THE RELATIONSHIP BETWEEN ATTENTION-DEFICIT/HYPERACTIVITY DISORDER AND ADVERSE EXPERIENCES IN CHILDHOOD.

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ABSTRACT

Objective: Attention-Deficit/Hyperactivity Disorder (ADHD) is currently the most commonly diagnosed psychiatric disorder among school aged children. Various studies have found an association between ADHD and adverse childhood experiences. However, there are many conflicting hypotheses about the nature and direction of this relationship. The first aim of the study was to establish whether there is a relationship between adverse childhood experiences and the symptoms, and diagnoses, of ADHD, Conduct Disorder (CD), Oppositional Defiant Disorder (ODD), and Post Traumatic Stress Disorder (PTSD). Secondly, the study aimed to determine whether there is a group of children/adolescents with symptoms of ADHD that constitute a 'complicated ADHD' group. The study aimed to investigate whether this 'complicated ADHD' group had higher rates of adverse childhood experiences, higher rates of diagnostic co-morbidity, and more serious externalizing symptoms.

Methodology: The clinical records of 217 consecutively admitted clients of a New Zealand Child and Adolescent Mental Health Service (CAMHS) were read in their entirety. Information regarding diagnoses, symptomology, and adverse childhood experiences was recorded and analysed. Latent class analysis was used to group children and adolescents into homogenous groups based on their profiles of ADHD, ODD and CD symptoms.

Results: The study found significant associations between adverse childhood experiences and the symptoms of ADHD, PTSD, and other disruptive behavioural disorders. Latent class analysis revealed a group of children and adolescents who had more serious externalizing symptoms (a high number of ADHD, ODD and CD symptoms) and a high rate of diagnostic co-morbidity. This group had a higher average rate of adverse childhood events, physical abuse, and

exposure to domestic violence, compared to the other children and adolescents with symptoms of ADHD.

Conclusions: The findings from this study confirm other findings that early adversive childhood experiences place children and adolescents at significant risk of developing ongoing issues with inattentiveness, hyperactivity and impulsivity. Furthermore, the study provides tentative evidence that early adversive experiences may create a more complicated profile of ADHD symptoms and perhaps impact the way that ADHD symptoms manifest.

DEDICATION

~ I dedicate this thesis to my family ~

Mum, Dad, Jamie, Sarah, Nana, Kylie, and Sam.

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The following people provided valuable input and helped contribute towards the completion of this thesis. I gratefully acknowledge their help and support:

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LIST OF ABBREVIATIONS

ADHD   *Attention Deficit Hyperactivity Disorder*

ADHD-NOS   *Attention Deficit Hyperactivity Disorder Not Otherwise Specified*

APD   *Antisocial Personality Disorder*

BASC   *Behavior Assessment System for Children*

BPT   *Behavioural Parent Training*

CAMHS   *Child and Adolescent Mental Health Service*

CBCL   *Child Behaviour Checklist*

CD   *Conduct Disorder*

CDI   *Child Depression Inventory*

CNV   *Copy Number Variants*

CPRS   *Conners Parent Rating Scale*

CTRS   *Conners Teacher Rating Scale*

CYFS   *Child Youth and Family Services*

DAT1   *Domapamine Transporter Gene*

DRD4   *Dopamine D4 Gene*

DRD5   *Dopamine D5 Receptor Gene*

DSM-II   *Diagnostic and Statistical Manual of Mental Disorders (2nd edition).*

DSM-III   *Diagnostic and Statistical Manual of Mental Disorders (3rd edition).*

DSM-IV   *Diagnostic and Statistical Manual of Mental Disorders (4th edition).*

DSM-IV-TR   *Diagnostic and Statistical Manual of Mental Disorders (4th edition, text revision).*

FAS	*Feotal Alchohol Syndrome*
GAF	*Global Assessment of Functioning*
HTR1B	*Serotonin Recepter Gene*
ICD-10	*International Classification of Disease (10th edition.).*
IYPP	*Incredible Years Parenting Programme*
LCA	*Latent Class Analysis*
MRI	*Magnetic Resonance Imaging*
MTA	*Mulitmodal Treatment Study*
NHI	*National Health Index Number*
O-3FAS	*Omega-3 Fatty Acid Supplement*
ODD	*Oppositional Defiant Disorder*
OECD	*Organization for Economic Cooperation and Development*
PTSD	*Post Traumatic Stress Disorder*
SNAP-25	*Synaptosomal Associated Protein 25 Gene*
SPSS	*Statistical Package for the Social Sciences*
Triple P	*Positive Parenting Programme*
UK	*United Kingdon*
UNICEF	*United Nations International Children's Emergency Fund*
USA	*United States of America*
WHO	*World Health Organisation*
WMT	*Working Memory Training*

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CHAPTER I: INTRODUCTION

SYNOPSIS

The purpose of the study is to explore, and attempt to disentangle, the complicated relationship between Attention Deficit-Hyperactivity/Disorder (ADHD) and adverse childhood experiences. This study examines the diagnoses, symptoms, and rates of adverse childhood events of children and adolescents at a New Zealand Child and Adolescent Mental Health Service (CAMSH). The following literature review provides an overview of ADHD including: history, diagnostic features, assessment, diagnostic complications, co-morbidity, and prognosis. This is followed by a section on the main treatment approaches for ADHD. The main theories of the aetiology of ADHD will then be discussed. This is followed by a section on childhood adverse experiences and sequential psychopathology. The final section examines the empirical link between adverse experiences in childhood and attention deficit hyperactivity disorder, which will lead to the aims of the current study.

INTRODUCTION TO ATTENTION-DEFICIT/HYPERACTIVITY DISORDER

History of Attention-Deficit/Hyperactivity Disorder

For the past 100 years, 'ADHD' has been a major focus of research. The identification of an ADHD-type syndrome dates back to the early 1900's, and since this time the definition of 'ADHD' and theories around the aetiology of the disorder have continuously evolved.

In 1902, George Frederick Still described a group of children as having a deficit in "volitional control or a deficit in moral control over their behaviour" (Barkley, 1997, p. 4). Some of the symptoms observed in these children were; inattentiveness, hyperactivity, impulsivity, aggressive behaviour, excessive emotion, decreased inhibition, spitefulness, cruelty, and overactivity. Thus, the symptoms observed among this group of children were very similar to what

is now known as ADHD. Still (1902) noted that these symptoms were more prominent in male children.

Two decades later, during the 1917-1918 encephalitis pandemic, Ebaugh (1923) observed children who had survived brain infections and described these children as having high levels of impulsivity, quarrelsome behaviour, and being unable to progress in school. Furthermore, they were difficult to manage in the classroom, frequently disturbed their classmates, and did not respond well to discipline. These observations lead to the idea that children exhibiting ADHD-type symptoms were behaving this way as a result of brain damage (Ebaugh, 1923). Thus in the 1930's these children were thought to be suffering from 'brain damage syndrome' (Bradley, 1937).

Throughout the early to mid 1900's, children exhibiting ADHD-type symptoms were labelled with various psychiatric terms. Some of these labels included 'strauss syndrome', 'hyperactive child syndrome', and 'hyperkinetic impulse disorder' (Mash & Barkley, 1996; Strauss & Lehtinen, 1947; Weinstein, Staffelbach, & Biaggio, 2000; Whitlock, 2008).

In the 1960's the idea that ADHD-type symptoms were a consequence of brain damage shifted towards a more psychoanalytic based hypothesis (Barkley, 1997). This hypothesis suggested that behavioural abnormalities in childhood arose as a reaction to various environmental factors. Consequently, the *Diagnostic and Statistical Manual of Mental Disorders – II (DSM-II)* included 'hyperkinetic reaction of childhood disorder' as a category for children exhibiting ADHD-type symptoms that did not have a history of organic brain damage (American Psychiatric Association, 1968).

It was not until the 1980's that attention-deficit disorder (ADD) was first described in the *DSM-III* (APA, 1980). The *DSM-III* diagnosis of ADD provided more explicit criteria for defining the disorder. It also emphasised ADD as a cognitive and developmental disorder, rather than a behavioural reaction of childhood (Barkley, 1997).

In 1994 the *DSM-IV* made the most recent development and ADD was changed to ADHD. The *DSM-IV* diagnosis of ADHD has three distinct sub-types: ADHD-combined type, ADHD-predominantly inattentive type, and ADHD-predominantly hyperactive-impulsive type (APA, 1994; APA, 2000; Barkley, 1997).

Definition and Diagnostic Features of Attention-Deficit/Hyperactivity Disorder

In the *DSM-IV-TR* the 'attention deficit and disruptive behavioural disorders' section includes ADHD, Oppositional Defiant Disorder (ODD), and Conduct Disorder (CD) (APA, 2000). ADHD is defined by the *DSM-IV-TR* as a childhood disorder characterised by inattention, impulsivity, and hyperactivity to a degree that is maladaptive and developmentally impaired (APA, 2000). The disorder has a negative impact on the child's behavioural, social, academic, emotional, and cognitive domains of development (Barkley, 1997; Briscoe-Smith & Hinshaw, 2006; Weinstein, Staffelbach, & Biaggio, 2000).

The *DSM-IV-TR* criteria for ADHD contain a list of 16 symptoms spread over the three inattentive, impulsive, and hyperactive dimensions (*see Appendix A*). In order to warrant a *DSM-IV-TR* diagnosis of ADHD, at least some of the inattentive or hyperactive-impulsive symptoms have to be present before the age of seven (APA, 2000). Furthermore, some impairment from the inattentive and/or hyperactive-impulsive symptoms of ADHD must be present in at least two domains, e.g. at home and at school. Another condition for a diagnosis of ADHD is that the disorder must cause clinically significant impairment across social, academic, or occupational functioning (APA, 2000).

Similar to the *DSM-IV-TR* diagnosis of ADHD, the *International Classification of Disease, tenth revision (ICD-10)* includes 'hyperkinetic disorder' (Sandburg, 2002; World Health Organisation, 1994). Hyperkinetic disorder is based on a list of 18 inattentive, hyperactive, and

impulsive symptoms similar to those in the *DSM-IV-TR*. However, there are a few important discrepancies between the two widely used diagnostic classification systems (Sandburg, 2002). Unlike the *DSM-IV-TR,* the *ICD* does not have distinct subtypes, thus a diagnosis requires the presence of all three inattentive, hyperactive, and impulsive dimensions (WHO, 1994). Furthermore, the exclusion criteria for hyperkinetic disorder are strictly defined, with a separate disorder for hyperkinetic conduct disorder. This would be similar to a *DSM-IV-TR* diagnosis of ADHD co-morbid with CD (APA, 2000; WHO, 1994). In summary, the *ICD* diagnosis of hyperkinetic disorder constitutes a more strictly defined subset of the *DSM-IV-TR's* ADHD-combined type diagnosis (Sandburg, 2002). For the purpose of the present study, the *DSM-IV-TR* diagnosis of ADHD (including all three subtypes) will be referred to, as it is more inclusive of all individuals with inattentive and disruptive behavioural disorders as a whole.

In accordance with the *DSM-IV-TR,* individuals with ADHD-predominantly inattentive type are required to have at least six of the inattentive symptoms listed in criteria A1 (see *Appendix A*) (APA, 2000). Similarly, individuals with ADHD-predominantly hyperactive-impulsive type need to fit at least six of the hyperactive and/or impulsive symptoms listed in criteria A2. Individuals who meet the criteria for A1 and A2 are diagnosed with ADHD-combined type. Children and adults with prominent symptoms of inattention, hyperactivity, or impulsivity, that do not fit one the three sub-types of ADHD fall into the ADHD-not otherwise specified category (ADHD-NOS) (APA, 2000). Thus in accordance to the *DSM-IV-TR* criteria, those who fit a diagnosis of ADHD are a heterogeneous group who show great variance in not only their types of symptoms, but in the perseverance and severity of their ADHD (Connor, 2002; Kieling & Rohde, 2012; Nigg, 2006). Taking into account this variability when studying ADHD, it is beneficial to investigate the specific symptoms of the disorder, rather than viewing ADHD as a distinct and homogeneous disorder.

Inattention

One of the core features of ADHD is a persistent pattern of inattention to an extent that is maladaptive and higher in severity and frequency, compared to children of the same developmental level (APA, 2000; Barkley, 1997; Kieling & Rohde, 2012; Nevid, Rathus, & Greene, 2006). Mash & Barkley (2003, p.67), define inattention as "a multi-dimensional construct that can refer to problems with alertness, arousal, selective or focused attention, sustained attention, distractibility, or span of apprehension, upon others". Inattentive symptoms are observable in children with ADHD across various settings, including home, school, and social situations (APA, 2000; Kieling & Rohde, 2012).

In pre-school aged children, inattentiveness may manifest as frequent shifts in attention while playing with toys (Barkley, 1998). At school, children with ADHD may have difficulty completing tasks and listening to instructions, appear careless and messy in their school work, make frequent mistakes in school work, and have problems giving close attention to details (APA, 2000; Bennett, 2006; Kieling & Rohde, 2012). Children with ADHD also appear to be more disorganised, forgetful and distractible, compared to their aged matched pairs (Barkley, 1997; Kieling & Rohde, 2012). Inattentiveness is often observed in social situations as difficulty listening to others, frequent shifts in conversation, and having problems following the rules in games and activities (APA, 2000). While at home, inattentive children have difficulty following caregiver's instructions, problems getting organised, and difficulty completing chores and homework (APA, 2000; Barkley, 1998). Furthermore, individuals with ADHD often find tasks that require sustained mental attention 'adverse and unpleasant' (Mash & Barkley, 2003). Children with the inattentive symptoms of ADHD are often described by parents and teachers in terms such as: "doesn't seem to listen", "daydreams", "can't concentrate", or "easily distracted" (Barkley, 1998, p. 58).

Hyperactivity

Children exhibiting the hyperactive symptoms of ADHD are highly restless, active and fidgety in comparison to their aged matched peers (Barkley, 1998; Carr, 1999; Kieling & Rohde, 2012; Mash & Barkley, 2003). Parents and teachers describe children with the hyperactive component of ADHD as though they are 'driven by a motor' or 'always on the go' (APA, 2000; Kieling & Rohde, 2012). Hyperactivity often manifests as persistent fidgeting, climbing and running in inappropriate situations, difficulty sitting still, and excessive talking (APA, 2000). Hyperactivity is also observable across various settings, including school, home, and social situations (APA, 2000; Nevid, Rathus, & Greene, 2006).

The hyperactivity symptoms in children with ADHD vary in relation to the child's particular stage of development. Some studies suggest that the hyperactive and impulsive symptoms of ADHD decline with age and reside by early adolescence, whereas the inattentive symptoms are found to be more stable during the schooling years and even persist into adolescence (Barkley, 1997; Mash & Barkley, 2003; Sandburg, 2002).

Impulsivity

Individuals exhibiting impulsive symptoms of ADHD have difficulty inhibiting their responses, particularly when their response would gain immediate reinforcement (Barkley, 1997). Many studies have found children with ADHD to have poor self regulation, impulse control, and behavioural inhibition (Mash & Barkley, 2003). Children with ADHD will often respond in situations before instructions have been completed or consequences have been evaluated (Barkley, 1997; Kieling & Rohde, 2012). Consequentially, children with ADHD often blurt out answers, have difficult awaiting their turn, and engage in potentially dangerous activities without considering the consequences (APA, 2000). Furthermore, impulsivity often leads to accidents such as banging into people, knocking over objects, and grabbing hot pans (APA, 2000).

Some suggest it is the highly impulsive behaviour associated with ADHD that is the most detrimental to the individuals social functioning (Holowenko, 1999). For example, impulsive behaviours such as interrupting conversations, snatching and grabbing objects from others, and failing to listen to peers, all have a negative impact on social functioning (APA, 2000; Barkley, 1997; Holowenko, 1999). Games and situations that require cooperation and sharing with peers are particularly problematic for children with ADHD (Barkley, 1998).

Epidemiology

ADHD is currently the most common psychiatric disorder diagnosed in children, affecting approximately 3-7% of school aged children (APA, 2000; Hodgkins et al., 2012; Mayes, Bagwell, & Erkulwater, 2008). However, studies around the world report variable rates of ADHD with prevalence rates ranging from 1-26% (Barkley, 1998; Brown et al., 2001; Carr, 1999; Faraone & Biederman, 2005; Ford et al., 2000; Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007; Sandburg, 2002; Skounti, Philalithis, & Galanakis, 2007). In a review study, Brown et al. (2001) found prevalence rates among community samples of school-aged children (6-12 years) to range from 4% to 26%. Prevalence rates in preschool aged children (2-5 years) have been found to range from 2% to 59% across a variety of cultures (Connor, 2002). This large variability in prevalence rates of ADHD probably reflects the variation in what is defined as 'ADHD'. Other factors that contribute to this variability include: differences in the method of diagnosis (e.g. observing individuals in one setting rather than two), inconsistent samples (community samples vs. clinic-referred samples), along with differences in the populations examined (age, cultural and gender differences) (Brown et al., 2001; Faraone & Biederman, 2005; Sandburg, 2002; Skounti, Philalithis, & Galanakis, 2007).

ADHD is predominantly diagnosed in male children, with males being more than three times more likely to be diagnosed (APA, 2000; Das Banerjee, Middleton, & Faraone, 2007;

Hinshaw & Blackman, 2005). Females with ADHD are also found to exhibit less disruptive behaviour than males (Gershon & Gershon, 2002).

Rates of ADHD diagnosis have been found to vary between different ethnicities. In the US, white american children have been found to have higher rates of ADHD diagnoses than Hispanic, Mexican and African American children (Hodgkins et al., 2012). A review found that prevalence rates of ADHD were lower in some countries (e.g. Australia, Sweden, Italy, and Iceland), compared to the United States of America (USA) (Faraone, Sergeant, Gillberg, & Beirdman, 2003). Further research compared the prevalence of ADHD symptoms in a sample of chidlren living in the USA, to a sample of children living in the Ukraine (Gadow et al., 2000). The study found prevalence rates of 19.8% in the Ukraine and 9.7% in the US. One explanation for the high descrepancy between the two countries was that the chidlren in the Ukraine lived within 30 km of the Chernobyl neclear power plant. Many of the children in the study were in families that had been evacuated 10 years earlier. The authors suggested that the high rates of ADHD symptoms among the Ukrainian children may have related to high levels of environmental adversity and the psycho-social dislcoation associated with the Chernobyl disaster (Gadow et al., 2000). Taylor & Sandburg (1984) suggest that the descrepancy in prevalence rates found between different countries probably reflects a variation in the way ADHD is defined and diagnosed, rather than real behavioural differences between the countries.

Until recently, ADHD was considered to be a disorder of childhood and adolescence. However, recent research has found prevalence rates of adult ADHD to range from 2% to 5% (Faraone & Biederman, 2005; Rucklidge, Brown, Crawford, & Kaplan, 2006). This research suggests that ADHD is one of the most common psychiatric disorders of adulthood (Faraone & Biederman, 2005). Retrospective studies have found that 50-80% of individuals diagnosed with ADHD in childhood will show persistent symptoms of ADHD into their adult years (Weiss & Hechtman, 1993).

In recent years ADHD has become of particular concern due to rising numbers (Coleman, 2007; Hodgkins et al., 2012; Mayes, Bagwell, & Erkulwater, 2008; Radcliffe & Timimi, 2004; Taylor, 2009; Timimi & Taylor, 2004). The biggest increase in rates of ADHD diagnosis occurred in the 1990's (Mayes, Bagwell, & Erkulwater, 2008; Taylor, 2009). One study found that the rates of ADHD diagnosis in the USA increased from 947,208 to 2,357,838 (232%) from 1990-1995 (Robinson, Sclar, Skaer, & Galin, 1999). The increasing trend in the diagnosis of ADHD has shown no signs of tapering off (Taylor, 2009). Some suggest that the dramatic increase in the prevalence of ADHD over the last two decades is due to an increase in knowledge and understanding of the disorder. This has led to ADHD being more commonly recognised in practice, along with corresponding higher rates of diagnosis, rather than an increase in true prevalence rates of the disorder (Olfson, Gameroff, Marcus, & Jensen, 2003; Taylor, 2009). As stated by Taylor (2009, p. 127), "the increase in administrative prevalence – the rate with which the diagnosis is made in practice – has been striking".

In summary, ADHD is currently the most common psychiatric disorder diagnosed in school-aged children, with prevalence rates ranging from 1-26%. The large discrepancy in prevalence rates of ADHD likely reflects the variation in what is defined as 'ADHD', differences in the method of diagnosis, inconsistent sample ascertainment, and differences in the populations examined. Furthermore, rates of ADHD diagnosis have been found to vary between different ethnicities and cultures. In recent years ADHD has become of particular concern due to rising numbers. It is thought that this increaese in prevalence reflects an increaese of knowledge and awareness of ADHD, which has corresponded with a higher rate of diagnosis rather than an increase in actual prevalence of inattentive/hyperactive behavoiur.

Assessment of Attention-Deficit/Hyperactivity Disorder

In addition to the *DSM-IV-TR* and the *ICD* systems, many assessment instruments are utilized in the assessment of ADHD in children (APA, 2000; Whitlock, 2008; WHO, 1994). Behaviour check-lists and inventories have proven to be fast, easy to administer, and economical in assisting the assessment of ADHD and other disruptive behavioural disorders (Spencer, Biederman, & Wilens, 1999; Whitlock, 2008). Some of these checklists can be administered by health professionals, teachers, and/or parents (Brown et al., 2001).

Behavioural scales are either classified as ADHD-specific, which measure the core symptoms of ADHD, or broader checklists which cover a variety of behavioural problems (Brown et al., 2001). A widely used ADHD-specific measure is the revision of the Conners parent and teacher rating scales (CPRS and CTRS) (Conners, Sitarenios, Parker, & Epstein, 1998). Studies have found the CPRS/CTRS to be reliable in predicting ADHD and co-morbid externalising disorders in children (Whitlock, 2008). The CPRS and CTRS have also been shown to demonstrate high sensitivity, specificity, and predictive validity in assessing ADHD in children (Levitt, Saka, Hunter Romanelli, & Hoagwood, 2007).

Some common broad assessment tools used to identify externalising behavioural disorders are the child behaviour checklist (CBCL) and the behaviour assessment system for children (BASC) (Achenbach, 1992; Carr, 1999; Whitlock, 2008). Although these broad checklists are not generally used to specifically diagnose ADHD, they can be useful in identifying co-morbid disorders (Brown et al., 2001). The CBCL is thought to be particularly useful as it contains an inattentive symptom subscale, along with subscales for co-morbid conduct and emotional problems which are often present in ADHD (Achenbach, 1992; Carr, 1999).

Although behaviour rating scales are widely utilized in the diagnosis of ADHD, the objectivity of these scales has been questioned. Some argue that having a 'cut off' point for problematic behaviour is a culturally and subjectively driven process (Radcliffe & Timimi, 2003;

Timimi, 2004). This is supported by research that has found descrepancies in the way ADHD behaviours are rated between different cultures and ethnicities (Faraone, Sergeant, Gillberg, & Beirdman, 2003; Gadow et al., 2000; Hodgkins et al., 2012; Taylor & Sandburg (1984).

Diagnostic Complications

Although ADHD is one of the most thoroughly researched childhood disorders, the diagnostic criteria can be problematic. The development of diagnostic criteria and assessment tools for ADHD has helped provide clinicians with a common language and criteria for children exhibiting attention problems, hyperactivity and impulsivity (Faraone et al., 2003; Nevid, Rathus, & Greene, 2006). However, the development of the diagnostic criteria for ADHD has also led to a complication in the understanding of ADHD and this has led many to question the validity of ADHD as a clinical 'disorder' (Baldwin & Cooper, 2000; Faraone et al., 2003; Radcliffe & Timimi, 2004; Timimi et al., 2004; Taylor, 2009; Weinstein, Staffelbach, & Biaggio, 2000).

Many children exhibit symptoms of ADHD at some point in development and the line between 'normal' age-appropriate hyperactive behaviour and 'abnormal' behaviour can be unclear (Bennett, 2006). Some argue that a diagnosis of ADHD is merely a process of labelling children who are difficult to control at home or in the classroom (Nevid et al., 2006). Furthermore, there is a lack of consistency around a clear definition for what constitutes 'ADHD' (Taylor, 2009; Timimi et al., 2004). Over the last 30 years, the definition of ADHD has constantly evolved, and it seems the definition of ADHD now emcompasses even more children (Timimi et al., 2004). This is supported by research that has found inconsistenies in how ADHD is defined and diagnosed between different mental health professional's and across various cultures. One study investigated the way that classroom teachers rate children of various cultures, using common behaviour rating scales used to diagnose ADHD. The study found a significant association between the teacher-ratings of ADHD

symptoms and the culture/ethnicity of the child (Sonuga-Barke, Minocha, Taylor, & Sandberg, 1993). Another study examined the consistency of mental health professionals ratings of ADHD symptoms across four countries: USA, China, Indonesia, and Japan (Mann et al., 1992). The study found that mental health professionals in China and Indonesia gave significantly higher scores for hyperactive and disruptive behaviours, compared to Japanese and American mental health professionals. Similar findings have been noted even without cultural or ethnic differences. One study found that rates of ADHD diagnosis varied by a factor of 10 within the same state in the USA (Rappley, Gardiner, Jetton, & Howang, 1995).

Further complicating a diagnosis is the fact that there is currently no medical test that exists for ADHD and there are no distinguishable physical deficits (Baldwin & Cooper, 2000; Radcliffe & Timimi, 2004). Therefore, a diagnosis of ADHD is based on the subjective interpretation of the clinician making the diagnosis (Radcliffe & Timimi, 2004). Some argue the judgments on behaviour checklists made by health professionals, teachers, and parents are unreliable and invalid (Baldwin & Cooper, 2003).

In summary, research suggests that perceptions of ADHD symptoms vary significantly between countries and mental health professionals, despite using the same diagnostic system. It is clear that there are on-going issues around what constitutes "ADHD" and how the disorder is defined and diagnosed.

Co-morbidity of Attention Deficit/Hyperactivity Disorder

A diagnosis of ADHD is further complicated due to the high overlap with symptoms of other psychiatric disorders such as: ODD, CD, learning disorders, anxiety disorders and both uni-polar and bi-polar mood disorders (Bauermeister et al., 2007; Biederman, 2005; Brown et al., 2001; Lahey & Willcutt, 2002; Spencer, Biederman, & Wilens, 1999; Weinstein et al., 2000). Studies

have found 30-90% of individuals with ADHD to have at least one co-morbid psychiatric disorder (APA, 2000; Barkley, 1998; Spencer, Biederman, & Wilens, 1999; Whitlock, 2008). The very high rate of diagnostic co-morbidity challenges the specificity of the diagnosis of ADHD (Timimi & Taylor, 2004).

