrums) used to define the Aggressive Behaviour subscale. This issue was considered when selecting measurement instruments for this study. However, it was thought to add additional questionnaires for both parents and teachers to complete would risk an excessive amount of questionnaires to complete, that could have resulted in poor completion rates, reduced motivation and accuracy of reporting (McColl et al., 2001). Interestingly, when looking at the mean and standard deviation for each of these factors the scores reflect a similar range to the general population (from the normal range, through to a clinical range), suggesting that in this case they are appropriate measures.

Nevertheless, the clinical sample investigated evinced a number of characteristics that partially addressed these methodological limitations: the large number of referring schools, the predominantly treatment naïve cases referred by school professionals, and the standardized medication algorithms used post baseline assessment with all the cases clear responders according to criteria outlined by Weiss et al. (2019). The two follow-up groups did not differ with respect to baseline type of medication used, final dose titrated to, or duration of baseline medication treatment. They also did not differ in medication status at follow-up. So, the associations reported are most likely due to changes over the 3-year follow-up period rather than the baseline medication treatment offered. However, in future, these limitations should be addressed via careful longitudinal examination of ADHD and follow up ODD compared to ADHD alone in epidemiologically derived samples with cases receiving standardized medication and psychological interventions in the community (Sciberras et al., 2013).

Another factor to consider is the use of a stepwise regression with a relatively small sample size that could result in explanatory variables that have causal effects on the dependent variable not being statistically significant (Smith, 2018), creating a false

confidence in the final model (Thompson, 1995) and an oversimplification of the real models of the data (Roecker, 1991). Understanding risk factors for young people developing or maintaining mental health disorders is complex and caution must be exercised so as to not overgeneralize these findings.

### 8.5.2 Future Research

Further understanding of the bidirectional nature of ODD, family functioning and parental psychopathology is crucial (Kazdin & Wassell, 1999; McKee et al., 2008; Neece et al., 2012; Chronis et al., 2003). Many researchers have noted the effects of the child's ADHD symptoms on parental behaviour (Allmann, 2018; Danforth et al., 1991; McKee et al., 2004), suggesting that child behaviour can modify parenting behaviours in maladaptive ways over time (Deater-Deckard, 2000). In addition, how the relationship between parent management skills and parental psychopathology develops and their contributions to the development, maintenance and severity of ODD in children needs careful systematic investigation (Burke et al., 2002; Kaplan & Liu, 1999). Further longitudinal research is needed to help us understand the complex interaction between parental psychopathology, parent management skill and young people with ADHD developing ODD.

Future longitudinal studies addressing the interactions of multiple factors (child characteristics, psychosocial factors, ADHD subtype and comorbidities) are required. Previous research has acknowledged that building models to help us understand the interactions of multiple factors is complex and requires further consideration. A number of models have been proposed, but are only in the early stages of evaluation: Cox and Paley (2003) proposed that families are a dynamic and interactive system consisting of interdependent subsystems, including whole-family factors, parent-child subsystem, co-parenting subsystem, and marital subsystem. Lin et al. (2013) proposed a three-level model that delineated the mechanism of ODD development (based on the Family System Theory (Cox & Paley, 2003) and the Bioecological Model (Bronfenbrenner, 2009)). This model divided family factors into three levels, including the whole level, the dyadic level (including couple dyadic and parent-child dyadic levels) and the individual level (including parental individual and child individual levels). This study (study 4) looked at two levels: child characteristics; and psychosocial factors. The findings of this study

suggested the best predictors for ADHD and ODD at follow-up at the child characteristics level were: temperament activity; parent reported aggression; teacher reported social problems; ADHD-C; ODD; and CD. GAD was a protective factor. At the psychosocial level, the best predictors were: mothers' education level; and total parental psychopathology. Gaining a more comprehensive understanding of how these factors relate and what model best helps us to understand these interdependent subsystems is imperative in being able to target effective intervention in the prevention of ODD. This work is indeed complex and requires further development and exploration.

Future research addressing the effectiveness of combined systemic treatment (pharmacological and non-pharmacological) for young people with ADHD (particularly ADHD-C) and their families in the prevention of ODD for young people is crucial. Development of clinical guidelines for ADHD that include emerging evidence based non-pharmacological interventions for both the young person and their family need to be put forward to assist clinicians to achieve effective long term outcomes. Longitudinal research assisting clinicians to understand the protective nature of Anxiety in young people with ADHD preventing the development of ODD also needs further exploration.

## 8.6 OVERALL SUMMARY

These four studies expand the literature investigating the risk and protective child and psychosocial factors linked with the development of ODD in young people with ADHD. It also helps explain the relationship between these factors. It highlights key child characteristics (temperament activity, parent reported aggression, teacher reported social problems), psychosocial factors (mothers education level, Total psychopathology), ADHD-C and comorbidities (ODD, CD) that place young people with ADHD more at risk of developing or maintaining ODD. Interestingly GAD was found to be a protective factor in preventing the development of ODD. Overall, three factors were found to be the best predictors of young people with ADHD developing ODD: child temperament activity level; parent reported child aggression; and teacher reported child social problems. This highlights the importance of assessing for broader difficulties than just the core ADHD diagnostic symptomatology. Although not found as significant predicting factors for young people with ADHD developing ODD, family functioning

difficulties and parental psychopathology were found to be clinically significant for both the ADHD alone and ADHD with ODD at follow up groups. Therefore, they must also be assessed, and if difficulties in these areas found, included in treatment plans for effective treatment for young people with ADHD. Interpretation of these results are constrained by this study using strict inclusion criteria and a clinical sample rather than an epidemiological sample. Therefore, it may only represent a subsample of young people with ADHD (Mannuzza et al., 2004; Ogden & Hagen, 2018). Future longitudinal research is needed using epidemiological samples and controlling both medication and psychosocial treatment interventions. Further understanding of the interactions between multiple child and family factors and their bidirectional nature in the development ODD in young people with ADHD is important. Developing effective combined child and family treatment, both pharmacological and non-pharmacological, is essential in the prevention of ODD for young people with ADHD.

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