It is widely recognised that children and adolescents with ADHD have higher rates of oppositional and defiant behaviour, conduct problems, aggressive behaviour, and in many cases, antisocial behaviour (Bauermeister et al., 2007; Barkley, 1998). The co-morbidity between ADHD and ODD is highly prevalent with 30-67% of children fitting the criteria for both disorders (APA, 2000; Barkley, 1998; Brown et al., 2001; Spencer, Biederman, & Wilens, 1999).

Similarly to ADHD, ODD is a common diagnosis among school aged children and prevalence rates range from 2% to 16% (APA, 2000; Nevid et al., 2006). ODD is a childhood disorder characterised by a pattern of negativistic, hostile, and defiant behaviour (APA, 2000). Children with ODD commonly lose their temper, argue with authority, have problems complying with adult requests, disobey rules, and deliberately do things that annoy other people. Other features of ODD include: spitefulness, vindictive behaviour, and anger issues (Spencer, Biederman, & Wilens, 1999). The typical onset of the disorder is after the age of 8 years and before the age of 12 years (APA, 2000; Nevid et al., 2006). ODD commonly develops into later conduct problems and antisocial behaviour (Nevid et al., 2006).

In addition to the high overlap between ADHD and ODD, many have co-morbid conduct problems. Among those with an ADHD diagnosis, 30-67% will have co-morbid CD (Barkley, 1998; Brown et al., 2001; Spencer, Biederman, & Wilens, 1999; Weinstein et al., 2000). CD can be defined by the *DSM-IV-TR* as a pattern of behaviour that violates the basic rights of others and/or age-appropriate societal norms or rules (APA, 2000; Nevid et al., 2006). This involves destruction of property, aggression to people and animals, deceitfulness, theft, and serious violation of the rights of others (APA, 2000; Nevid et al., 2006). It is also common for individuals with CD to

engage in delinquent behaviour such as stealing, truancy, fighting, threatening others, and arson (Nevid et al., 2006).

Adolescents with ADHD and co-morbid CD or ODD have a greater rate of delinquent behaviours such as substance abuse, traffic offences, and school disciplinary consequences than those with ADHD alone (Bauermeister et al., 2007; Barkley, 1998; Findling, Arnold, Greenhill, Kratochvil, & McGough, 2007; Spencer, Biederman, & Wilens, 1999). Furthermore, individuals with ADHD and CD have been found to have an earlier age of onset of conduct-related problems, are more aggressive, and have more persistent anti-social behaviour than individuals with CD alone. Individuals with ADHD and co-morbid ODD or CD also have a higher likely hood of adverse life factors such as family psychiatric problems and family conflict (Bauermeister et al., 2007; Barkley, 1998). In summary, the co-morbid presentation of ADHD and CD and/or ODD is associated with an overall poorer prognosis than ADHD alone (Bauermeister et al., 2007; Barkley, 1998; Findling, Arnold, Greenhill, Kratochvil, & McGough, 2007; Spencer, Biederman, & Wilens, 1999).

In addition to the high co-morbidity of ADHD with externalising behavioural disorders, children with ADHD are often found to exhibit internalising symptoms (Briscoe-Smith & Hinshaw, 2006; Spencer, Biederman, & Wilens, 1999). Studies have found 15-75% of individuals with ADHD to have depressive symptoms during their child and adolescent years (Brown et al., 2001; Spencer, Biederman, & Wilens, 1999). Other research has found 12-22% of children with ADHD to have a co-morbid learning disorder (Brown et al., 2001). Furthermore, anxiety disorders are common among those with ADHD, with 25% having a co-morbid diagnosis of agoraphobia, generalised anxiety disorder, and/or simple phobia (Brown et al., 2001; Spencer, Biederman, & Wilens, 1999; Weinstein, Staffelbach, & Biaggio, 2000).

To further complicate the diagnosis of ADHD, the disorder also shares many common symptoms to Post-traumatic Stress Disorder (PTSD). ADHD and PTSD in children share the

common characteristics of difficulty concentrating, restlessness or irritability, and impulsivity (Weinstein et al., 2000). The high co-morbidity rate between ADHD and PTSD in children has become an area of significant research interest and this will be discussed in greater detail later in this review (Briscoe-Smith & Hinshaw, 2006; Coleman, 2007; Endo, Sugiyama, & Someya, 2006; Famularo, Fenton, Kinscherff, & Augustyn, 1996; Glod & Teicher, 1996; Ouyang, Fang, Mercy, Perou, & Grosse, 2008; Spencer et al., 1999; Weinstein et al., 2000; Whitlock, 2008).

Prognosis and Course of Attention Deficit/Hyperactivity Disorder

In most cases of ADHD, parents, teachers and health professionals recognise symptoms during the child's kindergarten and early school years. School adjustment and academic performance are often compromised as a result of the inattentive or hyperactive-impulsive symptoms (APA, 2000; Mash & Barkley, 2003). Many children with ADHD have co-morbid learning problems (Kieling & Rohde, 2012). Furthermore, ADHD often has a negative impact on the child's peer relationships and family functioning (Briscoe-Smith & Hinshaw, 2006; Goldstein & Ellison, 2002). Having a child with ADHD puts pressure on the family system and leads to higher rates of parental frustration, marital discord, and divorce (Whitlock, 2008). Additionally, children with ADHD are more likely to experience sexual abuse, physical abuse, and neglect (Ouyang, Fang, Mercy, Perou, & Grosse, 2008).

Approximately two thirds of children with ADHD will have inattentive, impulsive and hyperactive symptoms that persist into adolescence (APA, 2000; Carr, 1999). The impulsivity symptoms may manifest as excessive risk taking behaviour such as drug abuse, road traffic accidents, and leaving school early (Carr, 1999). During their adolescent years, more than half of those with ADHD will go onto participate in delinquent behaviour consistent with CD (Barkley, 1998; Goldstein & Ellison, 2002). ADH

While only a small minority will experience the full complement of ADHD symptoms into adulthood, up to 80% will continue to show significant symptoms into their adult years (Weiss & Hechtman, 1993). In these cases, a diagnosis of ADHD-in partial remission is used (APA, 2000). While these individuals no longer have the complete disorder, they retain symptoms that cause impairment across their academic, social, and occupational functioning (Rucklidge et al., 2006). Furthermore, one in six adolescents with ADHD will go onto develop antisocial personality disorder (APD) in adulthood (Weiss & Hechtman, 1993). APD is a personality disorder characterised by a pattern of adult behaviour including: disregard for the rights of others, a lack of empathy, and high levels of impulsivity (Mueser et al., 2011). APD is associated with an increased rate of felony, substance abuse, and serious driving accidents (Carr, 1999; Mueser et al., 2012).

Regardless of whether ADHD persists into adulthood, the presence of the disorder in childhood has been consistently shown to predict later psychiatric, psychosocial, and occupational problems (Goldstein & Ellison, 2002). For example, those with ADHD are found overall to have poorer occupational performance, higher rates of divorce, and an overall lower socioeconomic status, in comparison to matched non-ADHD individuals (Rucklidge, Brown, Crawford, & Kaplan, 2006; Weiss & Hechtman, 1993).

ADHD Summary

In summary, ADHD is the most common psychiatric disorder among school aged children and one of the most well researched. Not only is the disorder wide-spread among school children, the number of cases worldwide appear to be dramatically increasing. Despite the high prevalence of ADHD, the diagnostic criteria remain problematic. Of particular relevance to the current study, there are high rates of diagnostic co-morbidity; particularly with ODD, CD, and PTSD.

ADHD is of particular concern due to the significant negative sequelae associated with the disorder. The presence of ADHD is consistently shown to predict psychiatric, occupational, social

and academic problems later in life. Furthermore, a large proportion of children with ADHD go onto develop conduct problems and APD.

TREATMENT OF ATTENTION DEFICIT/HYPERACTIVITY DISORDER

Since the early 1990's, there has been a focus on finding evidence based treatments for ADHD (Fabiano et al., 2009). It is generally agreed in the literature that the two main evidence-based treatments for ADHD are psychosocial treatment (behaviour modification and parent training programmes) and pharmacological treatments (Brown et al., 2005; Jensen et al., 2001; Swanson et al., 2008; Van der Oord, Prins, Oosterlaan & Emmelkamp, 2008). While there is extensive research looking into the short-term efficacy of pharmacological treatments, there is a relative paucity of research investigating psychosocial options (Van der Oord, Prins, Oosterlaan & Emmelkamp, 2008). The studies looking at the short term benefits of ADHD medication constitute the largest body of treatment literature on any child-onset psychiatric disorder (Van der Oord, Prins, Oosterlaan & Emmelkamp, 2008).

Psychosocial Treatments

Psychosocial treatments include parent training, school interventions, and individual work with children (MTA Cooperative Group, 1999). The majority of psychosocial treatments involve behaviour modification, which is based on theories and principals of social learning theory, classical conditioning, operant conditioning, and cognitive-behavioural theory (Fabiano et al., 2009; MTA Cooperative Group, 1999).

Behavioural parenting training

The symptoms of inattentiveness, hyperactivity, and impulsivity in children with ADHD often lead to a strained parent-child relationship and increased parental stress (Chronis, Raggi, & Jones, 2006). As a result, many psychosocial treatments focus on behavioural principals for parents. These can be individual or group based. Behavioural Parenting Training (BPT) is based on social learning theory and teaches parents to identify and control their child's behaviour using positive reinforcement for desirable behaviour and punishment/negative reinforcement for undesirable behaviour (Fabiano et al., 2009).

A recent meta-analysis revealed that BPT is an effective intervention for children with ADHD (Fabiano et al., 2009). The review concluded that BPT is an effective treatment for a variety of disruptive behaviour problems and improves ADHD and externalizing symptoms. Furthermore, these findings were consistent for children with ADHD with and without pharmacological treatment. This finding has been consistently supported across many studies and meta-analyses which all suggest that BPT is efficacious in improving a range of child behaviour problems and ADHD symptoms (Barlow & Stewart-Brown, 2000; Piquero, Farrington, Welsh, Tremblay, & Jennings, 2009; Sereketich & Dumas, 1996; Thomas & Zimmer-Gembeck, 2007).

BPT programmes are commonly utilised in New Zealand Child and Adolescent Mental Health Services (CAMHS) and among other community care centres (Fergusson, Stanley, & Horwood, 2009). The Incredible Years Parenting Program (IYPP) and the Triple P—Positive Parenting Program (Triple P) are particularly popular in New Zealand for treating a range of child disruptive behavioural problems (Fergusson, Stanley, & Horwood, 2009).

The IYPP was developed by Carolyn Webster Stratton and is a comprehensive set of programmes designed to promote social competence and prevent the development of aggressive and conduct problems in children aged 3-10 years (Webster-Stratton, Reid, & Hammond, 2004). A recent study investigating the efficacy of the IYPP for pre-school children, found significant

improvements in parent-rated inattentiveness, hyperactivity, and impulsivity in children with ADHD symptoms, compared to a control group (Jones, Daley, Hutchings, Bywater, & Eames, 2007). These improvements were consistent at post intervention and at follow up. Further research has found the IYPP effective in improving both conduct and ADHD symptoms in boys aged 4-7 years (Hartman, Stage, &Webster-Stratton, 2002). In a similar study, the IYPP was effective in reducing anti-social behaviours in children aged 3-8 years old (Scott, Knapp, Henderson, & Maughan, 2001).

The Triple P—Positive Parenting Program (Triple P), is based on developmental and social learning theories (Sanders, 1999; Thomas & Zimmer-Gembeck, 2007). It is a program designed to promote positive parenting and caring relationships between parents and children aged 2-16 years (Sanders, 1999). Studies looking specifically at the Triple P parenting programme have also found positive results (Bor, Sanders, & Markie-Dadds, 2002; Thomas & Zimmer-Gembeck, 2007). Triple P has been associated with significant reductions in parent-reported child behaviour problems, lower levels of dysfunctional parenting, and greater parental competence in comparison to a control group of parents (Bor, Sanders, & Markie-Dadds, 2002). Furthermore, parents that attended Triple P reported a reduction in disruptive behaviour, inattentiveness, and hyperactivity at one year follow up (Bor, Sanders, & Markie-Dadds, 2002).

Summary

In summary, research has consistently demonstrated that BPT is an effective intervention for the treatment of ADHD symptoms and a range of externalizing behaviour problems. Furthermore, BPT is found to be effective with and without the use of pharmacological treatments. BPT programmes such as the IYPP and Triple-P are commonly used in New Zealand and have been associated with a reduction in a range of behaviour problems and ADHD symptoms.

School management strategies

Children with ADHD often have significant difficulty functioning in a classroom. This can have a negative impact on their academic performance, classroom behaviour, and peer relationships (Chronis, Raggi, & Jones, 2006). As a result, classroom interventions, school management strategies, and academic approaches are commonly used to help children with ADHD (Chronis, Raggi, & Jones, 2006; Goldman et al., 1998).

Classroom behavioural interventions generally involve consultation with classroom teachers and the implementation of individualized contingency management programs. Specific classroom techniques include: specific seating of the child in the classroom, praise, planned ignoring, effective commands, daily report cards, and time out (Chronis, Raggi, & Jones, 2006; Goldman et al., 1998). Research indicates that behavioural classroom interventions are an effective means of improving behaviour and ADHD related symptoms in the school setting (Chronis, Raggi, & Jones, 2006). In addition to behavioural approaches, academic interventions are commonly used to help with academic functioning, e.g. task and instructional modifications, peer tutoring, computer-aided instructions and programmes, and strategy training (Chronis, Raggi, & Jones, 2006). Reviews of research around academic interventions for children with ADHD suggest that they help to improve academic performance (DuPaul & Eckert, 1998). In summary, classroom behaviour interventions and academic interventions are associated with an overall improvement in ADHD-related symptoms and academic performance in the school setting.

Social skills training

Children with ADHD very commonly experience interpersonal difficulties and issues in peer relationships. This is associated with a longstanding adverse impact on their social development and functioning (Chronis, Raggi, & Jones, 2006; De Boo & Prins, 2007; Paek et al.,

2009). Therefore, social skills training is commonly utilized as an intervention to help develop and reinforce the use of appropriate social skills (Chronis, Raggi, & Jones, 2006).

Research into the efficacy of social skills training is mixed, with reviews suggesting that a combination of social skills training and parent interventions results in more robust effects than social skills training alone (Chronis, Raggi, & Jones, 2006). Other review studies have found positive results and concluded that social skills training stands as an effective intervention in helping ADHD children function socially (De Boo & Prins, 2007). This is supported by a recent study demonstrating that short term social skills training for children with ADHD leads to an improvement in child self esteem, teacher reported social skills, and parent-reports of inattentiveness (Paek et al., 2009). However, this particular study emphasized the need for well established social skills programmes catered specifically for ADHD that target specific ADHD subtypes and common co-morbid problems (Paek et al., 2009).

In summary, it seems that social skills' training is helpful in targeting ADHD-related problems such as poor social skills and low esteem. However, it seems the intervention alone is not an all encompassing treatment for core ADHD symptoms.

Cognitive training

Studies have found that individuals with ADHD commonly have problems in their executive functioning ability (Beck, Hanson, Puffenberger, Benninger & Benninger, 2010). In particular, children with ADHD have difficulty with working memory ability (Beck et al., 2010). Therefore, treatments that target executive functioning ability and working memory (e.g. targeting attention, working memory, and inhibition) are commonly used for children with ADHD.

Recent research suggests that working memory training (WMT) is an effective intervention in improving executive functioning and ADHD symptoms (Beck et al., 2010). However, research looking into WMT has produced inconsistent results. A recent review suggests that cognitive

training treatments for ADHD are not effective alone as interventions for ADHD (Markomichali, Donnelly & Sonuga-Barke, 2009). There is a need for further research around the use of working memory training for children with ADHD and how this could be integrated into a holistic treatment plan.

Pharmacological Treatments

Over the last 20 years the use of pharmacological interventions to treat ADHD has increased dramatically (Faraone et al., 2006; Mayes, Bagwell, Erkulwater, 2008; Mitchell & Read, 2011; Olfson et al., 2003; Zuvekas, Vitiello, & Norquist, 2006). Over the last two decades it is estimated that the money spent on ADHD medications globally has increased 9-fold. For example, in 2003 an estimated US $2.4 billion was spent globally on ADHD medication, which is nine times that which was spent in 1993 (Scheffler, Hinshaw, Modrek, & Levine, 2007). Furthermore, in the United Kingdom (UK), prescriptions of ADHD medication grew from 6000 per year in 1994, to 550 000 in 2006 (Timimi, 2009). It is estimated globally, that at least 85% of children diagnosed with ADHD are medicated with stimulants (Olfson et al., 2003). Research in the US has demonstrated that males in the 6-12 year age bracket of European ethnicity have the highest use of stimulant medication (Zuvekas, Vitiello, & Norquist, 2006).

The amount of research into the use of stimulant medication in treating ADHD is perhaps overwhelming (Chronis, Jones, & Raggi, 2006). The studies looking at the short term benefits of ADHD medication constitute the largest body of treatment literature on any child onset psychiatric disorder (Van der Oord et al., 2008).

Over the last two decades, the stimulant medications methylphenidate, dextroamphetamine, and mixed amphetamine salts have been the most common in the treatment of ADHD (Faraone et al., 2006; Mitchell & Read, 2011). Stimulant medications work in the child's brain to increase the

availability of synaptic dopamine, which helps reduce over-activity, impulsivity, and inattentiveness (Faraone et al., 2006).

Meta-analyses have demonstrated robust effects of stimulant medication and other ADHD-medications in reducing the core symptoms of ADHD (e.g. inattentiveness, hyperactivity and impulsivity) (Chronis, Jones, & Raggi, 2006; Faraone & Buitelaar, 2010; Goldman et al., 1998; Van der Oord, Prins, Oosterlaan & Emmelkamp, 2008). A review study by Farone et al., (2006) revealed that all types of ADHD medication were effective in improving core ADHD symptoms. While both stimulant and non-stimulant medications were found to be effective, stimulant medications were found to be significantly more effective in improving ADHD symptoms (Faraone et al., 2006). A further meta-analysis investigating different types of stimulant medication, found that both methylphenidate and amphetamine products were significantly effective in reducing ADHD symptoms. However, amphetamine products had a significantly greater effect than methylphenidate products (Faraone & Buitelaar, 2010).

Further research suggests that stimulant medication helps improve classroom behaviour, academic performance, oppositional and aggressive behaviour, peer interactions, and parent-child relationships (Chronis, Jones, & Raggi, 2006; Goldman et al., 1998). However, other studies have failed to find significant improvements in academic functioning and social behaviour (Connors, 2002; Rickel & Brown, 2007; Van der Oord et al., 2008). It seems that the most robust effects of stimulant medication have been shown for core ADHD symptoms, where as improvements in academic achievement and social behaviour have been less pronounced (Brown et al., 2005; Chronis, Jones, & Raggi, 2006; Van der Oord et al., 2008).

Although stimulants are the most common form of pharmacological treatment for ADHD, there are several non-stimulant medications that have proven to be effective in the short-term. These include tricyclic antidepressants, bupropion, modafinil, monoamine oxidase inhibitors, and atomoxetine (Faraone et al., 2006). Atomoxetine is a relatively new pharmacological treatment for

ADHD and was approved in the USA in 2002 (Cheng, Chen, Ko, & Ng, 2007). Atomoxetine is a non-stimulant medication and works as a selective noradrenergic reuptake inhibitor (Kratochvil et al., 2008).

In recent reviews, atomoxetine was associated with improved parent ratings of core ADHD symptoms and oppositional behaviour. These effects were also demonstrated at two year follow up (Cheng, Chen, Ko, & Ng, 2007; Kratochvil et al., 2006). Further research has compared the efficacy of atomoxetine in young (6-7 years) and old (8-12 years) children (Kratochvil et al., 2008). Atomoxetine significantly reduced core ADHD symptoms in both age groups, however, this effect was greater in the older age group. A further study compared atomoxetine to the stimulant medication methylphenidate. At six weeks of treatment there were no significant differences between the two groups, suggesting that atomoxetine is comparable in efficacy to stimulant medication (Hazel et al., 2010).

Limitations and side-effects of pharmacological treatments

Despite the considerable body of research supporting the efficacy of pharmacological treatments for ADHD, there are significant limitations. Many studies rely on parent-rating as an outcome measure which can by subjective and impacted on by treatment group bias (Kratochvil et al., 2006). Review studies investigating the efficacy of ADHD medication have challenged the methodology used in these studies. Some common problems identified were: poor quality trials, frequent and problematic medication side effects, inconsistent short-term effects, inconsistent use of rating scales to measure 'effectiveness', and a lack of research looking into long-term efficacy beyond four weeks of treatment (Schachter, Pham, King, Langford, & Moher, 2001).

Many argue that the long-term effects of most pharmacological treatments for ADHD have not been well researched (Goldman et al., 1998; Kratochvil et al., 2008). This questions the long term efficacy and safety of pharmacological treatments for ADHD. Furthermore, medications such

as atomoxetine have not been well researched in young children (under 8 years old), and the safety

of using these treatments in young children is questionable (Kratochvil et al., 2008). The safety of

ADHD medication has been further questioned in animal studies. Research examining the use of

methylphenidate in rats has found that the drug leads to long-term changes in the development of

the central dopaminergic system (Moll, Hause, Ruther, Rothenberger, & Huether, 2001; Sproson,

Chantrey, Hollis, Marsden, & Fonel, 2001). This suggests that some of the structural neurological

changes found in children with ADHD may relate to the use of stimulant medication, not the

disorder itself (Timimi et al., 2004).

Another major limitation of pharmacological treatments is the side-effects. The most

common side effects of stimulant medications are sleep difficulties, decreased appetite, stomach

aces, headaches, and jitteriness (Goldman et al., 1998; Van der Oord et al., 2008). The suppression

of appetite can lead to delays in weight and body mass index development, and suppressed height

gain (Van der Oord et al., 2008). A small group of children experience motor tics, cognitive

impairment, and psychotic symptoms (Goldman et al., 1998). Kratochvil et al., (2008) found that

younger children (under 8 years) taking atomoxetine were particularly susceptible to abdominal

pain, decreased appetite, vomiting, and somnolence. Other atomoxetine side effects include:

appetite suppression, abdominal pain, vomiting, sleep difficulties, and dyspepsia (Cheng et al.,

2007). While many studies have found adverse side-effects to ADHD medication, other researchers

argue that side effects are generally short-lived, generally mild, and responsive to dosage

requirements (Goldman et al., 1998). There is some research to support that ADHD medication can

act as a 'gateway' drug to later drug abuse/dependence problems (Goldman et al., 1998). Many

researchers recommend that children taking ADHD medication receive ongoing monitoring due to

these negative side-effects (Cheng et al., 2007; Kratochvil et al., 2008). Furthermore, it has been

argued that the commonly used stimulant medication methylphenidate is clinically

indistinguishable from other amphetamine substances such as methamphetamine and

dextroamphetamine (Baldwin & Cooper, 2000). Both of these drugs are renowned for their extreme abuse potential, addictive properties, and the risk to physical health (Baldwin & Cooper, 2000; Radcliffe & Timimi, 2003).

When considering the long-term use of medication to treat a 'mental health problem' such as ADHD it is also important to consider the psychological side-effects. The long-term use of ADHD medication can script a "potentially life-long story of disability and deficit that physically healthy children may end up believing" (Timimi et al., 2004, p. 61). Children could interpret ongoing medication treatment as 'punishment' for seemingly bad behaviour. The idea that children are taking medication for a mental health disorder could potentially lead to feelings of powerlessness in that the child is unable to control their own behaviour (Timimi et al., 2004).

In summary, it is important to consider the many methodological issues when interpreting the efficacy of the use of ADHD medication. Furthermore, it is clear that there are valid concerns around the long-term efficacy and safety of ADHD medications.

Dietary manipulations

Research into the efficacy of dietary manipulations in treating symptoms of ADHD is mixed, with many refuting the efficacy of diet changes in treating ADHD (Goldman et al., 1998)

Research has found children with ADHD to exhibit significant differences in their omega-3 fatty acid composition (Bloch & Qawasmi, 2011; Sin & Bryan, 2007). Therefore, there has been a recent interest in the effect of omega-3 fatty acid supplementation (O-3FAS) in children with ADHD. Many argue that supplementation could be valuable in the treatment of ADHD (Bloch & Qawasmi, 2011; Chalon, 2009). A recent review of the research found that O-3FAS was associated with small, but significant effects in improving core ADHD symptoms (Chalon, 2009. The study suggested that O-3FAS can be used to enhance pharmacological interventions or used alone for families that are adverse to other medication options. Other research has found that O-3FAS was

associated with a significant improvement in parent rated ADHD symptoms (Sin & Bryan, 2007). However, no effects were found in teacher ratings of ADHD symptoms (Sin & Bryan, 2007). The majority of researchers suggest that the efficacy of omega-3 fatty acid supplementation is promising, however, further investigation is necessary (Bloch & Qawasmi, 2011; Chalon, 2009; Sin & Bryan, 2007).

The 'few foods' approach is an elimination diet where children are restricted to eating a limited number of foods. A recent study investigating the few foods approach suggested that a 5-week few foods diet in young children with ADHD leads to significant changes in behaviour as rated by parents and teachers (Pelsser, Frankena, & Toorman, 2011). Similar results were found in an earlier study by the same authors (Pelsser, Frankena, Toorman, & Savelkoul, 2009). Despite promising results, the methodology and interpretation of these studies has been criticised. Adesman (2011) suggests that the results were subject to treatment group bias because the families and teachers were not blind to treatment status. It seems there is a need for further investigation around the effect of diet restriction in treating ADHD.

Multi-modal Treatment

There is an extensive body of research indicating that multi-modal treatment is perhaps the most effective for children with ADHD (Brown et al., 2005; Goldman et al., 1998; Jensen et al., 2001; Swanson et al., 2008; Van der Oord, Prins, Oosterlaan & Emmelkamp, 2008). Multi-modal treatment is the integration of pharmacological treatments, along with a number of environmental, educational, psychotherapeutic, and school based approaches (Goldman et al., 1998). Some suggest that multi-modal treatment is superior to psychosocial or pharmacological treatments alone (Goldman et al., 1998). While other clinicians and researchers argue that multimodal treatment is only necessary for those children with ADHD and co-morbid problems (Goldman et al., 1998; MTA Cooperative Group, 1999).

The Multimodal Treatment Study of Children with ADHD (MTA study) is one of the largest ADHD treatment studies carried out to date and is very commonly cited among the ADHD treatment literature (MTA Cooperative Group, 1999). In the MTA study, 579 children (aged 7-9 years) with a diagnosis of ADHD-combined type were randomly assigned to either a routine community care group or one of three 14-month intensive treatment groups: medication, behaviour treatments, or the combination of both (Jensen et al., 2001; MTA Cooperative Group, 1999; Swanson et al., 2008).

At 14 months (post treatment) the study found that children in all four groups had substantial reductions in ADHD symptoms (Jensen et al., 2001). However, the combined medication and behavioural treatment group produced superior results in comparison to the behaviour and medication groups alone (Jensen et al., 2001; Swanson et al., 2008). The combined treatment produced better outcomes on several measures including: oppositional/aggressive symptoms, internalizing symptoms, teacher rated social skills, parent-child relations, and reading achievement (Swanson et al., 2008). The medication alone group was superior to the behaviour therapy alone and community groups (Swanson et al., 2008). Despite these initial improvements, the differences between each treatment group were no longer apparent at 3 years post-treatment (Jensen et al., 2007). This suggested that type of treatment group did not predict the child's functioning 3 years post-treatment. This finding was maintained at 6 and 8 year follow up where once again, the type of treatment group did not predict functioning (Molina et al., 2009). At 8 years post-treatment there was a significant improved in core ADHD symptoms in comparison to baseline (pre-treatment). However as a whole, the ADHD participants in the study were functioning significantly lower than their aged-matched ADHD peers on a variety of outcome measures (Molina et al., 2009). Those participants continuing to take ADHD medication at 6-8 years follow up did not show greater improvement than their non medicated participants (Molina et al., 2009).

The MTA study is the first to confirm that a short-term ADHD treatment period in childhood does not have lasting effects into the adolescent years. Even though there were overall improvements in all of the treatment groups, the treatment did not normalize those children with ADHD at 6 and 8 year follow up. The study did highlight however, that in terms of short-term treatment, a multi-modal approach combining pharmacological treatment and psychosocial approaches has the greatest efficacy in treating children with ADHD.

Further research looking into the MTA study revealed that the child's initial clinical presentation (i.e. severity of ADHD symptoms, conduct problems, intellect ability, social advantage, and co-morbid problems) was a significant predictor of later adolescent functioning. This suggests that psycho-social factors and early ADHD symptom trajectory is a more accurate predictor of later functioning than type of treatment received in childhood (Molina et al., 2009). This also implies that those children with ADHD, who have a greater social, demographic, academic, and behavioural advantage, will most likely have a greater response to treatment. Furthermore, these children will be likely to have an overall better long-term prognosis (Molina et al., 2009).

There has been some critique into the methodology and interpretation of the MTA study. Some authors suggest that the comparisons made between the groups were not equal due to many children continuing pharmacological treatments beyond the 14 month treatment period. Thus the potency of the treatment groups may not have been equal (Arnold et al., 2004). Others emphasize the importance of including younger children in such studies as there is a paucity of research regarding treatment of this younger age group (Kratovich et al., 2006).

Further research looking into multimodal ADHD treatment has produced similar results (Brown et al., 2005). In a meta-analysis, Brown et al. (2005) revealed that a combination of both behaviour therapy and medication is thought to be the most effective in treating ADHD symptoms. Furthermore, the inclusion of behavioural therapy in a pharmacological based treatment is found to

improve functioning in other areas such as academic, social, and family functioning, and reduce the overall required dosage of medication (Brown et al., 2005).

Treatment Summary

In summary, it is generally agreed upon in the literature that the most effective treatment for ADHD is multi-modal approach including pharmacological treatment and behavioural approaches. While research consistently supports the short term efficacy of multi-modal treatment, the long-term efficacy is less pronounced. Furthermore, the long-term efficacy and safety of pharmacological interventions for ADHD are largely unknown. Studies have found that early ADHD symptom trajectory and social advantage are more accurate predictors of later functioning than the type of ADHD treatment received in childhood.

In terms of behavioural interventions, review studies consistently support the use of BPT as an efficacious treatment in improving a range of child behaviour problems and ADHD symptoms. Furthermore, behavioural classroom interventions, school management strategies, and academic interventions are an effective method for improving behaviour and academic functioning in the school setting.

There is an extensive body of research that supports the efficacy of a range of medication options in treating core ADHD symptoms. However, limitations of pharmacological treatments include: adverse side effects, a paucity of research investigating long-term efficacy and safety of ADHD medication, and a lack of research that supports an improvement in social and academic functioning.

AETIOLOGY OF ATTENTION DEFICIT/HYPERACTIVITY DISORDER

The aetiology of ADHD remains a topic of controversy (Barkley, 1997; Goldstein & Ellison, 2002; Nigg, 2006). The research regarding this topic is extensive and the proposed aetiologies are numerous (Mash & Barkley, 2003). Many suggest that ADHD is caused by a complex interaction of neurological, genetic, and environmental factors (Steinhausen, 2009). Further complicating this research is the fact that individuals with ADHD are a highly heterogeneous group who show great variance in not only their types of symptoms, but in the perseverance and severity of their ADHD (Connor, 2002; Nigg, 2006). This creates a complex aetiological picture and supports the idea that there are perhaps multiple factors that interact in producing the disorder, or multiple pathways that lead to various manifestations of the disorder (Nigg, 2006; Steinhausen, 2009). Therefore, a multiple pathways approach will be considered based on the hypothesis that many of the aetiological pathways in ADHD are not mutually exclusive. These aetiological factors can be grouped into genetic, neurological and environmental factors. It is beyond the constraints of this literature review to discuss all of the proposed aetiologies of ADHD in detail, thus only the perspectives with the most empirical support will be reviewed.

Biological and Genetic Factors

It is widely recognised that ADHD is more common among family members; leading many to believe there may be a genetic contribution (Bennett, 2006; Nevid et al., 2006; Nigg, 2006; Steinhausen, 2009; Whitlock, 2008). Research looking into the link between ADHD and genetics comes from a variety of sources including family, twin, adoption, genome, and candidate gene search studies (Brock, Jimerson, & Hansen, 2009).

Twin and family studies

Twin, adoption, and family studies have found evidence to support a familial link (Bennett, 2006; Carr, 1999; Hinshaw, 1994; Mash & Barkley, 2003; Steinhausen, 2009). Monozygotic twin studies have found concordance rates ranging from 51% to 90% (Bennett, 2006; Mash & Barkley, 2003; Nigg, 2006; Whitlock, 2008). Furthermore, prevalence studies have found that concordance of ADHD between siblings is approximately 30% (APA, 2000; Nigg, 2006). Adoption studies have found that biological parents of children with a diagnosis of ADHD are more likely to have a diagnosis of ADHD than adopted parents (Sprich, Bierderman, Crawford, Mundy, & Stephen, 2000). In summary, there is evidence to suggest that ADHD is more common among family members, suggesting there may be a genetic vulnerability to developing the disorder. However, this will be examined in closer detail in the limitations section.

Molecular Genetics

While family studies suggest that ADHD may be heritable, research investigating the molecular genetics of ADHD has provided mixed findings (Mick et al., 2010; Steinhausen, 2009; Williams et al., 2010). There has been an increase in research investigating various candidate genes involved in the development of ADHD (Mick et al., 2010; Neal et al., 2010; Steinhausen, 2009). Genes involved in the dopamine neurotransmitter system pathway have received particular interest. This is due to their involvement in the prefrontal regions of the brain that are associated with executive functioning, attentional, motivational, and exploratory behavioural processes (Carr, 1999; Laucht et al., 2007). There have been several genes implicated in the aetiology of ADHD and studies looking for risk alleles have identified the following genes: the dopamine D4 gene (DRD4), dopamine D5 receptor gene (DRD5), the dopamine transporter gene (DAT1), the serotonin receptor gene (HTR1B), and the synaptosomal-associated protein 25 gene (SNAP-25) (Brock, Jimerson, & Hansen, 2009; Carr, 1999; Howe, 2010; Mick & Faraone, 2008; Steinhausen, 2009). However,

these gene associations are small and this has led to the hypothesis that there are perhaps many genes that contribute to the overall genetic vulnerability to ADHD (Brock, Jimerson, & Hansen, 2009; Mick & Faraone, 2008; Stenhausen, 2009). One study found a specific genetic mechanism associated with a particular group of ADHD children that have co-morbid disruptive behaviour problems (e.g. CD and ODD) (Zhang, Wang, Wang, & Yang et al., 2005). The results from this study suggest that there may be different types of ADHD (with and without co-morbid CD and ODD) that have varying genetic mechanisms involved in the aetiology of the disorder.

More recent research has employed genome-wide linkage scans in an attempt to find chromosomal regions that may contain susceptibility genes for ADHD (Steinhausen, 2009; William et al., 2010). In a well publicized study, William et al. (2010) carried out a genome-wide analysis looking at the rate of chromosomal deletions and duplications (know as copy number variants - CNV's) in a sample of ADHD children and controls. The study found that 15.6% of the children with ADHD had rare CNV's, in comparison to 7.4% of the control group. This finding led to authors to conclude that "our findings provide genetic evidence of an increased rate of CNV's in individuals with ADHD and suggest that ADHD is not a purely social construct" (Williams et al., 2010, p1401).

However, this finding has been disputed in further research. A recent review looking into the results of all genome-wide association studies of ADHD to date, found no statistically significant genome-wide associations (Neal et al., 2010). There were some candidate genes with small effects found to be involved with the disorder (Neal et al., 2010). The authors concluded that "given that ADHD is a highly heritable disorder, our negative results suggest that the effects of common ADHD risk variants must, individually, be very small or that other types of variants, e.g., rare ones, account for much of the disorder's heritability" (Neal et al., 2010, p.884). The findings from this review were supported in further research carried around the same time (Mick et al., 2010). This study found little support for any statistically significant genome-wide findings and

very limited support for additional candidate genes for ADHD. The authors attributed the lack of significant findings to having an insufficient sample size and perhaps the heterogeneity among individuals with ADHD in the sample. These findings suggest that although there have been some significant findings for the association between candidate genes and ADHD, in terms of genome-wide associations the findings are far from conclusive (Faraone et al., 2005; Mick et al., 2010).

Summary and limitations of biological and genetic factors

In summary, while twin, family, and adoption studies suggest that ADHD may be heritable, findings from molecular genetic studies suggest that the genetic architecture of ADHD is complex (Neal et al., 2010; Mick & Faraone, 2008; Mick et al., 2010). The genome-wide analyses carried out thus far have found inconsistent results, and are therefore inconclusive (Mick & Faraone, 2008; Mick et al., 2010; Neal et al., 2010). Candidate genes research has provided evidence to support that there a number of genes implicated in the aetiology of ADHD. However, these associations have been small, and it is now clear that there are many genes that contribute with small effect to the overall genetic vulnerability to ADHD (Brock, Jimerson, & Hansen, 2009; Mick & Faraone, 2008; Stenhausen, 2009; Mick et al., 2010).

There are a number of limitations that are important to consider when interpreting research into the biological aetiology of ADHD (Steinhausen, 2009). The finding that ADHD may run in families is not necessarily evidence alone that ADHD is inherited genetically (Mitchell & Read, 2011; Read and Sanders, 2010). It is well known that the brain is responsive to, and interacts accordingly, with its physical and social environment. This suggests that even a strong genetic association to ADHD may be moderated through environmental factors (Brock, Jimerson, & Hansen, 2009; Steinhausen, 2009). This idea is supported in research which found that family adversity moderates the impact of the dopamine transporter gene (DAT1) on the development of ADHD symptoms (Laucht et al., 2007). The study found that individuals with the (DAT1) gene

only developed symptoms of ADHD if exposed to psychosocial adversity, in comparison to those living in less adverse family conditions. This idea is further supported in recent research which identified that parents with diagnoses of ADHD are more likely to exhibit adverse parenting styles and have insecure attachments with their child (Edel, Juckel, & Brune, 2010). This suggests that to a certain extent, genes associated with the development of ADHD symptoms may cause a susceptibility to high risk environments and it is probably a combination of genetic and adverse psychosocial factors that cause ADHD, not biological factors alone (Edel, Juckel, & Brune, 2010; Laucht et al., 2007; Steinhausen, 2009).

It is clear that there is a long way to go in terms of research before we can conclude that ADHD is in fact inherited or caused by biological factors. While there is some evidence to suggest that some individuals may have a small genetic vulnerability to developing ADHD, it seems it is a combination of both biological and psycho-social factors that interact in producing the symptoms of ADHD.

Neurological Factors

The neurological deficit hypothesis suggests that ADHD results from structural and/or functional brain abnormalities (Barkley, 1998; Carr, 1999). These neurological abnormalities are thought to stem from a combination of genetic and environmental factors (Brock, Jimerson, & Hansen, 2009; Carr, 1999; Millichap, 2008).

Overall brain size

Magnetic resonance imaging (MRI) studies have shown that on average, children with ADHD have smaller brains (cerebrum and cerebellum) in comparison to healthy controls (Castellanos et al., 2002; Keiling, Renata, Tannock, & Fransisco, 2008; Krain & Castellanos, 2006; Millichap, 2008; Sowell et al., 2003; Steinhausen, 2009). These differences in brain size are found

to be consistent throughout childhood and adolescence (Brock, Jimerson, & Hansen, 2009; Keiling et al., 2008). Studies have found that ADHD children with and without medication maintained this overall reduction in brain size (Castellanos et al., 2002). This suggests that decreased brain volumes were not associated with pharmacological treatments. It is unknown whether these differences in brain size persist into adulthood (Brock, Jimerson, & Hansen, 2009). From the research aviable, it is impossible to disentangle whether the overall reduction in brain size in individuals with ADHD is a result of biological or envionrmental factors, or both.

Brain regions and pathways involved in ADHD

Even though differences in brain size have been established, research looking into specific brain regions involved in the aetiology of ADHD has produced conflicting results (Keiling et al., 2008). While many propose that the frontal-striatal cerebella circuits play a central role in the aetiology of ADHD, some suggest that the disorder arises from a more distributed circuitry (Brock, Jimerson, & Hansen, 2009; Keiling et al., 2008). Despite some conflicting results, it seems the most significant findings from neuro-imaging studies of ADHD have identified the following areas to be involved in the aetiology of ADHD: the prefrontal cortex, basal ganglia (striatal connections), and cerebellum (Brock, Jimerson, & Hansen, 2009; Keiling et al., 2008; Krain & Castellanos, 2006; Millichap, 2008; Steinhausen, 2009). These brain regions will be looked at individually:

Prefrontal cortex

Research examining specific regions of the brain relating to ADHD largely stems from neuropsychological findings (Keiling et al., 2008). Children with ADHD are frequently found to have neuropsychological deficits in executive functioning, response inhibition, delay aversion, and tasks that require sustained attention (Barkley, 2006; Brock, Jimerson, & Hansen, 2009; Kieling et al., 2008). Research suggests that many of the behavioural signs of ADHD are related to these

underlying neuropsychological deficits, which in turn, have been linked to functional and structural abnormalities in the prefrontal cortex and its networks within the striatum and cerebellum (Barkely, 1997; Kieling et al., 2008; Krain & Castellanos, 2006; Sowell et al., 2003). A review by Seidman, Valera, & Makris (2005) found that all studies that measured at least one region of the prefrontal cortex found smaller sizes in ADHD individuals. This is supported in further research that has found children with ADHD to have smaller and less symmetrical prefrontal and basal ganglia structures in the right cerebral hemisphere (Millichap, 2008). Other studies have found that children with ADHD have relative cortical thinning in regions of the prefrontal cortex that are important for attentional control (Shaw et al., 2006). Some children in the study with ADHD had "fixed" thinning in the prefrontal cortex which was associated with an overall worse clinical outcome in terms of their ADHD symptoms.

Further supporting the role of the prefrontal cortex is research employing functional MRI's. These studies have found that activity in the prefrontal area of the brain during response-inhibition is reduced significantly in individuals with ADHD (Rubia, Overmeyer, & Taylor et al., 1999; Rubia, Smith, & Brammer et al., 2005). A meta-analysis examining 16 functional MRI studies, found that all of studies consistently found reduced activity in regions of the prefrontal cortex (Dickstein, Bannon, Castellanos, & Milham, 2006).

Basal ganglia

There is a growing body of evidence to support the role of the basal ganglia (striatum) in the aetiology of ADHD (Seidman, Valera, & Makris, 2005; Steinhausen, 2009). More specifically, it is the caudate nucleus, the putamen, and pallidum, which are the entry point to the basal ganglia that have been associated with ADHD (Krain & Castellanos, 2006). These brain structures play a role in the connection between the cerebrum and the cerebellum and relay information related to motor planning, sequencing, learning and execution (Brock, Jimerson, & Hansen, 2009; Keiling et al.,

2008). In a meta-analysis, 9 out of 13 studies found significantly smaller basal ganglia structures in individuals with ADHD, on either the left or right hemisphere (Seidman, Valera, & Makris, 2005). Further research compared monozygotic twins (one with and one without ADHD) and found that the ADHD twin had a significantly smaller caudate volume (Castellanos, Sharp, Gottesman et al., 2003).

Cerebellum

The cerebellum has connections to the frontal regions of the brain and plays a role in attention shifting (Brock, Jimerson, & Hansen, 2009). Although not as well researched as the prefrontal cortex and basal ganglia, there is some evidence to suggest that the cerebellum plays a role in the development of ADHD symptoms. In particular, an area of the cerebellum called the vermis has been found in numerous studies to be smaller or contain structural abnormalities in individuals with ADHD (Keiling et al., 2008; Seirdman et al., 2005).

Neurochemical dysfunction

In addition to research based on functional and structural MRI's, some research has looked into neurotransmitter functioning in the prefrontal cortex (Barkley, 2006; Carr, 1999). There is evidence to suggest that individuals with ADHD have deregulated dopamine and noradrenalin systems in the prefrontal cortex and basal ganglia (Barkely, 1997; Barkley, 2003). This neurochemical system is related to executive functioning (Brock, Jimerson, & Hansen, 2009). This is supported by the responses of ADHD children taking stimulant medication which acts on the dopamine system and serves to reduce inattentiveness and hyperactivity (Brock, Jimerson, & Hansen, 2009; Carr, 1999). Stimulant medication acts to increase the availability of dopamine (Brock, Jimerson, & Hansen, 2009). In support of this research, earlier studies have demonstrated

that children with ADHD have reduced levels of dopamine in their cerebral spinal fluid compared to healthy controls (Barkley, 2006).

Summary and limitations of neurological findings

In summary, there is evidence to suggest that children with ADHD have, on average, an overall smaller brain size compared to non-ADHD children. Furthermore, particular areas of the brain including the prefrontal cortex, the basal ganglia and the cerebellum have been implicated in the development of ADHD symptoms. More specifically, there is evidence to suggest that individuals with ADHD have deregulated dopamine and noradrenalin systems in the prefrontal cortex and basal ganglia regions.

It is important to consider that differences in brain structure and neurochemistry in children with ADHD do not necessarily suggest a biological aetiology. It is well recognised that the brain is highly responsive to, and can be altered by, the environment (Read & Sanders, 2010). Therefore, the differences in brain structure and function found in children in ADHD may reflect responsiveness to environmental factors such as stress, disrupted attachment relationships and trauma (Perry, 2002). This is particularly relevant when considering that the brain is highly plastic to the first 5 years of life and very susceptible to the impacts of chronic stress (Perry, 2002).

When interpreting these neurological findings there are important methodological limitations to consider. Some of these limitations include: the use of small sample sizes, a lack of consistency in MRI methodology, and inconsistency in the inclusion criteria for what is considered 'ADHD' (Krian & Castellanos, 2006; Timimi et al., 2004). This suggests that the children with ADHD included in the studies were a heterogeneous group with varying levels of ADHD symptom severity.

Another common methodological issue is that many of the children used in these studies were receiving ongoing pharmacological interventions for ADHD. Therefore, it is difficult to

establish whether the structural and functional differences in the brains of the ADHD children were related to medication side-effects or the disorder itself (Timimi et al., 2004). Furthermore, many of the MRI studies have been critiqued for publishing very small effect sizes. There is an apparent need for further research that takes into account these methodological limitations before any causal connections can be made between ADHD and structural and/or functional brain abnormalities.

<div align="center">

Environmental Risk Factors

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Prenatal risk factors

There is extensive research to suggest that pre-natal maternal factors such as smoking, alcohol, use, cocaine exposure, maternal exanthema and anaemia are risk factors for developing ADHD (Das Banerjee et al., 2007; Carr, 1999; Millichap, 2008). Pregnancy and birth related factors have received particular interest in research because they are thought to play a role in some of the structural and functional brain abnormalities found in children with ADHD (Barkley, 2006).

Maternal tobacco use

The research into the relationship between maternal smoking and ADHD has received particular research attention. This theory is based on the idea that prenatal exposure to nicotine may disrupt normal brain development that could lead to problems with attention and behaviour (Millichap, 2008). Some argue that there is an association between maternal smoking and inattention problems in offspring (Millichap, 2008; Motlagh et al., 2010). This is supported in a study that found heavy smoking during pregnancy (more than 10 cigarettes per day) was associated with problems in attentional control in offspring (Motlagh et al., 2010). However, other research has produced contradictory findings. A recent study examined the clinical profile of children with ADHD with and without prenatal exposure to maternal smoking. The study revealed that there were

no significant differences in symptom profiles between the ADHD children with and without prenatal smoking exposure (Biederman et al., 2011). In an earlier review study, Linnet et al. (2003) found that maternal lifestyle factors such as smoking during pregnancy, contributed to behaviour problems in offspring. The review found that the majority of studies showed an association between prenatal smoking exposure and problems with inattention and hyperactivity. However, not all of the studies reviewed found a statistically significant association.

It is clear there is a need for further research into the effects of exposure to pre-natal maternal smoking. Research suggests that there may be a small relationship between maternal smoking and ADHD, but this is by no means conclusive or causal (Linnet et al., 2003).

Maternal alcohol consumption

There is some research to suggest that maternal consumption of alcohol plays a role in the development of ADHD (Das Banerjee et al., 2007; Mick, Biederman, Faraone, Sayer, & Kleinmann, 2002). The most common and serious consequence of maternal alcohol consumption is fetal alcohol syndrome (FAS) which is thought to share many of the behavioural features of ADHD. Research supports that children with ADHD have an increased likelihood of prenatal alcohol exposure (Mick et al., 2007). Prenatal alcohol exposure is known to induce structural brain abnormalities, particularly in the cerebellum. The cerebellum has been linked to the aetiology of ADHD (Das Banerjee et al., 2007; Keiling et al., 2008; Seirdman et al., 2005). However, the research into the relationship between ADHD and maternal alcohol consumption has produced mixed findings (Millichap, 2008). In a meta-analysis, only half the studies reviewed found a significant association between symptoms of ADHD and maternal alcohol consumption (Linnet et al., 2003). Linnet et al., (2003) also noted methodological limitations in the research such as: studies only including mothers who consumed very high levels of alcohol and a lack of control for confounding factors such as social, dietary, genetic and family factors.

Birth related factors

There are a variety of birth related factors that have been associated with the development of ADHD (Brock, Jimerson, & Hansen, 2009; Das Banerjee et al., 2007). These include delivery complications such as fetal distress, fetal post-maturity, forceps delivery, toxemia or eclampsia, small head circumference, and low birth weight (Barkley, 2006; Brock, Jimerson, & Hansen, 2009; Barkely, 2003; Carr 1999; Das Banerjee et al., 2007; Millichap, 2007). One study compared monozygotic twins with and without ADHD and found that the ADHD twin was more likely to have a smaller birth size and a breech delivery than the non-ADHD twin (Sharp et al., 2003). The majority of these birth related factors are related to hypoxic insults, which can inturn affect the structure and function of the brain structures associated with ADHD (Das Banerjee et al., 2007). However, it is important to recognise that the majority of these birth complications lead to a relatively low proportion of children with ADHD. Some suggest these factors play a role some kind of gene-environment interaction (APA, 2000; Brock, Jimerson, & Hansen, 2009).

Diet, food additives, and allergies

The role of diet and food additives in the aetiology of ADHD has received particular attention in research and press and is a topic of great controversy (Das Banerjee et al., 2007). Some argue there are a subset of children with ADHD who are sensitive to specific foods which may be related in the development and maintenance of their ADHD (Schnoll, Burshteyn, Cea-Aravena, 2003). However, these findings have been disputed in recent review studies. These reviews did not find any significant associations between diet, ADHD, and behaviour in children (Brock, Jimerson, & Hansen, 2009; Das Banerjee et al., 2007; Ells et al., 2008). Research suggests that for the vast majority of children, refined sugar and food additives do not cause or perpetuate ADHD symptoms (Brock, Jimerson, & Hansen, 2009; Das Banjeree et al., 2007).

There is some research to suggest that iron and zinc deficiencies play a role in the aetiology of a range of neurological disorders in children (Millichap, 2008). Research supports that while iron supplementation is helpful in children with ADHD, a causal role has not been established (Millichap, 2008). Studies have found an association between low zinc levels in children and parent/teacher ratings of inattentiveness (Arnold, Bozzolo, Hollway et al., 2005). However, there is a need for further replication of these findings (Millichap, 2008).

There is some research to suggest that omega-3 fatty acids play a role in the aetiology of ADHD (Richardson, 2006). This is consistent with the finding that children with hyperactivity problems often have abnormally low serum levels of essential fatty acids (Millichap, 2008). A review study concluded that while this research was promising, further experimental studies are required before any causal connections can be made into the relationship between omega-3 fatty acids and ADHD (Richardson, 2006).

Child Temperament

The idea that child temperament is related to attachment problems and later behavioural issues is also widely supported in the literature. Mothers of inattentive and hyperactive children frequently report that during infancy their children were highly active, hyper-irritable, un-soothable, resistant to change, and unable to regulate difficult emotions such as anger and frustration (Clarke et al., 2002; Finzi-Dottan, Manor, & Tyano, 2006; McCartney et al., 2004; Pierrehumbert et al., 2000; Shaw et al., 1996). These infants are reported to be difficult to care for and their behaviours impose a unique strain on parents (Clarke et al., 2002). Parents of these children are more likely to consider their parent abilities to be lacking and to report lower parental satisfaction (Finzi-Dottan et al., 2006; Shelton et al., 1998). It is difficult to disentangle whether it is a negative parenting style that leads to a difficult child temperament, or whether a difficult child temperament leads to a negative parenting style. However, many theorists agree upon the idea that there is a transaction

between the parent and the child (Finzi-Dottan et al., 2006; Johnston & Mash, 2001; Pierrehumbert et al., 2000). This idea will be discussed further in the parenting and family factors section.

Parent- child attachment

It is now widely accepted that there is an association between insecure attachment styles and the development of disruptive behaviour problems in children (Bauermeister et al., 2007; Clarke, Ungerer, Charhoud, Johnson, & Ingeborg, 2002; DeVito & Hopkins, 2001; McCartney et al., 2004; Moss et al., 1998; Pierrehumbert, Milijkovitch, Plancherel, Halfon, & Ansermet, 2000; Shaw et al., 1996). It has been suggested that the deficits in behaviour and emotional regulation seen in children with ADHD may reflect disrupted primary attachments and dysfunctional parent-child interactions (Clarke et al., 2002; McCartney et al., 2004; Speltz et al., 1999). Research has demonstrated that children with ADHD experience greater disruptions in early attachment relationships, compared to children without ADHD (Conway et al., 2011). In addition, large-scale population studies have identified insecure parent-child attachments as risk factors for ADHD (Bauermeister et al., 2007). It is thought that early disruptions in attachment relationships can alter the way a child functions in the world (Conway et al., 2011).

More specifically, research has shown that disorganised and ambivalent attachment styles significantly predict the likelihood of externalising and problem behaviour in school age children (Clarke et al., 2002; Moss et al., 1998). Other research findings suggest avoidant attachment styles are linked to disruptive behaviour disorders, including ADHD (Pierrehumbert et al., 2000). Studies looking at preschoolers have found a significant relationship between disruptive behaviour problems and both ambivalent and disorganised attachment styles (Devito & Hopkins, 2001; Madigan et al., 2007).

In summary, there is a clear relationship between attachment insecurity and disruptive behaviour problems such as ADHD. However, research has produced mixed findings into the

specific type of insecure attachment style that is associated with ADHD. This suggests that this relationship is complex and may be moderated by many other factors such as child temperament and parenting styles (Greenberg et al., 2003).

Parenting and family factors

Studies of parents of children with ADHD suggest that some parents adopt a more coercive parenting style that is negative, reactive, directive, and commanding (Bauermeister et al., 2007; Johnston & Mash, 2001; Pfiffner et al., 2005; Stormshack, Bierman, McMahon, & Lengua, 2000). Furthermore, parenting styles that lack warmth and a failure to respond to positive/neutral behaviour have been found to relate to hyperactive and oppositional behaviour in children (DeVito & Hopkins, 2001; Clarke et al., 2001; Shelton et al., 1998).

While research into the nature and direction of the child-parent relationship has produced inconsistent results, many theorists agree upon the idea that there is a transaction between the parent and the child (Finzi-Dottan et al., 2006; Johnston & Mash, 2001; Pierrehumbert et al., 2000). This transaction could work in two ways. A child's difficult temperament may impact negatively on parenting style, which may in turn exacerbate the child's difficulties and have an overall detrimental effect on the parent-child attachment (Finzi-Dottan et al., 2006). Conversely, an early dysfunctional parenting style may play an aetiological role in the development of a 'difficult' child temperament, which in turn may have a negative impact on the parent-child attachment (Johnston & Mash, 2001; Pfiffner et al., 2005).

Maternal mental health problems such as anxiety, depression, and alcohol and drug use have been associated with inattentive, disruptive, and aggressive behaviours in children (Erdman, 1998; Kashdan et al., 2004; Shaw et al., 1996). Maternal anxiety is associated with parents having less warmth towards their children, a negative discipline style, and an overall negative impact on the parent-child attachment (Kashdan et al., 2004). Large-scale population studies have found parental

and maternal mental health problems to be significant risk factors for ADHD (Bauermeister et al., 2007). Furthermore, research investigating samples of children with a known ADHD diagnosis has found up to 71% have a parent with a mental health problem (Becker-Blease & Freyd, 2008).

Research has consistently shown that parental discord, low levels of marital satisfaction, and divorce are all linked to disruptive behaviour problems in children (Amato, 2000; Das Banerjee et al., 2007; DeVito & Hopkins, 2001; Johnston & Mash, 2001). The negative impacts of divorce are cumulative; the loss of a parent, financial stress and changes in family routine (Amato, 2000). Further studies have found psychosocial factors such as low parent education, adolescent parenting, single parenting, financial stress, and chaotic family environments are all significant predisposing factors associated with a diagnosis of ADHD (Amato, 2000; Connor, 2002; DeVito & Hopkins, 2001; Erdman, 1998; Johnston & Mash, 2001). In a large-scale Swedish study examining over one million school age children, there was a significant association between negative psychosocial factors and ADHD. These factors included: low maternal education (predicted 33% of cases), single parenting, financial stress (Hjern, Weitoft, Lindblad, 2009). Further research has found impoverishment to be significantly associated with a diagnosis of ADHD (Bauermeister et al., 2007).

Child maltreatment

There is a growing body of research to suggest that adverse life events such as neglect, physical abuse, and sexual abuse, play an aaetiological role in ADHD (Briscoe-Smith & Hinshaw, 2006; Endo et al., 2006; Famularo et al., 1993; Famularo et al., 1996; McLeer et al., 1994; McLeer, Deblinger, Henry, & Orvaschel, 1992; McLeer et al., 1998; Merry & Andrews, 1994). Similar adverse life experience that play a causal role for other mental health problems, have been associated with the development of ADHD (Read & Sanders, 2010).

An area that has received particular attention in recent literature is the relationship between ADHD and child maltreatment and/or trauma (Conway, Oster, & Szymanski, 2011; Szymanski, Sapanski, & Conway, 2011).Various studies have found a relationship between child maltreatment and/or trauma and the symptoms of ADHD (Briscoe-Smith and Hinshaw, 2006; Endo, Sugiyama, & Someya, 2006; Famularo, Fenton, & Kinscherff, 1993; Kendall-Tackett et al., 1993; McLeer et al., 1994). Recent studies have found that up to 71% of children with an ADHD diagnosis have experienced at least one form of child maltreatment and/or traumatic experience (De Sanctiset al., 2008). The research looking into the relationship between ADHD and child maltreatment and trauma is extensive and will be discussed in greater detail in next section of this review.

Despite many studies finding a relationship between ADHD and child maltreatment and/or trauma, this relationship is complex and there are many conflicting hypothesises about the nature and direction of this relationship (Szymanski et al., 2011). Of particular relevance to the current study, one hypothesis suggests that there may be a distinct sub-group of children with 'complicated ADHD', who are likely to have a history of childhood maltreatment and/or trauma. Children with 'complictaed ADHD' are thought to have higher rates of co-morbidity with PTSD, ODD, or CD, and higher levels of problematic externalising behaviour. Others argue that ADHD and PTSD have similar clinical presentations in children and that the high rates of maltreatment and/or trauma found in children with ADHD is merely an issue of high symptom overlap rather than an aetiological relationship (Findling et al., 2007; Weinstein et al., 2000). Further research argues that children with ADHD are more susceptible to child maltreatment and/or trauma because of their disruptive behaviour and impulse control problems (Ouyang et al., 2008; Whitmore, Kramer, & Knutson, 1993; Wozniak et al., 1999). The belief behind this finding is that parents, who are unaware that their child's inattentive behaviour is attributable to a disorder, consequently blame and punish the child through physical punishment and/or neglect. In summary, the relationship between

adverse life events and ADHD is complex, and the main hypotheses around this relationship will be discussed in greater detail in the next section.

Summary of environmental factors

Based on relevant literature, it is clear that there are a wide range of environmental risk factors that relate to the aetiology of ADHD. There is extensive research to suggest that pre-natal maternal factors such as maternal smoking and alcohol use are risk factors for developing ADHD (Das Banerjee et al., 2007; Carr, 1999; Millichap, 2008). These prenatal factors are thought to play a role in some of prefrontal brain abnormalities found in children with ADHD (Barkley, 2006). There are also a number of birth related risk factors (e.g. premature birth, fetal distress, and low birth weight) that are related to hypoxic brain insults, which in turn can affect some of the brain regions associated with ADHD (Das Banerjee et al., 2007). In terms of diet, there is some evidence to suggest that iron, zinc, and omega-3 fatty acids play a role in ADHD (Arnold et al., 2005; Millichap, 2008 Richardson, 2006). Other research suggests that for the vast majority of children, refined sugar and food additives do not cause or perpetuate ADHD symptoms (Brock, Jimerson, & Hansen, 2009; Das Banjeree et al., 2007).

Research has also found a number of psychosocial risk factors involved in the aetiology of ADHD. These include maternal mental health problems, parental discord, low levels of marital satisfaction, divorce, financial stress, low parent education, and single parenting (Amato, 2000; Connor, 2002; Das Banerjee et al., 2007; DeVito & Hopkins, 2001; Erdman, 1998; Hjern et al., 2009; Johnston & Mash, 2001; Kashdan et al., 2004; Shaw et al., 1996).

Furthermore, there is a growing body of evidence to suggest that child maltreatment and/or trauma plays a role the aetiology of ADHD (Briscoe-Smith and Hinshaw, 2006; Endo, Sugiyama, & Someya, 2006; Famularo, Fenton, & Kinscherff, 1993; Kendall-Tackett et al., 1993; McLeer et

al., 1994). The relationship between adverse childhood experiences and ADHD is complex, and this will be looked at in closer detail later in this introduction.

In summary, the wide range of environmental risk factors involved in ADHD creates a complex aaetiological picture. This supports the idea that there are multiple factors that interact in producing the disorder, or multiple pathways that perhaps lead to various manifestations of the disorder (Nigg, 2006; Steinhausen, 2009).

CHILDHOOD ADVERSE EXPERIENCES AND SEQUENTIAL PSYCHOPATHOLOGY

Before discussing the complex relationship between adverse childhood events and ADHD it is important to briefly review the literature on adverse childhood events, the negative impacts of abuse, post traumatic stress disorder (PTSD), and some of the research investigating childhood adverse experiences and sequential psychopathology.

Adverse Childhood Events in New Zealand

Childhood adverse events include: physical abuse, sexual abuse, emotional abuse, emotional neglect, physical neglect, chaotic family environment, impoverishment, parental discord, grief and loss. For detailed definitions of adverse events see *Appendix C*. Many children in New Zealand continue to suffer from abuse, neglect, and adverse events of all types. New Zealand was shown to have the fifth worst child death by maltreatment rate of 27 OECD (organisation for economic cooperation and development) countries included in a recent report by the United Nations International Children's Emergency Fund (UNICEF, 2003). This report estimated that there were 1.2 per 100,000 deaths from maltreatment in children under 15 years living in New Zealand over a 5 year period (UNICEF, 2003). Children and adolescents brought up in deprived families and communities in New Zealand are more likely to experience child maltreatment such as physical

abuse, sexual abuse and neglect (Centre for Social Research and Evaluation, 2008). Other research in New Zealand has identified that individual's of Maori ethnicity report higher rates of physical abuse and exposure to domestic violence in childhood (Marie, Fergusson, & Boden, 2009).

In a New Zealand prevalence study, 32% of women were found to have a history of sexual abuse before the age of 16 years (Mullen, Martin, Anderson, Romans, & et al., 1993). This prevalence rate increases in populations of mental health users, with approximately half (44-64%) of psychiatric inpatients experiencing some form of childhood trauma (Read, 2007). However, it is assumed this number is largely underestimated, as abuse and trauma is more often than not unreported (Briscoe-Smith & Hinshaw, 2006; Read, 2007; Read & Fraser, 1998; Read, Van Os, & Morrison, 2006). Other confounding factors in obtaining an accurate prevalence of childhood trauma include the reliance on retrospective information, subjective impressions, memory deterioration, and selective recall of abuse (Dubowitz, Black, Harrington, & Verschoore, 1993; Agar & Read, 2002).

The Impacts of Adverse Childhood Events and Post Traumatic Stress Disorder

Children with a history of abuse have been found to exhibit both externalising and internalizing symptoms (Briscoe-Smith & Hinshaw, 2006; Dubowitz et al., 1993). Child sexual abuse has been linked to PTSD type symptoms such as heightened anxiety, hyper-vigilance, impaired impulse control, fantasy, dissociation, and inattentiveness (Briscoe-Smith & Hinshaw, 2006; Dubowitz et al., 1993; Weinstein et al., 2000). PTSD is defined by the *DSM-IV-TR* as the development of characteristic symptoms following exposure to an extreme traumatic stressor. In which, the individual's response involves fear, helplessness, or horror (APA, 2000). In children, the response must involve disorganized or agitated behaviour. The characteristic symptoms following the exposure to the traumatic event are re-experiencing, avoidance of stimuli associated with the trauma and symptoms of increased arousal. To constitute a diagnosis these symptoms have to be

present for more than one month and the disturbance must cause clinically significant distress or impairment in social, education, and other important areas of functioning.

In addition to PTSD type symptoms, childhood physical and sexual abuse has been linked to externalising behaviour problems such as increased aggression, hostility, anger problems, and impulsivity (Weinstein et al., 2000). When considering the combination of the internalising and externalising symptoms exhibited by these children, it is perhaps unsurprising that PTSD and ADHD are the two most commonly diagnosed disorders in children with a history of trauma (McLeer, Callaghan, Henry, & Wallen, 1994; Weinstein et al., 2000).

Research Investigating Childhood Adverse Experiences and Sequential Psychopathology

Numerous studies have investigated the psychiatric status of children with a history of sexual abuse. For example, McLeer et al. (1994) compared the prevalence of psychiatric disorders among a sample of 26 sexually abused children in comparison to 23 matched, non-sexually abused children referred to an outpatient clinic. ADHD was the most frequently diagnosed disorder among the sexually abused children, with 46% warranting a *DSM-III-R* diagnosis, compared to 31% of the non-sexually abused group. However, when comparing the two groups, the prevalence of PTSD was the only significant distinguishing factor with 42% of the sexually abused group warranting a diagnosis of PTSD, in comparison to 8% of the non-sexually abused group. There was also a significant co-morbidity among the sexually abused children, in which 23% of these children had comorbid diagnoses of PTSD and ADHD. In further research by the same author, there were higher rates of PTSD, externalising behaviours, depression, and anxiety among non-clinically referred sexually abused children, in comparison to clinically and non-clinically referred aged-matched groups of non-abused children (McLeer et al., 1998). In support of this research, a New Zealand study assessed 95 sexually abused children in New Zealand using semi-structured interviews

(Merry & Andrews, 1994). The diagnoses ODD, PTSD, separation anxiety disorder, and ADHD were the four most commonly diagnosed disorders among the sexually abused children, all of which were diagnosed at a significantly higher rate than the community population. More specifically, children with a history of sexual abuse were twice as likely to warrant a diagnosis of ADHD compared to the community population. In an Australian study, sexually abused children who were clinically referred for assessment were compared to aged-matched school children. The sexually abused children were found to have higher rates of depression and behavioural disturbances (Stern, Lynch, Oates, Otoole, & Cooney, 1995). In summary, research suggests that children with a history of sexual abuse have higher than expected rates of PTSD, ADHD, depression, anxiety, and ODD in comparison to non-abused children. (McLeer et al., 1994; McLeer et al., 1998; Merry & Andrews, 1994; Stern et al., 1995)

From the literature it is clear that the impact of sexual abuse is serious and can manifest in a wide range of symptoms and psychopathology. This is further demonstrated in a review of 45 studies which found children with a history of sexual abuse were consistently shown to have a range of problematic symptoms and disorders (e.g. PTSD, ADHD, disruptive behavioural problems, depression, and sexualised behaviours) in comparison to non-abused children (Kendall-Tackett et al., 1993). In a more recent review, Webster (2001) found children with a history of sexual abuse to present with a wide range of symptoms including attention problems, hyper-vigilance, disruptive behaviour problems, social withdrawal, agitation, emotional reactivity, depressed mood and gender and identity problems. These reviews highlight the variability of sexually abused children. The research suggests that children with a history of sexual abuse are a highly heterogeneous group presenting with a range of symptomology that does not constitute a distinct, identifiable syndrome.

Consequentially, more recent research has attempted to cluster sexually abused children into mental health outcome groups (Hebert, Parent, Daignault, & Tourigny, 2006; Hinshaw &

Blackman, 2005; Trickett, Noll, Reiffman, & Putnam, 2001). For example, Hébert et al. (2006) assessed children with a history of sexual abuse and identified four different outcome groups. The first group (33%) constituted children with a history of chronic sexual abuse presenting mainly with anxiety/PTSD symptoms, the second (25%) revealed children with pervasive behavioural problems, reaching clinical levels for both externalising and internalising behavioural difficulties, the third (16%) included resilient children whom displayed less negative outcomes, despite having a history of severe, intra-familial sexual abuse and the fourth (26%) included those children experiencing less-severe sexual abuse whom displayed less negative outcomes. In summary, this research suggests children with a history of sexual abuse are a diverse group; some presenting with significant emotional trauma and psychological problems, while some children appear to be more resilient (Herbert et al., 2006).

A major limitation of the research discussed above is these studies only examined children with a history of sexual abuse which may lead to specific symptoms, thus diagnoses, in comparison to other forms of childhood maltreatment and/or trauma. Thus it is pertinent to consider other forms of maltreatment and/or trauma, other than sexual abuse, when investigating the impact of childhood adverse experiences. Further research has compared maltreated children (physical abuse, sexual abuse, neglect, and impoverishment) with matched, non-maltreated controls (Famularo, Fenton, & Kinscherff, 1993). The study found PTSD, ADHD, and ODD, all to be strong discriminates between the maltreated and non-maltreated groups of children. There were significantly higher rates of diagnosis for all three of these disorders among the maltreated children. In a further study, psychiatric co-morbidity was examined in maltreated children with PTSD and maltreated children without PTSD symptoms (Famularo et al., 1996). The results from this study suggest children with a history of trauma and a diagnosis of PTSD are more likely to fit a diagnosis of ADHD (37%), compared to children with a history of trauma that do not fit the criteria for PTSD (17%). This implies children with a history of child maltreatment are highly likely to fit a co-morbid diagnosis

of PTSD and ADHD. Furthermore, in a Japanese study, Endo, et al. (2006) assessed a sample of children who were admitted to a psychiatric inpatient clinic for various types of abuse (physical, emotional and sexual abuse, and neglect). The study found 18% of these children to fit the criteria for ADHD and 59% to fit the criteria for dissociative disorder. Of these children with a diagnosis of ADHD, 71% also fit the criteria for dissociative disorder, suggesting that among traumatised children there is a high co-morbidity between the two disorders (Endo et al., 2006).

When interpreting this research it is important to take into account some of the methodological limitations. One important limitation is that some of these studies examined children in psychiatric inpatient settings (Endo et al., 2006). These children may exhibit very specific symptoms and diagnoses in comparison to children in community or out-patient clinic populations (Merry & Andrews, 1994; McLeer et al., 1994). Furthermore, the methods of categorisation and assessment of particular diagnoses and symptoms varies between the studies. For example, the study by Stern et al., (1995) utilized the Children's Depression Inventory (CDI) and CBCL to measure symptoms of depression and externalising behaviour problems, in comparison to other studies, e.g. Merry & Andrews, (1994), who used clinical assessment interviews in order to make diagnoses. The different methods of assessment may have produced variable rates of diagnoses and/or symptoms.

In summary, research investigating populations of children with a known history of maltreatment and/or trauma has consistently found higher than expected rates ADHD and other disruptive behaviour problems (Endo et al., 2006; Famularo, Fenton, & Kinscherff, 1993; Famularo et al., 1996; Kendall-Tackett et al., 1993; McLeer et al., 1994; McLeer et al., 1998, Merry & Andrews, 1994; Stern et al., 1995; Webster, 2001). Other diagnoses that are common among children with a history of trauma and maltreatment are PTSD, anxiety, depression, and dissociative disorder (Endo et al., 2006; Kendall-Tackett et al., 1993; Mcleer et al., 1998; Merry & Andrews, 1994; Stern et al., 1995). While diagnoses along the lines of PTSD, anxiety, depression and

dissociative disorder seem to make sense in children with a history of trauma and/or maltreatment, it is the high rates of ADHD and other disruptive behaviour disorders that have sparked recent research interest. In particular, researchers are interested in the high rates of co-morbidity between ADHD and disorders such as PTSD and dissociative disorder (Endo et al., 2006; Famularo et al., 1996). The relationship between adverse childhood experiences, ADHD and other disruptive behaviour problems will be discussed in greater detail in the following section.

THE EMPIRICAL LINK BETWEEN ADVERSE EXPERIENCES IN CHILDHOOD AND ADHD

The link between adverse events in childhood, PTSD, ADHD, and other disruptive behaviour disorders is complex, and conflicting research findings have sparked recent debate into the nature and direction of this complicated relationship (Findling et al., 2007). There are currently three main, conflicting hypotheses that attempt to explain the high overlap of ADHD, disruptive behaviour disorders, PTSD, and adverse childhood experiences.

Hypothesis I

The first hypothesis proposes that adverse childhood experiences are an etiologic and/or exacerbating factor in the development of attention deficit and disruptive behavioural disorders. This could work in two ways. Firstly, adverse childhood events may be a predisposing factor to ADHD, and hence play a role in the aetiological pathway of ADHD. Secondly, there may be a distinct subgroup of children within ADHD populations who have 'complicated ADHD' (Findling, Arnold, Greenhill, Kratochvil, & McGough, 2007, p. 229). Relevant literature refers to 'complicated ADHD' as children with ADHD who have a history of adverse childhood experiences, higher than expected rates of problematic externalising behaviour and aggressive

behaviour, and high rates of co-morbidity with PTSD, ODD, and CD, compared to children with ADHD with no history of adverse events (Briscoe-Smith & Hinshaw, 2006; Endo et al., 2006; R. Famularo et al., 1993; Richard Famularo et al., 1996; McLeer et al., 1994; McLeer, Deblinger, Henry, & Orvaschel, 1992; McLeer et al., 1998; Merry & Andrews, 1994).

Neurological evidence

Before reviewing the research relating to hypothesis one, it is important to understand the neurological impacts that chronic stress/trauma can have on the brain of a developing child. As mentioned previously, there is extensive evidence to suggest that a child's brain develops in response to the pattern, intensity, and nature of their sensory and emotional experiences during childhood (Perry, 2006). Furthermore, the developing brain is highly sensitive to stress (Perry, 2002; Perry, Pollard, Blakley, Baker, & Vigilante, 1995). Studies have suggested that exposure to unpredictable stress and trauma early on in life can result in long-term psychological and neurological deficits (Perry, 2002; Perry et al., 1995).

When a child experiences trauma or stress that is extreme or chronic in nature, the child's stress-response system is altered and the child can experience a "traumatic stress response" which locks the brain into a maladaptive feedback cycle (Wilson et al., 2011, p.89). In other words, children growing up in violent, neglectful, and chaotic environments adapt to become hypersensitive to potentially threatening external stimuli and in return are in a persistent stress-response state (Perry et al., 1995). While these adaptive changes in stress-response circuits in the brain make these children better suited to perceive and act on threat in stressful environments, these "survival tactics" do not serve well in other environments such as school and with peers (Perry, 2004, p. 4). This is particularly true when considering the learning experience of a traumatised child sitting in a classroom in a persisting state of arousal and anxiety. This persistent state of hyper-

arousal could resemble hyperactivity or inattentiveness, especially in younger children (Glod &
Teicher, 1996).

The 'traumatic stress response state' can have a negative impact on the development of a
child's brain (Wilson et al., 2011). More specifically, the development of the hypothalamic-
pituitary-adrenal axis (HPA-axis), the prefrontal cortex, and the limbic system (DePrince et al.,
2009; Wilson et al., 2011). The prefrontal cortex is particularly susceptible to the affects of chronic
stress because it is still developing long into childhood and adolescent years (Wilson et al., 2011).
Neuropsychological research supports that that the prefrontal cortex is negatively affected by
chronic stress in childhood. A study by DePrince, Weinzeirl, & Combs (2009) found that trauma in
childhood is linked to poor executive functioning and attentional control. These findings are similar
to research that shows children with ADHD often have neuropsychological deficits in executive
functioning, response inhibition, delay aversion, and tasks that require sustained attention (Barkley,
2006; Brock, Jimerson, & Hansen, 2009; Kieling et al., 2008).

This hypothesis is supported by research that has found that family adversity moderates the
expression of genes that have been implicated in the aetiology of ADHD (Brock, Jimerson, &
Hansen, 2009; Edel, Juckel, & Brune, 2010; Steinhausen, 2009). More specifically, Laucht et al.,
(2007) study found that individuals with the (DAT1) gene only developed symptoms of ADHD if
exposed to psychosocial adversity, in comparison to those living in less adverse family conditions.
This suggests that it is a combination of genetic and adverse psychosocial factors (such as child
maltreatment and/or trauma) that cause ADHD (Edel, Juckel, & Brune, 2010; Laucht et al., 2007;
Steinhausen, 2009).

In summary, research shows that exposure to chronic trauma and/or stress can lead to long-
term neurological problems in children. In particular, exposure to stress and/or trauma has been
associated with problems in the prefrontal cortex, which in turn, can lead to problems in executive
functioning ability, and consequential ADHD-type symptoms.

Literature supporting an aetiological relationship

From research discussed earlier in this review, it is clear that ADHD is a common diagnosis among populations of children with known history of trauma and maltreatment (Endo et al., 2006; Famularo, Fenton, & Kinscherff, 1993; Famularo et al., 1996; Kendall-Tackett et al., 1993; McLeer et al., 1994; McLeer et al., 1998, Merry & Andrews, 1994; Stern et al., 1995; Webster, 2001). Based on this finding, there is now a growing body of research investigating the role of childhood adverse events in the aetiology of ADHD. In a very recent review of the literature, Szymanski et al. (2011) suggest that there are multiple studies have found evidence to support that adverse life events, such as childhood trauma, may exacerbate symptoms of ADHD or even cause ADHD.

The idea that adverse life events are aetiologically related to ADHD is supported in a study discussed earlier in this review by Endo et al., (2006). In a psychiatric inpatient sample, children with a history of various types of adverse events (physical, emotional and sexual abuse, and neglect), 59% fit the criteria for dissociative disorder, 18% fit the criteria for ADHD and there was 71% co-morbidity between DD and ADHD. Only a small proportion of the children diagnosed with ADHD in the sample showed ADHD–type symptoms before their documented abuse, suggesting abuse may be aetiologically related to ADHD.

Taking a slightly different approach, Glod et al. (1996) measured circadian rest-activity as an objective measure of the hyperactivity of children with and without a history of abuse. Overall, the abused group of children were 10% more active than the non abused control group, with the majority of these highly active/abused children fitting a diagnosis of PTSD. The authors concluded that children with a history of child abuse have activity profiles similar to those of children with ADHD, regardless of whether or not the child fits a diagnosis of PTSD. This supports the idea that childhood trauma may exert neurological changes in a child's brain that lead to hyperactive, ADHD-type symptoms.

Rucklidge et al. (2006) conducted a retrospective study in Canada to investigate the trauma histories of adults with ADHD. The adults with ADHD were significantly more likely to have a history of childhood trauma in comparison to adults without ADHD (56% vs. 18-20%, respectively). Females in this sample of adults were twice as likely to have a history of abuse. However, the authors concluded that it was difficult to distinguish whether childhood trauma preceded the ADHD diagnosis as it was hard to determine the exact timing of these events. Therefore, it is difficult to make any conclusions in whether or not the abuse played an aetiological role in the development of ADHD symptoms.

In a very recent study, the charts of 79 children and adolescents in an inpatient psychiatric unit were examined for symptoms of ADHD, environmental trauma, and attachment trauma (Conway, Oster, & Szymanski et al., 2011). Children with ADHD were found to experience higher rates of environmental trauma (e.g. domestic violence and exposure to chronic stress) and attachment trauma (e.g. neglect, maltreatment from caregivers, death of a parent or caregiver, adoption, foster care placement, or caregiver incarceration), in comparison to non-ADHD children in the unit. This study supports an association between ADHD and adverse life events, but again, cannot confirm that environmental and attachment trauma preceded the onset of ADHD symptoms.

In further research, the idea that there may be a subgroup group of children with 'complicated ADHD' has been investigated. Briscoe-Smith and Hinshaw (2006) examined the rates of child physical and sexual abuse in a large sample of girls with ADHD. Girls with ADHD were significantly more likely to have a history of abuse than their healthy peers. For some girls in the study, abuse from a young age clearly preceded their diagnosis of ADHD. Thus supporting the hypothesis that trauma could be aetiologically related to ADHD. Additionally, girls with both ADHD and abuse histories were found to exhibit higher levels of externalising behaviour in comparison to girls with ADHD and no history of abuse. More specifically, this distinct sub-group of girls with ADHD and abusive histories were more aggressive and had higher rates of peer

rejection. For a subgroup of girls, with ADHD and a history of abuse, to exhibit higher levels of aggression and peer rejection than the rest of the ADHD sample, provides evidence of a group of children with 'complicated ADHD' who exhibit higher levels of dysfunction.

This idea of 'complicated ADHD' is supported in an earlier study by King, Russel, & Barkley (1998) that found a particular group of children with ADHD who also had an impaired stress-response. The children with ADHD and an impaired stress-response were found to have a more developmentally persistent form of ADHD in comparison to the other ADHD children in the study (King et al., 1998). Although the study did not assess for previous history of adverse events, the study provides evidence that there may be a sub-group of children with ADHD that have an impaired stress-response, and a more serious and persistent form of ADHD.

In a recent preliminary study, Becker-Blease & Freyd (2008) examined the ADHD symptoms of children with a known history of abuse and a control group. The abused group of children were found to have higher rates of impulsivity and inattentiveness, but not hyperactivity, compared to the control group. The abused group of children were also more likely to have a parent with a mental health problems (71% had a parent with ADHD).

Summary

In summary, children who are exposed to chronic trauma and/or stress may have alterations in brain neurochemistry which puts them in a persistent stress-response state. Some argue that this constant state of hyper-arousal may lead to ADHD-type symptoms of hyperactivity and inattentiveness (Glod & Teicher, 1996; Perry, 2004). Furthermore, exposure to childhood trauma and/or chronic stress may exert neurological changes in a child's brain. In particular, problems in the prefrontal cortex have been associated with difficulties in executive functioning and attention.

Based on relevant literature, there is a clear relationship between adverse childhood events and symptoms of ADHD (Briscoe-Smith & Hinshaw, 2006; Endo et al., 2006; Falmaro et al., 1992;

Falmaro et al., 1996; McLeer et al., 1992; McLeer et al., 1994; Merry et al., 1994). There is also

evidence to suggest that adverse events in childhood may play an aetiological role in the

development of ADHD (Briscoe-Smith & Hinshaw, 2006; Endo et al., 2006; Glod et al., 1996;

Rucklidge et al., 2006). However, it is difficult to establish a causal connection between adverse

childhood events and ADHD because in most of the studies it was difficult to establish a time-line

of events (e.g. whether abuse/trauma preceded ADHD symptoms or vice versa). However, in

research by Briscoe-Smith & Hinshaw (2006) and Endo et al. (2006), there was some evidence that

abuse/trauma preceded abuse symptoms in some children. This suggests that abuse and/or trauma

may have played an aetiological role in the development of ADHD symptoms.

There is also research to support that there may be a distinct sub-group of children with

'complicated ADHD' (Findling et al., 2007), who have a history of abuse and/or trauma, along with

symptoms consistent with a diagnosis of ADHD. Furthermore, this subgroup of children are found

to have higher rates of co-morbid PTSD, problematic externalising behaviour, hyperactivity, and a

more developmentally persistent form of ADHD (Briscoe-Smith & Hinshaw, 2006; Glod et al.,

1996; King et al., 1998). However, this research is limited and there is a need for further

replication of these findings.

Hypothesis II

The symptoms of PTSD and ADHD are similar and some argue the high overlap of

childhood adverse events, PTSD and attention deficit and disruptive behavioural disorders is an

issue of high symptom overlap rather than an aetiological relationship (Conway et al., 2011;

Findling et al., 2007; Szymanski et al., 2011; Weinstein et al., 2000).

Although research supports the idea that there may be a distinct subgroup of children with

'complicated ADHD', others have challenged the idea that ADHD is aetiologically related to

childhood trauma. Upon closer examination, the symptoms of PTSD and ADHD are similar and

others argue the high overlap of childhood trauma, PTSD and ADHD my reflect symptom similarity rather than an aetiological relationship (Findling et al., 2008; Weinstein et al., 2000; Szymanski et al., 2011).

In a review article, Weinstein et al. (2000) outline the high degree of symptoms overlap and co-morbidity between ADHD and PTSD and state how differential diagnosis can be confusing in children with a history of sexual abuse. More specifically, Weinstein et al. (2000) argue that symptoms in a child with PTSD such as problems concentrating, hyper-vigilance to threat stimuli and re-experiencing of threat stimuli highly overlap with the inattention symptoms in a diagnosis of ADHD. In a more recent review, Szymanski et al., (2011) emphasise the similarity between the avoidance symptoms in PTSD and the inattentiveness cluster of ADHD, e.g. inattentiveness, distractibility, and avoidance of activities.

Furthermore, Weinstein et al. (2000) suggests symptoms such as; irritability, exaggerated startle response, anger outbursts, and avoidance in children with PTSD are highly similar to the hyperactivity and externalising behaviour found in children with a diagnosis of ADHD. This is also supported by Szymanksi et al. (2011) who compares the hyper-arousal symptoms in PTSD to hyperactive symptoms in ADHD such as: fidgeting, excessive moving around, and restlessness. It has also been suggested that anxiety in PTSD may be relate to the impulsive behaviour in ADHD (Szymanski et al., 2011).

Based on this hypothesis, some argue the similarity in symptoms between ADHD and PTSD can lead to misdiagnosis (Szymanski et al., 2011). Some argue that PTSD in children is often misdiagnosed as ADHD because ADHD symptoms are more external and easier to detect (Conway et al., 2011; Szymanski et al., 2011). Furthermore, this issue may be more prevalent in children because they are known to manifest PTSD symptoms differently to adults, and may display more externalising symptoms (Conway et al., 2011). The high overlap of ADHD and PTSD symptoms has led some researchers to caution that there may be a high number of children with a

diagnosis of ADHD, along with undetected trauma symptoms and in some cases, undiagnosed

PTSD (Ford et al., 2000). This is supported in other reviews that suggest that the risk of

misdiagnosis is very high, and that extra caution is necessary when diagnosing ADHD in children

with a history of trauma (Szymanski et al., 2011).

Hypothesis III

The third hypothesis proposes that ADHD, CD, and ODD are potential risk factors or

predictors for later adverse events such as physical abuse. Some argue that children with attention

deficit and disruptive behavioural disorders are more susceptible to abuse due to their disruptive

behaviour and impulse control problems (Ouyang et al., 2008; Whitmore, Kramer, & Knutson,

1993; Wozniak et al., 1999).

The literature supporting this hypothesis suggests that children with ADHD are susceptible

to abuse due to their disruptive behaviour and impulse control problems (Lam, 2005; Ouyang et al.,

2008; Weerts-Whitmore & Kramer, 1993; Wozniak et al., 1999). One study looked at whether

children with ADHD are more likely to be victims of violence by examining information routinely

collected on hospitalised patients owing to injuries (Lam, 2005). The study found children with a

diagnosis of ADHD are at higher risk of being victims of assaults. Other research has found that

over a course of 3-5 years, drivers with ADHD (aged 16-22 years) were four times as likely to be

involved in a motor accident, compared to drivers without ADHD (Barkley, Guevremont,

Anastopoulos, Dupaul, & Shelton, 1993). Similarly, Ouyang et al. (2008) carried out a regression

study to examine whether ADHD symptoms predicted later child abuse, in a large sample of 14,322

participants taken from the National Study of Adolescent Health in the USA. The study found a

significant relationship between inattentive symptoms of ADHD and subsequent physical and

sexual abuse, and/or neglect. Hyperactive ADHD symptoms were slightly associated with neglect

and physical abuse, however, not with sexual abuse. The belief behind this finding is that parents,

who are unaware that their child's inattentive behaviour is attributable to a disorder, consequently blame and punish the child through physical punishment and/or neglect.

Conversely, other research has not supported the theory that ADHD predicts maltreatment and trauma. In a longitudinal study, Wozniak et al. (1999) looked at the relationship between ADHD, PTSD, and trauma in boys to determine which precedes which. The study examined a large sample of boys with and without ADHD and measured whether the participants were exposed to any trauma and if they developed PTSD during the four year follow-up period. There were no significant differences found between the boys with ADHD and without ADHD, either in the rate of trauma exposure or in the development of PTSD. Thus suggesting ADHD may not be a risk factor for either trauma exposure or PTSD. Furthermore, Whitmore et al. (1993) examined the abuse histories and home environments of males with ADHD in comparison to their non-ADHD classmates and siblings. There were no significant differences in the rate of physical abuse between those with ADHD in comparison to their siblings or classmates. The findings of both these studies challenge the theory that children with ADHD are likely to provoke abuse and neglect from their parents, peers, and/or teachers due to their disruptive behaviour and inattentive problems (Whitmore et al., 1993; Wozniak et al., 1999).

Summary: The Empirical Link between Adverse Experiences in Childhood and ADHD

In summary, ADHD and PTSD are the two most common disorders diagnosed in children with a history of trauma. There is also a high level of co-morbidity between ADHD, ODD, CD, and PTSD in children who have experienced adverse childhood events. However, the link between adverse events in childhood, ADHD, PTSD, and other disruptive behaviour disorders is complex, and conflicting research findings have sparked recent debate into the nature and direction of this complicated relationship.

As discussed previously, the evidence to support each of the three hypotheses listed above is ambiguous, with studies producing conflicting findings. Thus the relationship between adverse experiences in childhood, ADHD, and other disruptive behavioural disorders remains unclear. Furthermore, aside from the study by Merry and Andrews (1994) looking specifically at the impact of sexual abuse, there has been little research carried out in New Zealand on this topic. This brings us to the aims of the current study:

<center>**Aims and Research Questions**</center>

The aim of the present study is to explore, and attempt to disentangle, the complicated relationship between ADHD and adverse childhood experiences by answering two main research questions:

1. Is there a relationship between (i) adverse childhood events and (ii) the diagnoses and symptoms of ADHD, PTSD and other disruptive behavioural disorders (ODD and CD), in children and adolescents referred to a child and adolescent mental health centre in New Zealand?

Based on international research findings, the present study expects to see a significant relationship between the symptoms of ADHD and adverse childhood events (Conway, Oster, & Szymanski et al., 2011; Endo et al., 2006; Famularo, Fenton, & Kinscherff, 1993; Famularo et al., 1996; Kendall-Tackett et al., 1993; McLeer et al., 1994; McLeer et al., 1998; Merry & Andrews, 1994; Stern et al., 1995). Furthermore, it can be expected that the symptoms of other disruptive behaviour disorders (ODD and CD) and PTSD are also significantly related to adverse childhood events, as found in previous studies (Famularo, Fenton, & Kinscherff, 1993; Famularo et al., 1996; Herbert et al., 2006; Merry & Andrews, 1994; Webster, 2001). These relationships will be explored in light of the three main hypotheses discussed above.

2. Secondly, do children with symptoms of ADHD and a history of adverse childhood experiences constitute a 'complicated ADHD' group?

Based on previous literature, it is hypothesised that there will be a group of children with symptoms of ADHD that will have a more 'complicated ADHD'. It can be hypothesised that children with 'complicated ADHD' will have a high rate of co-morbidity with other *DSM-IV* disorders and more serious externalising symptoms (i.e. a higher number of *DSM-IV* symptoms of the ADHD, CD and ODD disorders). It can also be hypothesised that this group of children will have a higher rate of childhood adverse events in comparison to other children with symptoms of ADHD (Briscoe- Smith & Hinshaw, 2006; Glod et al., 1996; King et al., 1998). This study will add to previous literature by examining the nature of this 'complicated ADHD' group. This will be achieved by looking specifically at the symptom profile of the group, the average number of adverse events, the rates of individual adverse events (e.g. sexual abuse, neglect etc), the rate of diagnostic co-morbidity, the levels of risk to self and others, rates of deliberate self-harm, and previous suicide attempts.

CHAPTER II: METHOD

Study design

A retrospective, non-experimental, file audit study design was employed. Data was collected from the electronic clinical files of child and adolescents at an urban Child and Adolescent Mental Health Service (CAMHS). This data included information regarding symptoms, diagnoses, and adverse childhood events. This was the method of choice for various reasons. Firstly, resource and time limits of a doctoral thesis limited research options such as using face to face interviews, particularly when a big sample was required in order to provide statistical power. Secondly, the aims of the study involve questioning a potentially vulnerable population about a sensitive subject. The researchers did not have the available resources to manage the repercussions of abuse and adverse childhood events being disclosed for the first time. In addition, seeking ethical approval for research options such as interviews may have been problematic due to this issue. Finally, a clinical file review is a good way of obtaining a wide range of information from various participants across a particular period of time.

There are also important limitations to this methodology, which are outlined in the discussion chapter.

Participants

The participants in the study were 217 child and adolescent clients of an urban, publicly funded, New Zealand CAMHS. The electronic clinical records of these clients were analysed. All data was anonymised by using a depersonalised number for each file. Therefore, individual identification was not possible.

The sample size of at least 200 was chosen to ensure that variables regarding types of adverse childhood events, symptomology, age, gender, ethnicity, and so on, were large enough to afford a reasonable degree of statistical power for analysis. Furthermore, this sample size was

selected to ensure an adequate number of children with symptoms of ADHD and other disruptive behaviour disorders were included within the sample, in order to address the main aims of the study. This was established by examining data already available within the last 1300 admissions to the CAMHS, regarding the proportion of children with a diagnosis of ADHD, ODD, and/or CD. Furthermore, the study intended to build on previous adult studies in which 200 consecutive files were audited (Read, Agar, Argyle, & Aderhold, 2003). The sample size was also limited to 217 participants due to time constraints, available resources, and the size limits of a Doctoral thesis. The inclusion and exclusion criteria for which participants were included in the study will be discussed later in the methods section in procedure.

The CAMHS used in the study is a publicly funded, community service that provides assessment and treatment for children and adolescents with a known or suspected mental disorder, at times of urgency or crisis. The CAMHS has a number of teams providing care. These include four community teams split geographically (Pohutakawa, Koru, Kowhai, and Ngakau), an early intervention team, a liaison service, and a forensic team. The teams are made up of mental health professionals including psychiatrists, psychologists, occupational therapists, nurses, social workers, and cultural support workers. The community teams provide assessment and treatment for children and adolescents with a known or suspected mental health disorder. The forensic team provides mental health assessments for young people with a known or suspected mental health disorder/s, who have engaged in offending behaviour and are involved with the justice system. The early intervention team is a specialty service for adolescents aged 13 - 19 years who may be experiencing a first episode of psychosis or bipolar affective disorder, who require specialist input. The liaison service is provided for children and adolescents with mental health needs who are under the care of the Department of Child Youth and Family Services (CYFS). In order to cover all admissions to the CAMHS within a certain time period, the clinical records from clients across all of the teams were included in the study.

The participants were all under the age of 18 years at the time of the referral and during the admission. When adolescents at the CAMHS turn 19 years, he/she is referred onto adult mental health services and the case is closed. All of the participants were clients of the CAMHS within a particular time period, and all had face to face contact with at least one mental health staff member of the service during their admission.

Apparatus

Information from the electronic clinical records was recorded by hand on a three page data collection form (*Appendix B*). The form was designed specifically for the present study. It was based on the data collection forms used in a similar study (Read et al., 2003). There was additional information recorded on the data collection form which was not included in the current study. This is because the current study only analysed the most salient variables and the additional information will be analysed at a later date. The data collection forms recorded the following information:

Demographics

The demographics recorded were National Health Index (NHI) number, date of birth, age at first admission, ethnicity, and gender. NHI number, date of birth, gender, and age at first admission were available in all of the clinical files read in the study. In two files, the ethnicity of the participant was inconsistent between the front page of the file and the information recorded in the clinical notes. In these cases, the ethnicity on the front page of the file was used in the statistical analysis.

Case information

For each child/adolescent, the number of admissions to the CAMHS was recorded. Some children/adolescents had multiple referrals to the service. For these children/adolescents, each

admission was a separate clinical record with its own front page, set of clinical notes, and separate assessment forms. For these children/adolescents, each separate admission record was read in its entirety and information from all admissions was recorded and included in the study as one participant.

Diagnostic information

Diagnosis was recorded if a formal diagnosis was stated on the front page of the file. Some participants had more than one formal diagnosis and all of these were recorded and included in the data analyses.

Symptomology

For each participant, information was recorded regarding specific symptomology relating to child and adolescent *DSM-IV* diagnoses, along with any other symptoms signifying problematic mental health. Symptoms were grouped into the symptoms of the main child and adolescent *DSM-IV* diagnostic categories. These symptom categories are defined in *Appendix C*.

Risk

An assessment RISK3 form was used in each participant's clinical record which was designed for recording risk to self and others. In the RISK3 form, 'risk to self' and 'risk to others' was rated as low, medium, or, high. In some clinical records there was more than one RISK3 form filled out. In other clinical records there were no RISK3 forms filled out. In the clinical records where the RISK3 was filled out, only the first recording of risk was recorded on the data collection sheet (this was generally assessed in the initial assessment interview). This risk rating was recorded directly onto the data collection sheet. In the files that did not have a RISK3 form filled out, a question mark was circled on the data collection form.

The RISK3 form also included the participant's history of suicide attempts and deliberate self-harm. If this information was available, it was recorded on the data collection form. Similarly to risk, a question mark was circled if this information was unavailable.

Aversive childhood events

This section of the data collection form included recorded disclosures of physical abuse, sexual abuse, emotional abuse, emotional neglect, physical neglect, chaotic family environment, grief/loss, parental discord, parental mental illness, impoverishment and 'other' significant adverse childhood events. The researcher's categorisation and recording of adverse childhood events was based on the definitions provided in *Appendix D.*

Procedure

Initial meetings, planning, and ethical approval

In order to obtain consent for the study, and as part of the ethics approval process, several meetings were held with the clinical director, researchers, and staff of the CAMHS. During these meetings the main aims of the study were discussed. In order to increase the usefulness and efficiency of the study, the clinical director and key staff members were asked if there was any information from the clinical files that would be helpful to record for the centre's own purposes. The only request was to include 'parental mental illnesses' in the adverse childhood events section of the data collection sheet. This was because there had been ongoing research at the CAMHS looking into the relationship between parents with mental health problems and the effects this has on their children. This suggestion was accepted and included in the study.

Due to there being no face to face contact with any of the participants involved, the study was considered an audit of mental health records focusing on specific issues. Ethics approval was obtained on this basis. The participants involved in the study were not informed that their files were

being audited for research purposes. This was to avoid any potential and unnecessary stress this may have caused the participants and to avoid jeopardising the relationship between the client and the CAMHS. Furthermore, the participant's data was entirely depersonalised in the data collection process. The principal researcher and her supervisor did see names and identifiable information while reading the participants files. In order to ensure the privacy of the participant's information, a confidentiality agreement with the CAMHS was signed by the principal researcher and her supervisor. The names of the clients were not recorded on the data collection sheets.

Once consent from the CAMHS was attained, along with ethical approval, a meeting was held with the staff of the service. The primary researcher attended this meeting with the clinical director of the service. The purpose was to introduce the main aims of the study, discuss what the researchers would be doing at the service during the data collection phase, and to hear any concerns the staff might have. One concern was raised regarding the privacy and confidentiality of the information of the client's that were to participate in the study. By law however, information contained within mental health files can be used for educational and research purposes, so long as the client's anonymity is maintained in this process.

Collection of data

Consultation with clinicians and researchers at the CAMHS began in June 2009. Therefore, the 217 files included in the study dated back from 1 June 2009 and no information from the files was included beyond this date. This was to eliminate any potential bias in the way clinical notes were recorded once clinicians had been made aware of the study.

An initial list of 507 consecutive admissions dating backwards from 1 June 2009 was obtained on the 12 December 2009. From this list, all of the admissions that were opened after April 2009 and were 'still open' were eliminated from the study. 'Still open' files included individuals who were still current clients of the CAMHS at 12 December 2009. These files were

eliminated due to the file being opened for a very short time period and the potential risk of important information of interest to the study not being added to file by that point in time or being added after June 2009. Information added to the file after June 2009 could not be included in the study because this is when staff at the CAMHS became aware of the study. Of the 507 files, 136 were 'open' (26.8%). The remaining files were 'closed', which meant those clients had been discharged from the service before 12 December 2009 (the date the list was produced).

The remaining 371 'closed' files were potential participants, with two exceptions. Firstly, clients that did not have any face to face contact with a clinician from the CAMHS during their admission were excluded from the study. There were two files excluded for this reason. Secondly, in the event of one of the participants in the study being recognised by one of the investigators, that participant was to be eliminated from the study. This was in order to protect the confidentiality and privacy of the individual. None of the participants in the study were eliminated for this reason.

The remaining 369 participants were presented in a list ordered by National Health Index (NHI) number. NHI numbers are unique numbers assigned to individuals in New Zealand to aid identification when using health and disability services. All New Zealand born children receive an NHI number at birth and approximately 95% of New Zealanders currently have an NHI number allocated (New Zealand Health Information Service, 2007). Individuals that do not have an NHI number for various reasons, e.g. being born outside of New Zealand, are allocated an NHI number when they use a health or disability service within the country. Therefore, the NHI ordered list was approximately distributed by age.

All of the data was collected by the principal investigator and her supervisor. Each clinical file was read in its entirety. On average, each clinical record took approximately 90 minutes to read and code, representing a total of 325.5 hours of data collection. Due to the time and resource constraints of a doctoral thesis, 217 of the total 369 clinical records were read and coded. The principal investigator read and coded the records of 207 participants, and her supervisor read and

coded the records of the remaining 10. In order to have an approximately even distribution of age, the NHI ordered list was split into 6 equal groups and an equal amount of files from each group were included in the study.

Early in the data collection phase, each of the investigators independently read the records of 10 participants. During this process the data collection form was altered to improve precision and efficiency of data collection. For example, the data collection form was changed from a two page sheet, to a more efficient and spaced-out three page sheet. Information was transferred word for word onto the data collection sheet to avoid subjective interpretations on behalf of the investigators, particularly relating to symptomology and adverse childhood events.

Due to the data being restricted to a review of electronic clinical records, what was considered a significant symptom or an adverse childhood event was based on what the clinician, client, parents, and/or referrer perceived as problematic, or what the clinician considered important enough to record in the file. In cases where the recording of the symptoms or adverse childhood events were vague, inconclusive, or questionable, a decision was made by both researchers based on the information available in the file and the definitions provided in *Appendix C and D* (this is outlined in detail in 'inter-rater reliability' later in the methods section).

Symptoms that did not fit the main categories listed in *Appendix C* were recorded in the data collection form as 'other significant symptoms'. Once the data was collected these symptoms were categorised based on relevant *DSM-IV* diagnoses. For example, symptoms of selective mutism were grouped into 'OTHER1' and symptoms of somatoform disorders were grouped into 'OTHER2' (*Appendix C*).

Data entry and inter-rater reliability

The data collected from the clinical files was entered into Statistical Package for the Social Sciences (SPSS). Diagnoses, symptoms, and adverse childhood events were coded as either 'yes or

no' depending on whether or not the participant had this information in their file. Whether or not a symptom or adverse childhood event was included in the study was based on the definitions in *Appendix C and D.*

In order to assess inter-rater reliability, the principal researcher and her supervisor independently entered the information from 20 data collection sheets into SPSS. An overall percentage of accuracy for symptom variables and adverse childhood events was obtained. This was assessed by calculating the total number of errors for the coding of both 'yes' and 'no' for all symptoms and adverse childhood events, and then calculating an overall percentage of accuracy between the two researchers. This process was repeated three times, until a satisfactory level of inter-rater reliability was achieved, as seen in Table 1. After the third inter-reliability test, the level of consistency at all levels was greater than 95%. This was deemed as a satisfactory level of inter-rater reliability. The remaining data collection sheets were entered into SPSS by the principal researcher.

Each time the inter-rater process was repeated, the main areas of error and inconsistency between the two researchers were identified. Based on these areas of discrepancy, the definitions and inclusion criteria outlined in *Appendix C and D* were clarified and altered accordingly. For example, after the first reliability check it was evident that some of the symptoms and/or adverse childhood events documented in the clinical files were unclear or vague. This made it difficult to establish whether or not the symptoms and/or adverse childhood events would be coded as 'yes' or 'no'. To help clarify this, the inclusion criteria were changed so that in these vague or unclear instances, at least two mentions of the symptom or adverse childhood event were required in order to be coded as 'yes'. Furthermore, there were some more specific changes made for individual variables. For example, the 'physical abuse' variable seemed to generate a high level of inconsistency between the two researchers. Therefore, a new inclusion criterion was added for cases of 'physical abuse' that were vague or unclear. In these instances, an additional descriptor

was required in order to be coded as 'yes'. For example, for an event to be included as 'physical abuse', an additional descriptor was required such as 'X was hit *hard* by his father' or 'X was slapped *frequently*'. This inclusion criterion was necessary to avoid minor misdemeanours such as a 'one off slap' being included as 'physical abuse'.

Table 1.

Results from the three inter-rater reliability checks.

| | Inter-rater reliability test | | |
	First	Second	Third
Percentage of variables coded consistently as 'yes' and 'no'			
Symptoms	93.9%	96.4%	97.0%
Adverse Childhood Events	93.3%	90.0%	93.5%

Data analysis

Analysis of data was quantitative. The data analyses are presented in three main sections. The first section looks at the sample characteristics and rates of diagnoses. Means, standard deviations, and frequencies were used to examine the sample characterises. Group differences were examined using independent sample t-tests (two-tailed) for continuous variables, and chi-square tests of independence for categorical variables. Because of the large number of analyses, results were considered statistically significant only if the *p-value* was equal or less than .025, so as to reduce the probability of Type 1 errors, i.e. false positives.

The second section looks at adverse childhood events. There were 13 categories of adverse events included in the data analysis (*Appendix D*). Means, standard deviations, and frequencies were used to look at the rates of childhood adverse events. T-tests were used to compare the

average number of adverse childhood events across gender and age groups. A one-way analysis of variance was used to examine whether rates of adverse childhood events varied by ethnic group. Chi-square tests of association were carried out to investigate gender differences in the specific types of individual adverse childhood events.

The third section examines the relationships between adverse childhood events, symptoms, and diagnoses. Symptoms were grouped into symptom clusters based on the corresponding DSM-IV diagnoses. For example, there were three ADHD variables: ADHD1 (inattentiveness), ADHD2 (hyperactivity), and ADHD3 (impulsivity). The ADHD cluster represents ADHD1 + ADHD2 + ADHD3. Therefore, if a participant had all of the ADHD symptoms this would give an ADHD cluster value of 3. The associations between the mean number of adverse childhood events and formal diagnoses, individual symptoms, and symptom clusters were looked at using t-tests. The associations between individual adverse childhood events and formal diagnoses, individual symptoms, and symptom clusters were analysed using chi-square tests of association.

The final section relates to the second aim of the study, and examines whether or not there is a group of children/adolescents with might be considered 'complicated ADHD'. Relevant literature suggests that children with 'complicated ADHD' have more serious externalising symptoms (i.e. a higher number of *DSM-IV* symptoms of the ADHD, CD and ODD disorders) (Briscoe- Smith & Hinshaw, 2006; Findling, 2007; Glod et al., 1996; King et al., 1998). Latent class analysis (LCA) was used to explore this aim. LCA is a statistical method used to identify homogeneous groups, or classes, from categorical multivariate data. Because the focus of this part of the study was externalising symptoms LCA was employed to examine the clustering of the symptoms of ADHD: inattentiveness (ADHD1), hyperactivity (ADHD2), and impulsivity (ADHD3), the symptoms of conduct disorder: aggression to people and animals (CD1), destruction of property (CD2), deceitfulness of theft (CD3), serious violation of rules (CD4), and the symptoms of ODD: non-compliant and defiant (ODD1) and oppositional (ODD2). Latent class analysis grouped participants

based on their relative probability of endorsing symptoms of externalising behaviour problems (ADHD, ODD, and CD). This produced five separate classes of participants with similar symptom profiles. The association between latent class and mean number of adverse childhood events was examined using t-tests. Chi-square tests of association were used to examine the relationships between latent class and individual adverse events, gender, diagnostic co-morbidity, risk to self and others, and rates of deliberate self-harm and previous suicide attempts.

CHAPTER III: RESULTS

Sample characteristics and demographics

Table 2.

Demographic information by gender.

		Gender of Participant		
		Female	Male	Total
Gender total		113 (52.1%)	104 (47.9%)	217 (*N*)
Ethnicity	European	69 (31.8%)	58 (26.7%)	127 (58.5%)
	Maori	19 (8.7%)	15 (6.9%)	34 (15.7%)
	Pacific Island	9 (4.2%)	10 (4.6%)	19 (8.8%)
	Asian	12 (5.6%)	13 (5.9%)	25 (11.5%)
	Other	4 (1.8%)	8 (3.7%)	12 (5.5%)
Age at fist contact	*M*	13.79 years**	10.96 years	12.39 years
	SD	3.48	4.29	4.18
	Under 13 years	35 (16.1%)	64 (29.5%)	99 (45.6%)
	13 years and over	78 (35.9%)	40 (18.4%)	118 (54.4%)
Number of admissions to service	One admission	88 (40.6%)	79 (36.4%)	167 (77.0%)
	Two admission	19 (8.8%)	16 (7.4%)	35 (16.1%)
	Three or more	6 (2.8%)	9 (4.1%)	15 (6.9%)
	M	1.23	1.36	1.32
	SD	0.64	0.74	0.69
Formal Diagnosis	Yes	93 (42.9%)	81 (37.3%)	174 (80.2%)
	No diagnosis/Unknown	20 (9.2%)	23 (10.6%)	43 (19.8%)
Co-morbidity	None/one diagnosis	72 (33.2%)	73 (33.6%)	145 (66.8%)
	Two diagnoses	28 (12.9%)	18 (8.3%)	46 (21.2%)
	Three or more diagnoses	13 (6.0%)	13 (5.9%)	26 (11.9%)

** *p* < .01 (two tailed t-test)

As seen in Table 1, there were 217 participants in the study. Of these, 113 (52.1%) were female, 104 (47.9%) were male. The participants ranged in age from three to 18 years. The average age at first contact with the service was 12.39 years ($SD = 4.18$). Just over half (54.3%) of the participants were 13 years or older. There was a significant difference between the average age at first contact of the male ($M = 10.96$ years, $SD = 4.29$) and female participants ($M = 13.79$ years, $SD = 3.48$), $t(214) = 5.34$, $p = .00$).

One hundred and twenty seven (58.5%) of the participants were of European ethnicity, 34 (15.7%) were Maori, 19 (8.8%) were Pacific Island, 25 (11.5%) were Asian, and 12 (5.5%) were of 'other' descent. 'Other' included African, Latin American/Hispanic, Iraqi, Fijian Indian, Australian/Denmark/USA, and 'other'.

Number of admission and service stream

One hundred and sixty seven (77.0%) participants had one admission to the service, 35 (16.1%) had two admissions, and 15 (6.9%) had three or more admissions. The maximum number of admissions was five with an average of 1.32 ($SD = 0.69$) admissions per participant.

The CAMHS service has a number of teams providing care. These include four community teams (Pohutakawa, Koru, Kowhai, and Ngakau), and early intervention team, a liaison service and a forensic team. Thirty nine (18%) of the participants were streamed into the Kowhai team, 66 (30.4%) were in the Pohutakawa team, 53 (24.4%) were in Koru, 7 (3.2%) were in the early intervention team, 47 (21.7%) were in Ngakau, and five (2.3%) did not have this information available in their file.

Diagnosis and co-morbidity

As seen in Table 1, 174 (80.2%) of the participants were given at least one formal diagnosis

on the front page of their file. Forty six (21.2%) of the participants had two formal diagnoses and 26 (11.9%) participants had three or more formal diagnoses.

For formal diagnoses where less than five participants were allocated that particular diagnosis, this was included in the analysis as 'other diagnosis'. As seen in Figure 1, the number of participants that had each formal diagnosis was: 28 (12.9%) 'other diagnosis', 27 (12.4%) depression – NOS, 25 (11.5%) ADHD, 22 (10.1%) adjustment disorder, 21 (9.7%) relationship problems, 21 (9.7%) anxiety disorder – NOS, 13 (7.4%) major depressive disorder, 13 (6%) aspergers disorder, 13 (6%) family dynamic issues, 12 (5.5%) generalized anxiety disorder, ten (4.6%) dysthymia, nine (4.1%) PTSD, eight (3.7%) social anxiety disorder, seven (3.2%) obsessive compulsive disorder, six (2.8%) panic disorder, six (2.8%) learning disorder, six (2.8%) conduct disorder, five (2.3%) separation anxiety disorder, five (2.3%) oppositional defiant disorder, five (2.3%) substance abuse/dependence, and five (2.3%) selective mutism.
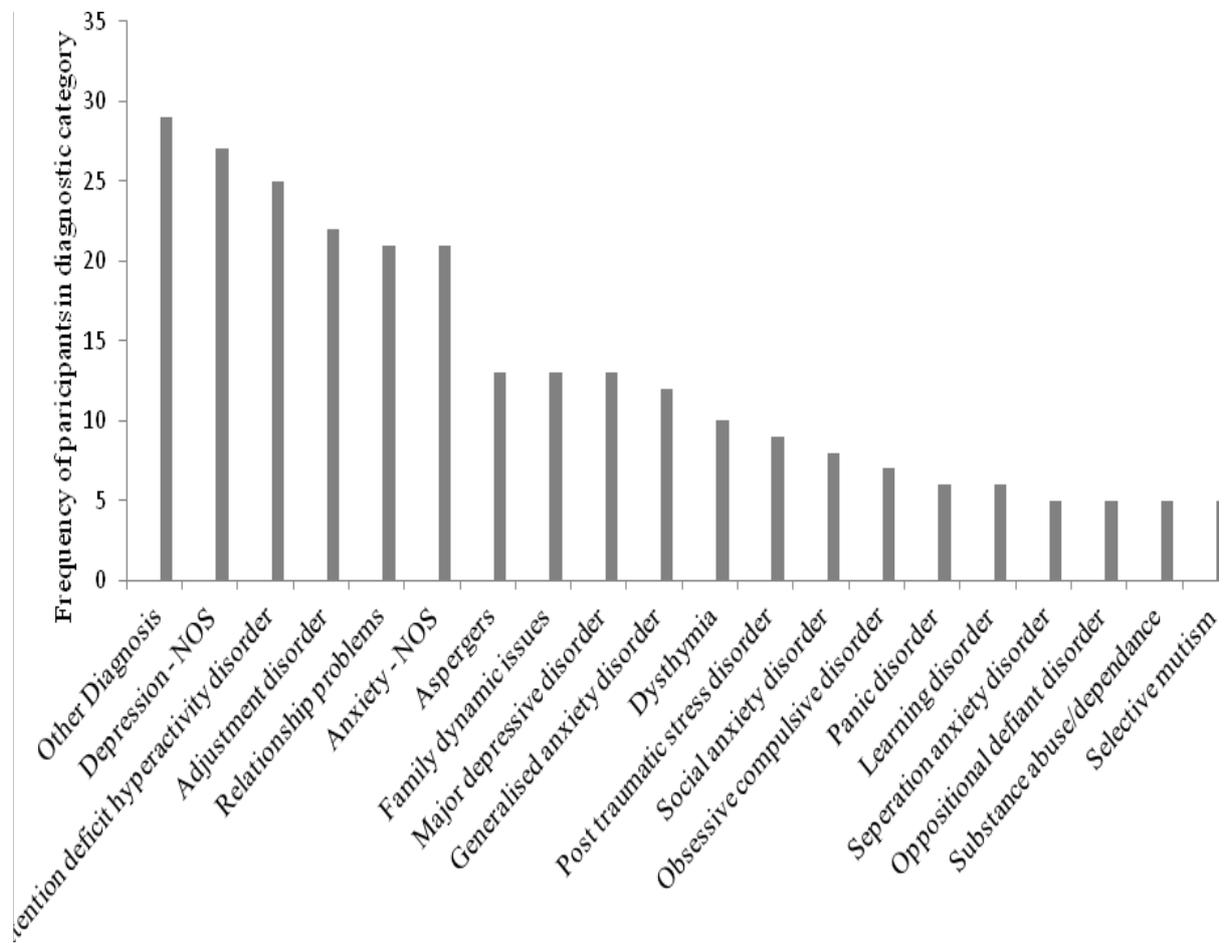
Figure 1.

Number of participants in each formal diagnostic category.

Age, gender, and ethnic group differences in formal diagnosis and co-morbidity

As seen in Table 3, Ninety three (82.3%) of the female participants and 81 (78.6%) of male participants were allocated a formal diagnosis. Forty one (36.3%) of the females and thirty one (29.8%) of the male participants had two or more co-morbid diagnoses. There were no significant differences between males and females in the number allocated a formal diagnosis x^2 (2, $N = 216$) = 1.72, $p = .420$, or rates of diagnostic co-morbidity x^2 (1, $N = 216$) = .436, $p = .610$.

Within the European participants, 111 (87%) were allocated a formal diagnosis and 48 (37.8%) had two or more formal diagnoses. Twenty seven (79.4%) Maori participants were allocated a formal diagnosis and 11 (32.4%) had at least two formal diagnoses. Ten (52.6%) Pacific Islanders were allocated a formal diagnosis and five (26.4%) had two or more formal diagnoses. Eighteen (72%) of Asian participants were allocated a formal diagnosis and five (20%) had at least two formal diagnoses. Chi square analyses revealed a significant association between ethnicity and the number of participants allocated a formal diagnosis x^2 (4, $N = 216$) = 16.15, $p = .003$. However, diagnostic co-morbidity rates did not significantly differ between the ethnicity groups x^2 (8, $N = 216$) = 5.32, $p = .720$.

When considering age groups, 96 (81.4%) of participants aged 13 years and over were allocated at least one formal diagnosis and 40 (33.9%) had two or more diagnoses. Of the participants under the age of 13 years, 78 (78.8%) were allocated a diagnosis with 32 (32.3%) having two or more diagnoses. There were no significant differences among young and old age groups in the number allocated a formal diagnosis x^2 (1, $N = 217$) = .22, $p = .730$, or rates of diagnostic co-morbidity x^2 (2, $N = 217$) = .11, $p = .950$.

Gender differences in specific diagnosis

Chi-square tests of association were carried out to investigate the relationship between gender and specific formal diagnoses. Females had higher rates of major depressive disorder

x^2 (1, $N = 216$) = 5.8, $p = .0019$, depression –NOS x^2 (1, $N = 216$) = 8.02, $p = .007$, and

relationship problems x^2 (1, $N = 216$) = 7.6, $p = .006$. Males had higher rates of ADHD x^2 (1,

$N = 216$) = 14.94, $p = .000$, and aspergers syndrome x^2 (1, $N = 216$) = 15.20, $p = .000$.

Table 3.

Age, gender, and ethnic group differences in formal diagnosis and co-morbidity

		Formal Diagnosis		Co-morbidity		
		Yes	No	None/one diagnosis	Two or more diagnoses	Total (*N*)
Gender	Female	93 (84%)	20 (16%)	72 (63.7%)	41 (36.3%)	113
	Male	81 (78.6%)	23 (21.4%)	73 (70.2%)	72 (29.8%)	104
Ethnicity	European	111 (87.0%)**	16 (13%)	79 (62.2%)	48 (37.8%)	127
	Maori	27 (79.4%)	7 (20.6%)	23 (67.6%)	11 (32.4%)	34
	Pacific Island	10 (52.6%)	9 (47.4%)	14 (6.5%)	5 (2.3%)	19
	Asian	18 (72.0%)	7 (28.0%)	20 (80.0%)	5 (20.0%)	25
	Other	8 (66.7%)	4 (33.3%)	9 (75.0%)	3 (25.0%)	12
Age at first contact	Under 13 years	78 (78.8%)	21 (21.2%)	67 (67.7%)	32 (32.3%)	99
	13 years and over	96 (81.4%)	22 (18.6%)	78 (67.1%)	40 (33.9%)	118
	Total	174	43	145	72	217

** $p < .01$ (x^2 **coefficient**)

Risk assessment, deliberate self-harm, and previous suicide attempts.

Risk to self and risk to others was recorded in the participants RISK3 form. As seen in Table 4, 145 (66.8%) of the participants were assessed on their 'risk to self'. Sixteen (7.4%) participants were rated as 'high' risk to self, 32 (14.7%) were rated as 'medium' risk to self, and 97 (44.7%) were rated as 'low' risk to self. One hundred and forty three (65.9%) of the participants were assessed on their risk to others. Five (2.3%) were rated as 'high' risk to others, 16 (7.4%) were rated as 'medium' risk, and 122 (56.2%) were rated as 'low' risk.

Rates of deliberate self-harm and previous suicide attempts were documented in the participants RISK3 form. Eighty (36.9%) of the participants were reported to have engaged in deliberate self-harm and 137 (63.1%) did not engage in deliberate self-harm or did not have this information included in their RISK3 form or clinical record. Fourteen (6.5%) had multiple previous suicide attempts and 35 (16.1%) had one previous suicide attempt. One hundred and sixty eight (77.4%) of the participants had no known suicide attempt or did not have this information included in their RISK3 form or clinical file. Females were more likely to engage in deliberate self-harm than male participants, χ^2 (2, $N = 216$) = 43.9, $p = .000$. Females were more likely to have a previous suicide attempt than male participants, χ^2 (2, $N = 216$) = 9.7, $p = .008$.

Table 4.

Assessment of risk, deliberate self-harm, and previous suicide attempts by age and gender groups

| | | Gender | | |
		Female	Male	Total (*N*)
Total		113	104	217
Risk to self	High	11 (5.1%)	5 (2.3%)	16 (7.4%)
	Medium	22 (10.1%)	10 (4.6%)	32 (14.7%)
	Low	45 (20.7%)	52 (24.0%)	97 (44.7%)
	Not assessed	35 (16.1%)	37 (17.1%)	72 (33.2%)
Risk to others	High	2 (0.9%)	3 (1.4%)	5 (2.3%)
	Medium	9 (4.2%)	7 (3.2%)	16 (7.4%)
	Low	67 (30.9%)	55 (25.3%)	122 (56.2%)
	Not assessed	35 (16.1%)	39 (18.0%)	74 (34.1%)
Engaged deliberate self-harm	Yes	65 (30.0)***	15 (6.9%)	80 (36.9%)
	No/Unknown	48 (22.1)	89 (41.0%)	137 (63.1%)
Previous suicide attempt	Yes	26 (12.0%)**	9 (4.2%)	35 (16.1%)
	Multiple	9 (4.2%)**	5 (2.3%)	14 (6.5%)
	No/Unknown	78 (35.9%)	90 (41.5%)	168 (77.4%)

** $p < 0.01$ *** $p < 0.001$ (x^2 **coefficient**)

Adverse childhood events

Table 5.

Total number of adverse events by gender, ethnicity, and age groups.

		Average number of adverse events		
		M	*SD*	Total
Total		3.30	2.30	217 (*N)*
Gender	Female	3.57	2.24	113 (52.1%)
	Male	3.00	2.34	104 (47.9%)
Ethnicity	European	3.14	2.21	127 (58.5%)
	Maori	4.65**	2.67	34 (15.7%)
	Pacific Island	3.00	1.94	19 (8.8%)
	Asian	2.60	1.86	25 (11.5%)
	Other	3.09	2.34	12 (5.5%)
Age at first contact	Under 13 years	3.01	2.32	99 (45.6%)
	13 years and over	3.52	2.27	118 (54.4%)

** $p < .01$ (one-way analysis of variance)

There were categories of adverse childhood events included in the study (*Appendix D*). As seen in Table 5, the average number of reported adverse events per participant was 3.30 (*SD* = 2.30). As seen in Figure 2, twenty five (11.5%) of the participants had no adverse childhood events recorded in their file. Twenty one (9.7%) had one adverse event, 44 (20.3) had two adverse events, 36 (16.6%) had three adverse events, 35 (16.1%) had four adverse events, 20 (9.2%) had five adverse events, 15 (6.9%) had six adverse events, 14 (6.5%) had seven adverse events and seven (3.2%) of the participants had eight or more adverse events recorded in their file.

As seen in Figure 3, the most common adverse event was parental mental health problems, with 90 (41.5%) of the participants having this event noted in their file. The rates of other adverse

events were: 77(35.5%) divorce, 75 (34.6%) emotional/psychological abuse, 71 (32.7%) bullying, 63 (29%) physical abuse, 47 (21.7%) parent discord, 40 (18.4%) substance abuse in family, 33 (15.5%) neglect, 30 (13.8%) "other" adverse event, 30 (13.8%) sexual abuse, 27 (12.4%) domestic violence, 19 (8.8%) serious physical illness disability, 14 (6.5%) family illness or disability, and 16 (6%) impoverishment (Figure 2).

Mean number of adverse events across gender, ethnicity, and age groups

As seen in Table 3, females had a marginally higher rate of adverse events ($M = 3.57$, $SD = 2.24$) in comparison to males but this was not statistically significant ($M = 3.00$, $SD = 2.34$), $t(214) = 1.82$, $p = .070$. Participants over the age of 13 years reported an average of 3.52 ($SD = 2.27$) adverse events and participants under the age of 13 years an average of 3.01 ($SD = 2.32$), $t(215) = 1.62$, $p = .110$.

Maori participants had an average of 4.65 ($SE = 2.67$) adverse events, European participants 3.14 ($SD = 2.21$), Pacific Islander participants 3.0 ($SD = 1.94$), Asian participants 2.60 ($SE = 1.86$), and 'other' 3.0 ($SD = 2.34$). A one-way analysis of variance was used to test for the effect of ethnicity. Maori participants on average, reported a higher rate of adverse events in comparison to other ethnic groups, $F(4, 211) = 3.97$, $p = .004$.

Gender Differences between types of adverse events

Chi-square tests of association were carried out to investigate the relationship between gender and type of adverse events. Females reported a higher rate of sexual abuse x^2 (1, $N = 216$) = 8.3, $p = .005$, and emotional/psychological abuse x^2 (1, $N = 216$) = 7.8, $p = .006$. There were no significant gender differences in the rates of other types of adverse childhood events.
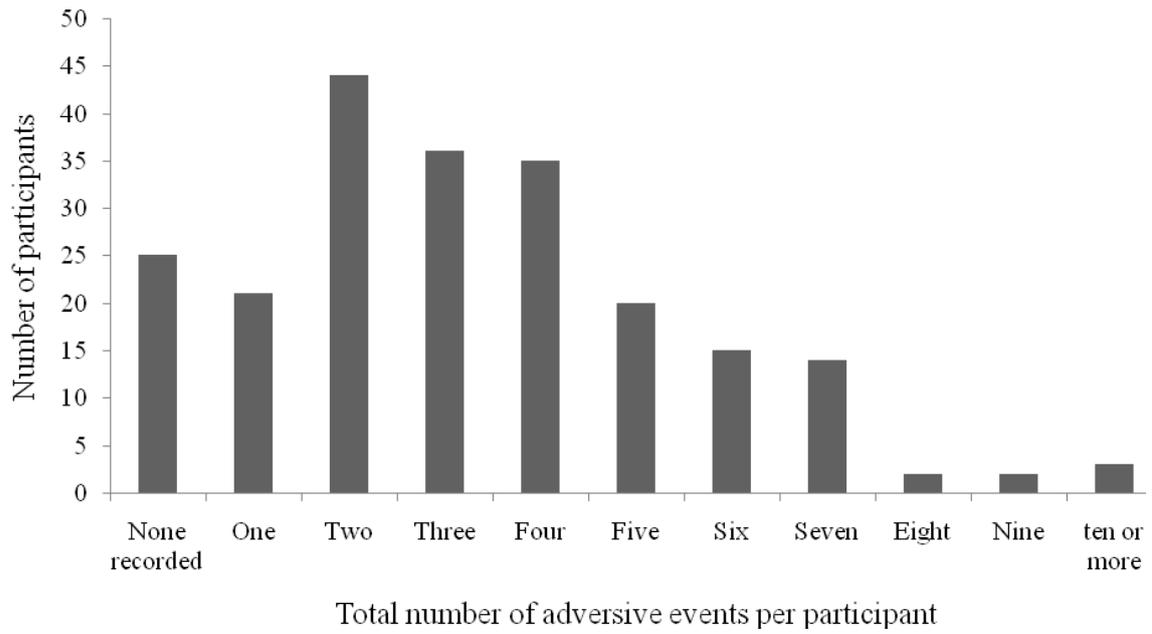
Figure 2.

Total number of adverse events per participant.
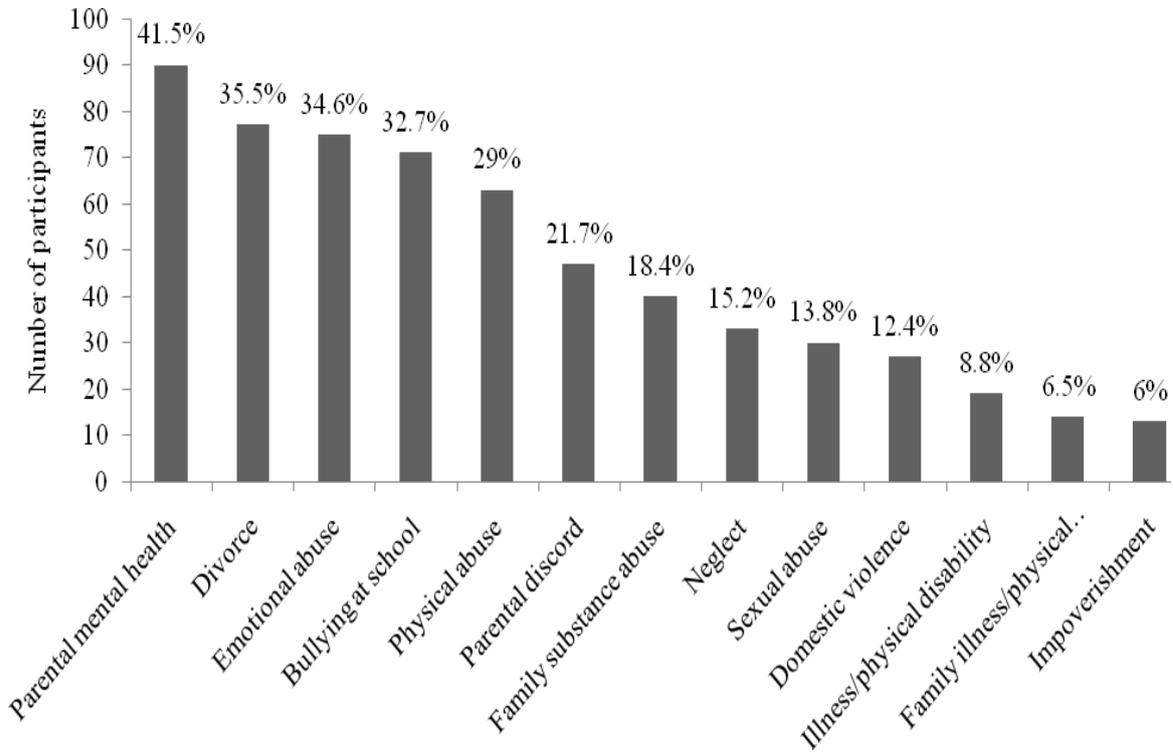
Figure 3.

Rates of individual adverse events recorded in participant files.

Relationship between adverse events, symptoms, and diagnosis

Relationship between diagnoses and adverse events

T-tests were used to examine the associations between formal diagnoses and the mean number of adverse events. There were no significant differences in the mean number of adverse events of those with an ADHD diagnosis (*M* = 3.24, *SE* =2.83) and those without an ADHD diagnosis (*M* = 3.29, *SE* =2.2), *t*(215) = 0.11, *p* =.920. Participants with a diagnosis of ODD had a significantly higher average rate of adverse events (*M* = 6.40, *SE* =3.65) than participants without a diagnosis of ODD (*M* = 3.21, *SE* =2.22), *t*(215) = 3.12, *p* =.002. There were no significant differences in the mean number of adverse events of those with a CD diagnosis (*M* = 5.17, *SE* =4.40) and those without a diagnosis of CD (*M* = 3.23, *SE* =2.21) *t*(215) = 2.05, *p* =.042. There was also no significant difference in the mean number of adverse events of those with a diagnosis of PTSD (*M* = 4.89, *SE* =1.97) and those without a diagnosis of PTSD (*M* = 3.22, *SE* =2.29), *t*(215) = 2.15, *p* =.032.

Chi-square tests of association were carried out to investigate the relationships of individual diagnoses (ADHD, ODD, CD and PTSD) and specific adverse events (see *Appendix E*). A fisher's exact test was used when cell counts had an expected frequency less than 5. In terms of specific adverse events, a diagnosis of ADHD was not significantly associated with any individual adverse events. A diagnosis of conduct disorder was not significantly associated with any individual adverse events. A diagnosis of ODD was associated with divorce x^2 (1, *N* = 217) = 9.31, *p* = .005 and domestic violence x^2 (1, *N* = 217) = 10.63, *p* = .015. A diagnosis of PTSD was associated with sexual abuse x^2 (1, *N* = 217) = 32.23, *p* = .000 and physical abuse x^2 (1, *N* = 217) = 6.46, *p* = .019.

Relationship between symptom clusters and adverse events

Chi-square tests of independence were carried out to investigate the relationships of

symptom clusters to total number of adverse events and specific types of adverse events (see *Appendix F*).

The total number of adverse events was significantly related to the ADHD cluster x^2 (36, $N = 217$) = 66.42, $p = .001$, the CD cluster x^2 (48, $N = 217$) = 113.40, $p = .000$, the PTSD cluster x^2 (36, $N = 217$) = 54.63, $p = .024$, but not to the ODD cluster.

In terms of specific types of adverse events, the ADHD cluster was significantly associated with physical abuse x^2 (3, $N = 217$) = 9.58, $p = .022$. The CD cluster was associated with physical abuse x^2 (4, $N = 217$) = 12.19, $p = .016$, sexual abuse x^2 (4, $N = 217$) = 12.94, $p = .012$, neglect x^2 (4, $N = 217$) = 11.67, $p = .020$, and domestic violence x^2 (4, $N = 217$) = 16.86, $p = .002$. The ODD cluster was associated with physical abuse x^2 (2, $N = 217$) = 12.49, $p = .002$ and neglect x^2 (2, $N = 217$) = 9.44, $p = .009$. The PTSD cluster was associated with sexual abuse x^2 (3, $N = 217$) = 28.39, $p = .000$ and loss x^2 (4, $N = 217$) = 17.56, $p = .001$.

Relationship between specific symptoms and adverse events

T-tests were used to examine the associations between specific symptoms and the mean number of adverse events. Participants with Hyperactivity (ADHD2) had a significantly higher average rate of adverse events ($M = 4.19$, $SE = 3.2$) than those without ADHD2 ($M = 3.10$, $SE = 2.03$), $t(215) = 2.66$, $p = .008$. Participants with Deceitfulness and/or theft (CD3) had a significantly higher average rate of adverse events ($M = 4.33$, $SE = 2.80$) than those without CD3 ($M = 3.10$, $SE = 2.16$), $t(215) = 2.89$, $p = .004$. Participants with Serious violation of rules (CD4) had a significantly higher average rate of adverse events ($M = 4.44$, $SE = 2.93$) than those without CD4 ($M = 3.06$, $SE = 2.09$), $t(215) = 3.39$, $p = .001$. Participants with Increased arousal (PTSD3) had a significantly higher average rate of adverse events ($M = 4.91$, $SE = 3.21$) than those without PTSD3 ($M = 3.20$, $SE = 2.22$), $t(215) = 2.43$, $p = .016$. There were no significant associations between the mean number of adverse events and Inattentiveness (ADHD1), Impulsivity (ADHD3), Aggression

to people and animals (CD1), Destruction of property (CD2), Non-compliance (ODD1),

Oppositional behaviour (ODD2), Re-experiencing of traumatic events (PTSD1) and Avoidance and

numbing (PTSD2).

Chi-square tests of association were carried out to investigate the relationships between

specific symptoms, total number of adverse events, and individual adverse events (see *Appendix G*).

The total number of adverse events was significantly associated with the following symptoms:

Hyperactivity (ADHD2) x^2 (12, $N = 217$) = 31.81, $p = .001$, Serious violation of rules (CD4)

x^2 (12, $N = 217$) = 25.10, $p = .012$ and Increased arousal (PTSD3) x^2 (12, $N = 217$) = 25.78, $p =$

.012.

In terms of specific types of adverse events, Inattentiveness (ADHD1) was not significantly

associated with any individual adverse events. Hyperactivity (ADHD2) was associated with

physical abuse x^2 (1, $N = 217$) = 6.19, $p = .017$, parent substance use x^2 (1, $N = 217$) = 5.81, $p =$

.02, and domestic violence x^2 (1, $N = 217$) = 12.24, $p = .002$. Impulsivity (ADHD3) was

associated with physical abuse x^2 (1, $N = 217$) = 7.21, $p = .017$.

Aggression to people and animals (CD1) was associated with physical abuse x^2 (1, $N =$

217) = 5.71, $p = .020$, domestic violence x^2 (1, $N = 217$) = 7.61, $p = .000$, impoverishment

x^2 (1, $N = 217$) = 6.88, $p = .014$, and family illness and/or disability x^2 (1, $N = 217$) = 5.25, $p =$

.021. Destruction of property (CD2) was associated with neglect x^2 (1, $N = 217$) = 6.50, $p = .022$.

Deceitfulness and/or theft (CD3) was not significantly associated with any individual adverse

events. Serious violation of rules (CD4) was associated with physical abuse x^2 (1, $N = 217$) =

9.21, $p = .004$, domestic violence x^2 (1, $N = 217$) = 6.25, $p = .023$, and parent substance use

x^2 (1, $N = 217$) = 15.50, $p = .000$.

Non-compliance (ODD1) was not significantly associated with any of the individual

adverse events. Oppositional behaviour (ODD2) was associated with physical abuse x^2 (1, $N =$

217) = 5.92, $p = .021$.

Re-experiencing of traumatic events (PTSD1) was associated with sexual abuse χ^2 (1, $N =$ 217) = 17.10, $p = .001$. Avoidance and numbing (PTSD2) was associated with loss χ^2 (1, $N =$ 217) = 13.31, $p = .000$, and parent mental health problems χ^2 (1, $N = 217$) = 8.21, $p = .003$. Increased arousal (PTSD3) was associated with sexual abuse χ^2 (1, $N = 217$) = 33.75, $p = .000$ and loss χ^2 (1, $N = 217$) = 5.65, $p = .025$.

Latent Class analysis among ADHD, CD and ODD symptoms

Latent class analysis (LCA) was carried out in order to investigate an observed association among the categorical symptom variables. LCA grouped participants based on their relative probability of endorsing a particular set of symptoms. Because the focus of this part of the study was externalising symptoms, LCA was employed to examine the clustering of the symptoms of: ADHD, ODD, and CD. The fit of six models (one-class latent class model through to a six-class model) was assessed.

Selection of the optimal number of latent classes was based on several statistical fit indices. The statistical fit indices were: likelihood ratio chi-square (LRχ^2), Akaike information criterion (AIC; Akaike, 1987), Bayesian information criterion (BIC; Schwartz, 1978), sample-size adjusted BIC (SSABIC; Sclove, 1987), the Lo-Mendell-Rubin's adjusted likelihood ratio test (LRT; Lo, Mendell, & Rubin, 2001), and entropy measures (Ramaswamy, DeSarbo, Reibstein, & Robinson, 1993). A non-significant likelihood ratio chi-square indicates acceptable model fit. The information statistics AIC, BIC, and SSABIC are goodness-of-fit measures used to compare competing models; lower observed values indicate better fit. The Lo-Mendell-Rubin's LRT (2001) statistic was used to compare models with differing numbers of latent classes; a non-significant value ($p>.05$) suggests that the model with one less class should be accepted. Entropy (Ramaswamy et al., 1993) is a standardised measure of how accurately participants are classified. Entropy values can range from 0

to 1 with higher values indicating better classification. The LCA analysis was conducted using

Mplus 3.12 (Muthén & Muthén, 2005).

Table 6 shows the fit statistics from the latent class analysis. The interpretation of these

complex analyses is deferred until the Discussion. The findings only will be reported here.

	Loglikelihood	AIC	BIC	ssBIC	LRT	Entropy
1 class	-950.866	1919.732	1950.151	1921.631		
2 classes	-857.175	1752.350	1816.568	1756.359	187.383 (.000)	0.763
3 classes	-834.375	1726.750	1824.767	1732.870	45.599 (.0000)	0.851
4 classes	-818.360	1714.721	1846.537	1722.950	32.030 (.000)	0.817
5 classes	-805.848	1709.695	1875.310	1720.035	25.025 (.0300)	0.843
6 classes	-796.620	1711.241	1910.654	1723.691	18.455(.2000)	0.848

Table 6.

Fit Statistics from the Latent Class Analysis

The 5-class model was considered to be the best model. Although the BIC was lowest for

the 4-class solution the AIC and the ssBIC were the lowest for the 5-class solution, and the LRT

indicated that the addition of the sixth class did not significantly improve the fit of the model.

Figure 4 shows the profile plot of the 5-class solution with the probabilities for all of the ADHD,

ODD, and CD symptoms on the y-axis. Class 5 (47.5% of participants) was referred to as the

"baseline class". The baseline class was low in all ADHD, CD and ODD symptom endorsement.

Class 4 (24.9% of participants) was referred to as "high ODD". Class 4 included participants with

high endorsement of CD1 (0.4), ODD1 (0.35), and ODD2 (0.75). Class 3 (9.2% of participants)

was referred to as "High ADHD and ODD" and grouped participants with high probabilities of endorsement of ADHD1 (0.6), ADHD2 (1.0), ADHD3 (0.35), CD1 (0.55), ODD1 (0.4) and ODD2 (0.8). Class 2 (14.3% of participants) was referred to as "High ODD and CD". Class 2 had high probabilities of endorsements for CD1 (0.9), CD 2 (0.4), CD3 (0.5), ODD1 (0.4) and ODD2 (0.45). Class 1 (4.2% of participants) which is referred to as "'high all'class" had very high probabilities of endorsements for ADHD1 (0.78), ADHD2 (1.0), ADHD3 (0.65), CD1 (1.0), CD2 (0.85), CD3 (0.75), CD4 (0.65), and ODD2 (1.0).
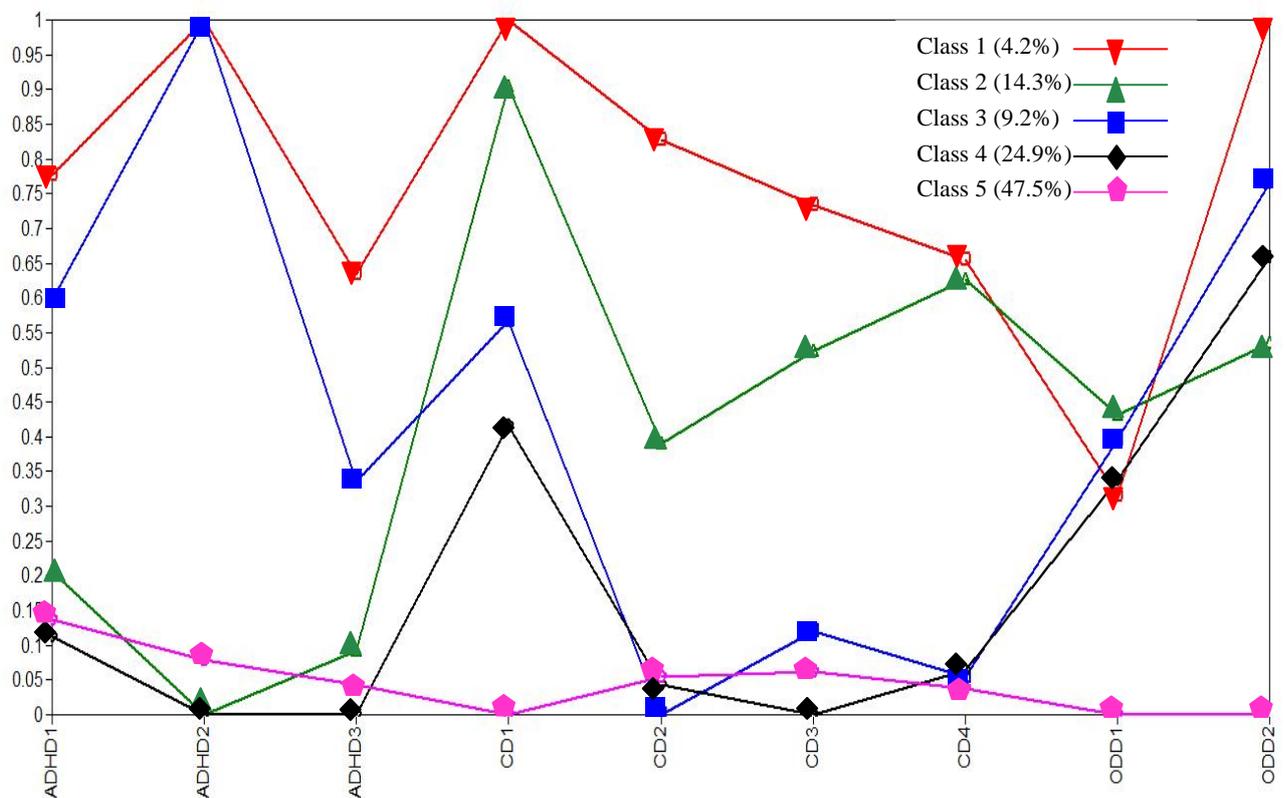
Figure 4.

5-class model of expected symptom endorsement for ADHD, CD and ODD symptoms.

Table 7.

Mean number of adverse events and rates of individual adverse events per latent class.

	Class					
	1. High all	2. CD and ODD	3. ADHD and ODD	4. ODD	5. Baseline	Total
Total	9	31	20	54	103	217
Mean no# of total adverse events.	6.22 ┼┼┼	3.29	3.70	3.41	2.88	3.29
Standard Deviation	3.93	1.88	2.89	2.19	1.99	2.30
Physical abuse	7 (77.8%)**	10 (32.3%)	8 (40%)	18 (33.3%)	20 (19.4%)*	63 (29%)
Sexual abuse	2 (22.2%)	6 (19.4%)	3 (15%)	5 (9.3%)	14 (13.6%)	30 (13.8%)
Neglect	4 (44.4%)	5 (16.1%)	3 (15.0%)	13 (24.0%)	8 (7.8%)	33 (15.2%)
Emotional abuse	5 (55.6%)	9 (29.0%)	6 (30.0%)	19 (35.2%)	36 (35.0%)	75 (34.6%)
Divorce	5 (55.6%)	11 (35.5%)	5 (25.0%)	22 (40.7%)	34 (33.0%)	77 (35.5%)
Loss	5 (55.6%)	11 (35.5%)	12 (60.0%)	22 (40.7%)	34 (33.0%)	84 (38.7%)
Parental mental health problem	5 (55.6%)	9 (29.0%)	8 (40.0%)	25 (46.3%)	43 (41.7%)	90 (41.5%)
Domestic violence	4 (44.4%)*	5 (16.1%)	6 (30.0%)*	5 (9.3%)	7 (6.8%)***	27 (12.4%)
Parental discord	3 (33.3%)	8 (25.8%)	5 (25.0%)	10 (18.5%)	21 (20.4%)	47 (21.7%)
Parental substance abuse	4 (44.4%)	7 (22.6%)	5 (25.0%)	8 (14.8%)	16 (5.5%)	40 (18.4%)
Impoverishment	2 (22.2%)	3 (9.7%)	1 (5.0%)	4 (7.4%)	3 (2.9%)	13 (6.0%)
Bullying	5 (55.6%)	9 (29.0%)	8 (40.0%)	18 (33.3%)	31 (30.1%)	71 (32.7%)

* $p < .025$ ** $p < .01$ *** $p < .001$ (x^2 **coefficients**)   ┼┼┼ $p < .0001$ (2-tailed t-test)

Total number of adverse events per latent class

T-tests were used to examine differences between latent classes, in terms of the mean number of adverse events. As seen in Table 7, the 'high all' class had an average of 6.22 (*SE* = 3.93) adverse events. Class 1 'high all' was the only class that had a statistically significant higher rate of adverse events (*M* = 6.22, *SE* = 3.93), than the average rate of adverse events among participants not in that class (*M* = 3.16, *SE* = 2.13, *p*=.0001). Class 1 'high all' had a significantly higher average than class 2 'CD and ODD' (*M* = 3.29, *SE* = 1.88, *p*=.01), class 4 'ODD' (*M* = 3.41, *SE* = 2.19, *p*=.01), and class 5 'baseline' (*M* = 2.88, *SE* =1.99, *p*=.001). Class1 'high all' had a marginally higher average of adverse events than class 3 'ADHD and ODD' (M = 3.70, *SE* = 2.89, *p*= 0.062), however this was not statistically significant.

Individual adverse events by class

Chi square tests of association were carried out to investigate the relationship between individual adverse events and class. A fisher's exact test was used when cell counts had an expected frequency less than 5.

Class 1 'high all' was significantly associated with higher levels of physical abuse x^2 (1, N = 217) = 10.82, p = .003 and domestic violence x^2 (1, N = 217) = 8.83, p = .016, and total number of adverse events x^2 (12, N = 217) = 61.59, p = .000. Class 2 'CD and ODD' was not significantly associated with any of the individual adverse events. Class 3 'ADHD and ODD' was significantly associated with and domestic violence x^2 (1, N = 217) = 6.23, p = .024. Class 4 'ODD' was not significantly associated with any of the individual adverse events. Class 5 'baseline' was significantly associated with lower rates of physical abuse x^2 (1, N = 217) = 8.80, p = .002 and lower rates of domestic violence x^2 (1, N = 217) = 5.74, p = .013.

Gender differences between latent classes

As seen in Table 8, chi square analyses were carried out to examine the association between latent class and gender. Class 1 'high all' had significantly more males than females x^2 (1, $N =$ 216) = 6.39, p = .012. Class 5 'baseline', Class 2 "CD and ODD", Class 3 "ADHD and ODD", and Class 4 "ODD" did not have any significant gender differences.

Differences in diagnostic co-morbidity between latent classes

Chi square analyses were carried out to examine the association between each latent class and diagnostic co-morbidity (see Table 8). Class 1 'high all' x^2 (2, N = 217) = 9.44, p = .009 was significantly associated with higher rates of diagnostic co-morbidity. There were no significant associations between diagnostic co-morbidity and class 2 "CD and ODD", class 3 'ODD and CD', Class 4 "ODD", or Class 5 "baseline".

Differences in risk to self and others, self-harm and suicide attempts between latent classes

Chi square analyses were carried out to examine the association between risk to self, risk to others, rates of self-harm, and previous suicide attempts (see table 8). There were no significant associations between latent class and risk to self. Class 1 "high all" x^2 (3, N = 217) = 9.42, p = .024, and Class 2 "ODD and OD" x^2 (3, N = 217) = 17.61, p = .001, were significantly associated with a higher risk to others. There were no significant associations between latent class and rates of self-harm or previous suicide attempts

Table 8.

Gender, diagnostic co-morbidity, and risk differences between latent classes.

		Class					
		1. High	2. CD & ODD	3. ADHD & ODD	4. ODD	5. Baseline	Total (*N*)
	Total	9	31	20	54	103	217
Gender	Females	1 (11.1%)	17 (54.8%)	9 (45.0%)	25 (46.3%)	61 (59.2%)	113
	Males	8 (88.9%)**	14 (45.2%)	11 (55.0%)	29 (53.7%)	41 (39.8%)	103
Co-morbidity	No diagnosis /unknown	4 (44.4%)	19 (61.3%)	13 (65.0%)	34 (63.0%)	75 (72.8%)	145
	Two diagnosis	1 (11.1%)	4 (12.9%)	6 (30.0%)	14 (25.9%)	21 (20.3%)	46
	Three or more diagnoses	4 (44.4%)*	8 (25.8%)*	1 (5.0%)	6 (11.1%)	7 (6.8%)	26
Risk to self	Not assessed	2 (22.2%)	8 (25.8%)	8 (40.0%)	17 (31.5%)	36 (35.0%)	72
	Low	4 (44.4%)	13 (41.9%)	7 (35.0%)	24 (44.4%)	49 (47.6%)	97
	Medium	2 (22.2%)	7 (22.6%)	2 (10.0%)	9 (16.7%)	12 (11.7%)	32
	High	1 (11.1%)	3 (9.7%)	3 (15.0%)	4 (7.4%)	5 (4.9%)	16
Risk to others	Not assessed	2 (22.2%)	8 (25.8%)	8 (40.0%)	19 (35.2%)	37 (36.0%)	74
	Low	4 (44.4%)	17 (54.8%)	7 (35.0%)	32 (59.3%)	62 (60.2%)	122
	Medium	3 (33.3%)*	4 (12.9%)	2 (10.0%)	3 (5.6%)	4 (3.9%)	16
	High	0	2 (6.5%)***	3 (15.0%)	0	0	5
Deliberate self-harm	No/Unknown	6 (66.7%)	18 (58.1%)	15 (75.0%)	33 (61.1%)	65 (63.1%)	137
	Yes	3 (33.3%)	13 (41.9%)	5 (25.0%)	21 (38.9%)	38 (36.9%)	80
Previous suicide attempt	No/Unknown	8 (88.9%)	20 (64.5%)	17 (85.0%)	41(76.0%)	82 (79.6%)	168
	Yes	0	8 (25.8%)	2 (10.0%)	7 (13.0%)	18 (33.3%)	35
	Multiple	1 (11.1%)	3 (9.7%)	1 (5.0%)	6 (11.1%)	3 (2.9%)	14

* *p* < .025   ** *p* < .01   *** *p*< .001   ($x^\dagger 2$ **coefficients**)

CHAPTER IV: DISCUSSION

Overview

This study examined the diagnoses, symptoms, and rates of adverse childhood events of children and adolescents at a New Zealand CAMHS. The purpose of the study was to explore, and attempt to disentangle, the complicated relationship between ADHD and adverse childhood experiences. The first specific aim was to establish whether or not there is a relationship between the symptoms of ADHD, PTSD, disruptive behavioural disorders, and adverse childhood events (e.g. sexual abuse, physical abuse, emotional/psychological abuse, neglect, exposure to domestic/family violence, grief/loss, parental discord/conflict, parental mental illness, parental substance use, serious illness/disability, family illness/disability, impoverishment, and bullying at school). Secondly, the study aimed to determine whether there is a sub-group of children and adolescents with symptoms of ADHD that constitute a 'complicated ADHD' group. The study aimed to investigate whether or not this 'complicated ADHD' group had higher rates of adverse childhood experiences, higher rates of diagnostic co-morbidity, and more serious externalizing symptoms. These two aims are examined below under two main research questions:

1. Is there a relationship between adverse childhood experiences and symptoms of ADHD, PTSD and disruptive behaviour disorders?

This relationship was examined in three ways. Firstly, this study looked at the association between individual diagnoses of ADHD, ODD, CD and PTSD and adverse childhood events. Secondly, the study examined the relationship between symptom clusters of ADHD, CD, ODD, and PTSD and adverse childhood events. Lastly, the relationships between individual symptoms of ADHD, OD, CD, and PTSD and adverse childhood events were investigated. These relationships, along with the demographic rates of diagnosis and adverse childhood experiences in the sample, are discussed next:

Diagnosis and co-morbidity

The majority (80.2%) of the participants were allocated at least one formal diagnosis. There were also high rates of diagnostic co-morbidity among the participants, with 21.2% having two diagnoses, and 11.9% having three or more diagnoses. The most common ten diagnoses, in order from most to least prevalent, were: depression -NOS, ADHD, adjustment disorder, relationship problems, anxiety-NOS, aspergers, family dynamic issues, major depressive disorder, generalised anxiety disorder, and dysthymia. These findings are similar to large-scale international reviews which have found the most common psychiatric problems among children and adolescents aged 5-7 years are: mood disorders, anxiety disorders, and disruptive behaviour disorders (Costello, Egger, & Angold, 2005).

Females in the current study had higher rates of major depressive disorder, relationship problems, and depression-NOS. Comparatively, males had higher rates of ADHD and aspergers. These findings are similar to epidemiology studies which have consistently found girls to have higher rates of anxiety and mood disorders compared to males. Comparatively, males have been shown in previous studies to experience higher rates of disruptive behaviour disorders than girls (Merikangas, Makamura, & Kessler, 2009).

Rates of adverse childhood events

The children and adolescents in the study were found to experience, on average, 3.3 adverse childhood events and 88.7% experienced at least one adverse childhood event. A further 58.0% experienced three or more adverse childhood experiences. The most common adverse childhood experiences in the current study were parent mental health problems, followed by divorce, emotional/psychological abuse, bullying, and physical abuse (in order from most prevalent to least prevalent). It is important to note that the prevalence rates of adverse childhood experiences documented in this study represent only that which is disclosed and recorded in the participant's

clinical file. It can be safely assumed that these prevalence rates are an under-representation of the true prevalence rates of adverse childhood events experienced by the children and adolescents in the study (Agar & Read, 2002; Wurr & Partidge, 1996).

Children and adolescents of Maori ethnicity experienced a significantly higher rate of adverse childhood events, compared to the other children and adolescents included in the study. This is consistent with previous research in New Zealand which found that individuals of Maori ethnicity reported higher rates of physical abuse and exposure to domestic violence in childhood, compared to individuals of other ethnic groups (Marie, Fergusson, & Boden, 2009). There were no differences in the average rates of adverse childhood experiences among gender or age groups. However, females were more likely to experience sexual abuse and emotional/psychological abuse compared to males. This is consistent with similar research carried out in New Zealand using a chart review method in adult mental health service users. This research found that adult females were more likely to have a history of childhood sexual abuse recorded in their clinical files, compared to male adults (Agar and Read, 2002).

Twenty nine percent of the children and adolescents in the study had documented experiences of physical abuse. This rate is higher than Agar and Read (2002), who found that 17% of the adults in the study had experienced childhood physical abuse. Review studies indicate slightly higher prevalence rates of physical abuse among mental health service users. A review of 46 studies looking at female in-patients and out-patients revealed that 48% had been subjected to childhood physical abuse (Read, Van Os, Morrison, & Ross, 2005. A review of 31 studies looking at men in in-patient and out-patient mental health facilities, found that 28% had experienced childhood physical abuse (Read, et al., 2005). Overall, the physical abuse rates found in the clinical files in this study are slightly higher than those found in New Zealand research looking at adults and similar to the physical abuse rates identified in review studies.

In terms of sexual abuse, 13.8% of the children and adolescents in the current study had this documented in their clinical files. This was a slightly lower rate compared to other chart review studies of adults in New Zealand which found rates of childhood sexual abuse at 20% (Agar and Read, 2002). This is also lower than sexual abuse rates identified in a review study, which found 48% of females and 28% of males in adult in-patient and out-patient mental health facilities had been subject to childhood sexual abuse. In a further New Zealand study, 30% of women in a community population were found to have an 'unwanted and abusive sexual experience' prior to the age of 16 years (Mullen, Martin, Anderson, & Romans et al., 1993). Overall, the sexual abuse rates found in the clinical files in this study are slightly lower than those found in previous research. This variability may reflect the fact that this study looked at the clinical notes of children and adolescents, whereas the studies discussed above investigated adult populations and looked at retrospective accounts of sexual abuse. The disclosure of sexual abuse may be complicated in child and adolescent populations due to legal issues, children feeling uncomfortable talking about sexual abuse, children being told by family members not to disclose such events to mental health professionals, or children not being asked by mental health professionals.

Other factors that may relate to the discrepancies found in rates of adverse childhood events in the current study compared to previous research may include: differences in sample sizes, variable definitions of what constitutes 'abuse', varying age limits for what is included as child abuse, and the subjective interpretation of individual researchers in the inclusion/exclusion of adverse childhood events.

Nevertheless, it is a significant finding and consistent with previous studies, that the majority of users of mental health services, child or adult, have experienced adverse childhood events. With more than half (58%) of the children and adolescents in the current study reporting three or more adverse childhood experiences.

The relationship between attention deficit hyperactivity disorder and adverse childhood events

In the current study, children and adolescents with a diagnosis of ADHD did not have a higher average rate of adverse childhood events or a higher rate of individual adverse events than children and adolescents in the study without a diagnosis of ADHD. However in terms of the ADHD symptom cluster, children and adolescents with a high number of ADHD symptoms were found to have a significantly higher number of adverse childhood events, and a higher rate of physical abuse compared to children and adolescents in the study without these symptoms.

When looking at individual symptoms, children and adolescents with the hyperactivity symptom had a higher average rate of adverse events, and a higher rate of physical abuse, parent substance use, and domestic violence compared to children and adolescents in this study without these symptoms. Children and adolescents with the impulsivity symptom did not have a higher average number of adverse events. However, they had a higher rate of physical abuse. Children and adolescents with the inattentive symptom did not have a higher average rate of adverse events or a higher rate of individual adverse events.

In summary, the findings from this study support the hypothesis that there is a relationship between adverse childhood events and the symptoms of ADHD, in particular, the hyperactivity and impulsivity symptoms. This supports a substantial amount of previous research that has found a link between the symptoms of ADHD and experiences of maltreatment, abuse and/or trauma (Conway, Oster, & Szymanski et al., 2011; Endo, et al., 2006; Famularo, Fenton, & Kinscherff, 1993; Famularo, et al., 1996; Kendall-Tackett, et al., 1993; McLeer et al., 1994; McLeer, et al., 1998; Merry & Andrews, 1994; Stern, et al., 1995).

As stated above, the hypothesis that a diagnosis of ADHD would be related to adverse childhood events was not confirmed. This finding is variable to previous research that has found children with a diagnosis of ADHD to experience higher rates of abuse, trauma, and/or maltreatment (Callaghan, Henry, & Wallen, 1994; Conway, Oster, & Szymanski et al., 2011; Endo,

et al., 2006; McLeer, Merry & Andrews, 1994; Kendall-Tackett, et al., 1993; Rucklidge et al., 2006; Weinstein, et al., 2000). This discrepancy may have occurred for a number of reasons. Firstly, the current study examined a sample of clients attending an out-patient CAMHS. This included children and adolescents with a variety of diagnoses and presenting problems. Due to this study design, there were only 25 children and adolescents in the sample with a diagnosis of ADHD. In contrast, previous research has examined a variety of populations including: groups of girls with a known diagnosis of ADHD (Briscoe-Smith & Hinshaw, 2006), groups of children and adolescents with a known diagnosis of ADHD (King et al., 1998), children and adolescents at inpatient psychiatric facilities (Endo et al., 2006), adults with a known ADHD diagnosis (Rucklidge et al., 2006) and children and adolescents with a known history of abuse and/or trauma (Conway, Oster, & Szymanski et al., 2011; Famularo, et al., 1996; Glod et al., 1996; McLeer et al., 1994; Merry & Andrews, 1994). It can be hypothesized that the rates of adverse childhood experiences and the diagnostic profiles of the participants included in these various studies would have varied. Secondly, this discrepant finding could reflect that children with symptoms of ADHD and a known history of adverse childhood events may not receive a diagnosis of ADHD. This could be because these children have more 'complex' symptom presentations and the aetiology of their symptoms may be understood in a more psychosocial nature, and perhaps be diagnosed as PTSD. Therefore, these children may not receive a diagnosis of ADHD, despite having the inattentive, hyperactive and impulsive symptoms. Finally, this discrepant finding may reflect quite the opposite, in that there may be a lack of enquiry around topics such as abuse and trauma in out-patient CAMHS's in New Zealand. In children with a known diagnosis of ADHD, there may be a lack of assessment around psychosocial factors such as abuse history. This may reflect the more biological or medical understanding of ADHD among mental health professionals (Kildea, Wright, & Davies, 2011; Mitchell & Read, 2011; Timimi et al., 2004). Never the less, it is impossible to extricate with any certainty why the current study did not find an association between a diagnosis of ADHD and a

history of adverse childhood events. Further research would need to include a larger sample of children and adolescents and control for factors such as the rates of enquiry and documentation of adverse childhood events.

The current study does provides evidence that children exposed to a high rate of adverse childhood events, particularly physical abuse, domestic violence, and parental substance abuse, are likely to be hyperactive and impulsive. However, these adverse childhood events were not associated with inattentiveness. From the data gathered, it is difficult to understand why this was the case. However, it can be hypothesized that children who experience adverse events such as physical abuse and exposure to domestic violence are in a 'traumatic stress response' which puts them in a state of hyper-arousal. In children and adolescents, this persisting state of hyper-arousal may manifest as hyperactivity and/or impulsivity, rather than inattentiveness (Glod & Teicher, 1996; Perry, 2004; Perry, et al., 1995; Wilson et al., 2011). Furthermore, the finding that adverse childhood events were associated with hyperactivity and impulsivity, but not inattentiveness, supports the idea that ADHD is caused by multiple pathways that lead to various manifestations of the disorder (Connor, 2002; Nigg, 2006; Steinhausen, 2009). This also suggests that adverse childhood events such as physical abuse and exposure to domestic violence may be associated with the ADHD predominantly hyperactive-impulsive type. This provides tentative evidence that there may be a group of children with 'complicated ADHD', who vary in terms of their symptoms, diagnostic co-morbidity, and experiences of adverse childhood events. This will be looked at later in this discussion.

Relationship between oppositional defiant disorder and adverse childhood events

Children and adolescents with a diagnosis of ODD were found to have a higher average rate of adverse events and domestic violence compared to children and adolescents in the study without that did not have this diagnosis. In terms of the symptom cluster, those with a high rate of ODD

symptoms did not have a higher average rate of adverse events. However, they did experience higher rates of neglect and impoverishment. In terms of specific ODD symptoms, children and adolescents with the non-compliance symptom did not have a higher average rate of adverse events or a higher rate or specific adverse events. Children and adolescents with the oppositional behaviour symptom did not have a higher average rate of adverse events. However, they experienced higher rates of physical abuse.

In summary, the findings from this study indicate that there is a significant relationship between adverse childhood events and the diagnoses and symptoms of ODD. This finding is consistent with previous research that found a relationship between the symptoms and diagnoses of ODD and adverse childhood events (Famularo, Fenton, & Kinscherff, 1993; Famularo, et al., 1996; Herbert et al., 2006; Merry & Andrews, 1994; Webster, 2001). More specifically, high rates of ODD diagnoses have been found in children and adolescents with a history of maltreatment (Falmularo, Fenton, & Kinscherff, 1993).

There are some discrepancies between the findings from the current study and previous research findings. The current study did not find a significant relationship between sexual abuse and a diagnosis of ODD. This is discrepant to New Zealand research that has found a relationship between ODD and sexual abuse (Merry & Andrews, 1994). This variability could relate to a number of factors. The study by Merry and Andrews (1994) investigated a population of children with a known history of sexual abuse. In contrast, only 13.8% of the children and adolescents in the present study had a known history of sexual abuse. Due to this discrepancy, it can by hypothesized that the diagnostic and symptom profiles of the participants included in the two studies may have varied.

Relationship between conduct disorder and adverse childhood events

Children and adolescents with a diagnosis of CD were not found to have a higher average rate of adverse events or a higher rate of individual adverse events compared to children and adolescents without this diagnosis. In terms of symptom clusters, those with a high number of CD symptoms were found to experience a higher total number of adverse events, a higher rate of physical abuse, sexual abuse, neglect, and domestic violence, compared to children and adolescents in this study without these symptoms. When considering individual CD symptoms, children and adolescents with the aggression to people and animals symptom did not have a higher average rate of adverse events. However, they had higher rates of physical abuse, domestic violence, impoverishment, and family illness/disability. Children and adolescents with the destruction of property symptom did not have a higher average rate of adverse events. However, they experienced higher rates of neglect. Children and adolescents with the deceitfulness and/or theft symptom had a significantly higher average rate of adverse events. Individuals with the serious violation of rules symptom had a higher average rate of adverse events, and they experienced higher rates of physical abuse, domestic violence, and parent substance abuse.

As stated above, the hypothesis that a diagnosis of CD would be related to adverse childhood events was not confirmed. However, the findings from this study support the hypothesis that there is a relationship between adverse childhood events and the symptoms of CD. This is consistent with findings in New Zealand longitudinal research, which have established that there is an association between the symptoms of CD and neglect, verbal/psychological abuse, physical abuse, and sexual abuse (Young, et al., 2006). However, the findings from the current study do not support international studies which have consistently found a relationship between a diagnosis of CD and adverse experiences such as poor parenting, parental mental health problems, impoverishment, sexual and physical abuse, and neglect (Burke, Loeber, & Birmaher, 2002).

This discrepancy may have occurred for a number of reasons. Firstly, there were only a small number of children and adolescents in the study with a diagnosis of CD (2.8%). Secondly, this discrepant finding could reflect that children with symptoms of CD and a known history of adverse childhood events may not receive a diagnosis of CD. Similarly to children with symptoms of ADHD and a history of adverse experiences, this could be because these children have more 'complex' symptom presentations. The aetiology of their symptoms may be understood in a more psychosocial nature, and perhaps be diagnosed as PTSD. It is impossible to conclude with any certainty why the current study did not find an association between a diagnosis of CD and adverse childhood experiences. It is clear there is a need for further research that includes a larger sample of children and adolescents.

The relationship between post-traumatic stress disorder and adverse childhood events

Children and adolescents with a diagnosis of PTSD did not experience a higher average number of adverse childhood events compared to children and adolescents in this study without this diagnosis. However, children and adolescents with a diagnosis of PTSD did experience a higher rate of sexual abuse and physical abuse, compared to children and adolescents in the study without this diagnosis. In terms of symptom clusters, children and adolescents with a high number of PTSD symptoms were found to experience a higher average number of adverse events. When looking at individual PTSD symptoms, children and adolescents with the increased arousal symptom experienced a higher average number of adverse events, sexual abuse, and loss. Children and adolescents with the re-experiencing of traumatic events symptom did not have a higher average number of adverse events. However, they experienced higher rates of sexual abuse. Finally, children and adolescents with the avoidance and numbing symptom did not experience a higher average rate of adverse events. However, they experienced higher rates of loss and parent mental health problems.

The hypothesis that children and adolescents with a diagnosis of PTSD would experience a higher average number of adverse childhood experiences was not confirmed. This may have occurred for a number of reasons. Firstly, a diagnosis of PTSD may be associated with specific abusive and/or traumatic experiences, rather than a collective number of adverse experiences. Secondly, there were a very small number of children and adolescents in the study with a diagnosis of PTSD (4.1%).

However, the study did find that children and adolescents with symptoms of PTSD had higher rates of sexual abuse, physical abuse, loss, and parent mental health problems. These findings make sense when considering that PTSD is one of the most commonly diagnosed disorders in children and adolescents with a history of trauma and/or abuse (McLeer, et al., 1994; Weinstein, et al., 2000). In addition, research has demonstrated that children with a history of child maltreatment (i.e. physical abuse, sexual abuse, neglect, and impoverishment) are found to have high rates of PTSD in comparison to children without a history of child maltreatment (Falmularo, Fenton, & Kinscherff, 1993).

The findings from this study support previous research which has found that children with a history of sexual abuse have higher rates of PTSD symptoms, compared to those without a history of sexual abuse. For example, New Zealand research has found high rates of PSTD among children with a known history of sexual abuse (Merry and Andrews, 1994). This is further supported in international research that has found children with a history of sexual abuse to have higher rates of PTSD, compared to community populations (McLeer et al., 1998). Furthermore, childhood sexual abuse had been consistently linked to PTSD symptoms such as heightened anxiety and increased arousal (Briscoe-Smith & Hinshaw, 2006; Dubowitz et al., 1993; Weinstein et al., 2000). This is also supported by a review of 45 studies which found children with a history of sexual abuse to exhibit high rates of PTSD (Kendall-Tackett, et al., 1993).

Summary

In summary, the current study provides evidence that children and adolescents who experience adverse childhood events are more likely to exhibit symptoms of ADHD, ODD, CD, and PTSD. Furthermore, these children and adolescents are likely to have a diagnosis of ODD. This finding supports the evidence in international studies investigating symptoms of ADHD and adverse childhood experiences. The current study also builds on previous research by Merry and Andrews (1994) and Rucklidge et al. (2006), by examining a sample of children and adolescents in a New Zealand context.

The Empirical Link between ADHD and Adverse Childhood Experiences

The findings from the current study suggest that adverse childhood events may play an aetiological role in the development of ADHD. These findings will be considered in light of the three main hypotheses relating to the empirical link between ADHD and adverse childhood experiences (as outlined in the Introduction).

Hypothesis I

When referring back to the introduction, it would seem that this study provides evidence to support the first hypothesis: "adverse childhood experiences are an aetiological and/or exacerbating factor in the development of attention deficit and disruptive behavioural disorders" (p.54). This supports research by Briscoe-Smith & Hinshaw (2006), which found girls with ADHD and a history of abuse to exhibit higher levels of problematic externalizing behaviour, compared to girls with ADHD and no history of abuse. The girls with ADHD and a history of abuse were also found to have higher levels of aggressive behaviour and peer rejection. This finding is further supported by research which identified a particular group of children with ADHD, who have an impaired stress-response and a more developmentally persistent form of ADHD (King et al., 1998). This is

also consistent with Endo et al. (2006) who established that a high number of children with abuse histories fit the criteria for ADHD and dissociative disorder, and that many of these children began displaying symptoms of ADHD following their documented abuse.

Further supporting this hypothesis is the fact that the current study found significant relationships between symptoms of ADHD and adverse childhood experiences such as physical abuse and domestic violence. This finding has been consistently demonstrated in previous research (Conway, Oster, & Szymanski et al., 2011; Endo, et al., 2006; Famularo, Fenton, & Kinscherff, 1993; Famularo, et al., 1996; Kendall-Tackett, et al., 1993; McLeer et al., 1994; McLeer, et al., 1998; Merry & Andrews, 1994; Stern, et al., 1995). Therefore, as found in this study, it is widely accepted that children with symptoms of ADHD have higher than expected rates of childhood adverse experiences. Based on this understanding, it is logical to conclude that there is some kind of aetiological relationship where adverse childhood experiences place children at a significant risk of developing and/or exacerbating the symptoms of ADHD and other disruptive behaviour disorders.

The theory underpinning these findings is that children who are exposed to chronic trauma and/or stress have alterations in brain neurochemistry, which puts them in a persistent stress-response state. Some argue that this constant state of hyper-arousal may lead to ADHD-type symptoms (Glod & Teicher, 1996; Perry, 2004). Exposure to childhood trauma and/or chronic stress may exert neurological changes in a child's brain that lead to ongoing issues with inattentiveness, hyperactivity, and impulsivity. Similarly, one study found that family adversity moderates the impact of the DAT1 gene, which has been implicated in the development of ADHD symptoms (Laucht, et al., 2007). The study found that individuals with the DAT1 gene only developed symptoms of ADHD if they were exposed to psychosocial adversity, compared to those living in less adverse family conditions. This supports the broader notion that ADHD is perhaps caused by an interaction of multiple biological, neurological, and environmental pathways that lead to various manifestations of the disorder (Connor, 2002; Nigg, 2006; Steinhausen, 2009).

When considering the aetiological relationship between ADHD and adverse childhood experiences it seems there are some areas that remain unclear. From the information available, it is difficult to untangle whether or not: a) the early adverse childhood experiences led the children and adolescents in the study to develop ADHD-type symptoms, or b) the children and adolescents in the study had pre-existing symptoms of ADHD and the adverse childhood experiences exacerbated the symptoms of ADHD or c) both of the above. Based on the findings from this study and from previous research, it is impossible to make any causal connections, or pull apart which of these is in fact correct.

Overall, this study provides evidence that there is a significant relationship between adverse childhood experiences and the symptoms of ADHD in children and adolescent clients of a New Zealand CAMHS. This evidence suggests that adverse childhood experiences are perhaps an aetiological and/or exacerbating factor in the development of attention deficit and disruptive behavioural disorders. This is consistent with previous research that has found an aetiological relationship between ADHD and adverse childhood experiences (Briscoe-Smith & Hinshaw, 2006; Endo et al., 2006; King et al., 1998). However, this relationship is by no means clear cut and it is important to consider the results in light of hypothesis two and three.

Hypothesis II

The second hypothesis states that the "high overlap of childhood adverse events, PTSD and attention deficit and disruptive behavioural disorders is an issue of high symptom overlap rather than an aetiological relationship" (p. 62) (Conway et al., 2011; Findling, et al., 2007; Szymanski, et al., 2011; Weinstein, et al., 2000). The idea behind this hypothesis is that the symptoms of PTSD, and ADHD are similar, and the high overlap of childhood adverse experiences, PTSD, and ADHD found in the literature may reflect symptom similarity, rather than an aetiological relationship (Findling et al., 2008; Weinstein et al., 2000; Szymanski et al., 2011). The high overlap of ADHD

and PTSD symptoms has led some researchers to caution that there may be a high number of children with a diagnosis of ADHD, along with undetected trauma symptoms and in some cases, undiagnosed PTSD (Ford et al., 2000). Furthermore, this issue may be more prevalent in children because they are known to manifest PTSD symptoms differently to adults. There is research to suggest that children manifest PTSD as externalizing symptoms that are similar to ADHD (Conway et al., 2011). The findings from the current study cannot confirm, nor disconfirm this hypothesis. It could be argued that the significant relationship between adverse childhood experiences and the symptoms of ADHD found in this study may reflect a similarity between the symptoms of ADHD and PTSD in children and adolescents. For example, the avoidance symptoms in PTSD, e.g. inattentiveness, distractibility, and avoidance of activities are similar to the inattentiveness symptoms in ADHD (Szymanski et al., 2011). Furthermore, the hyper-arousal symptoms in PTSD are similar to the hyperactive and impulsive symptoms in ADHD, e.g. fidgeting, excessive moving around, and restlessness (Szymanski et al., 2011; Weinstein et al., 2000). Therefore, in the current study there is a chance that the symptoms of PTSD may have been misinterpreted by clinicians as symptoms of ADHD, and correspondingly included as inattentiveness, impulsivity, or hyperactivity.

On the contrary, the current study investigated not only diagnoses of ADHD, but the individual symptoms, i.e. inattentiveness, hyperactivity, and impulsivity. Therefore, one could argue that the children in the study did in fact have 'ADHD' and perhaps co-morbid symptoms of PTSD. It seems the issue of high symptom overlap between PTSD and ADHD in children and adolescents may reflect an issue in the diagnostic system of ADHD, and the lack of a clear aetiology of the disorder. The current understanding around the aetiology of ADHD is complex, and many agree there are multiple aetiological pathways that lead to various manifestations of the disorder (Nigg, 2006; Steinhausen, 2009). This suggests that ADHD may manifest as different symptoms that vary in their severity and persistency among different children. There is perhaps a

group of children with ADHD and a history of trauma that present differently to children without such a pervasive history of adverse childhood events.

The highly heterogeneous nature of ADHD as a diagnosis is problematic. These findings raise issues about the diagnostic system as a whole. It seems that the *DSM-IV* artificially categorises children into diagnostic groups which can wrongly imply biological (ADHD) or psychosocial/trauma (PTSD) aetiologies. In contrast, a focus on specific problems or symptoms may be more useful clinically, and in terms of causation research. It is clear there an urgent need for clarification of the diagnostic system around what is, and what is not ADHD.

Hypothesis III

The third hypothesis proposes that "ADHD, CD, and ODD are potential risk factors or predictors for later aversive events such as physical abuse" (p.64). The theory behind this is that children with ADHD are more susceptible to be abused and/or maltreated due to their disruptive behaviour and impulse control problems (Ouyang, et al., 2008; Whitmore, Kramer, & Knutson, 1993; Wozniak, et al., 1999). It is thought that parents, who are unaware that their child's inattentive behaviour is attributable to a disorder, consequently blame and punish the child through physical punishment and/or neglect. While there is some research to support that symptoms of ADHD predict subsequent abuse (Barkley, et al., 1993; Lam, 2005; Ouyang et al., 2008), other studies have provided evidence to challenge this idea (Whitmore et al., 1993; Wozniak et al., 1999).

A limitation of the current study is that the timing of adverse childhood experiences is not available. This makes it difficult to ascertain whether or not adverse childhood experiences such as physical abuse were experienced before or after the onset of ADHD symptoms. It could be hypothesized that some of the children and adolescents experienced both, i.e. adverse experiences before the onset of ADHD symptoms, along with ongoing adverse experiences and punitive parenting following the onset of ADHD. It has been clearly established in research that parents of

children with ADHD tend to adopt a more coercive parenting style that is negative, reactive, directive, and commanding (Bauermeister et al., 2007; Johnston & Mash, 2001; Pfiffner et al., 2005; Stormshack, et al., 2000). However, it is difficult to establish whether or not it is the child's difficult behaviour that impacts negatively on parenting style, which in turn leads to more negative and punitive parenting (Finzi-Dottanet al., 2006). Or conversely, a dysfunctional parenting style may play an aetiological role in the development of 'difficult' child behaviour and symptoms of ADHD (Pfiffner et al, 2005). Or as many may argue, it could be a combination of both (Johnston & Mash, 2001; Pierrehumbert et al., 2000). Either way, the findings from the current study cannot confirm, nor disconfirm this hypothesis. It is clear there is a need for longitudinal research that investigates the timing of adverse childhood experiences such as physical abuse, and the onset of ADHD-type symptoms.

2. Is there a group of children with "complicated ADHD"?

The second aim of the study was to determine whether there is a sub-group of children and adolescents with symptoms of ADHD that might be considered a 'complicated ADHD' group. The study aimed to investigate whether or not this 'complicated ADHD' group has a higher average number of adverse childhood experiences, a higher rate of diagnostic co-morbidity, and more serious externalizing symptoms. Based on previous literature, it was hypothesised that children and/or adolescents with 'complicated ADHD' would have a high rate of co-morbidity with other *DSM-IV* disorders and more serious externalising symptoms (i.e. a higher number of *DSM-IV* symptoms of the ADHD, CD, and ODD). It was also hypothesised that this group of children would have a higher rate of childhood adverse events in comparison to other children with symptoms of ADHD included in the current study (Briscoe- Smith & Hinshaw, 2006; Glod et al., 1996; King et al., 1998).

This question was addressed by grouping the children and adolescents into homogenous groups based on their symptom profiles of the inattentive and disruptive behaviour disorders (ADHD, ODD, and CD). This produced five separate groups of participants, each with different symptom profiles. The first group of children and adolescents were low across all ADHD, ODD, and CD symptoms. This group was the largest and constituted just under half of the participants in the study (baseline group 47.5%). The second group was high in ODD symptoms, but low in ADHD and CD symptoms (ODD group 24.9%). The third group of children and adolescents were high in ODD and ADHD symptoms, but low in CD symptoms (ODD and ADHD group 9.2%). The fourth group of participants were high in ODD and CD, but low in ADHD symptoms (ODD and CD group 14.3%). Finally, the fifth group of children and adolescents were high in all ADHD, CD, ODD symptoms ('high all' group 4.2%). The 'high all' group was the smallest group. However, children and adolescents in this group were consistently high across all of the inattentive and externalizing symptoms of ADHD, ODD, and CD.

The next step was to establish whether or not one of these groups did in fact constitute a 'complicated ADHD' group (Briscoe-Smith & Hinshaw, 2006; Endo, et al., 2006; Famularo, et al., 1993; Famularo, et al., 1996; McLeer, et al., 1994; McLeer, Deblinger, Henry, & Orvaschel, 1992; McLeer, et al., 1998; Merry & Andrews, 1994). This was achieved by examining the relationships between the five groups of children and adolescents in terms of a) the average number of adverse events, b) rates of individual adverse events, c) diagnostic co-morbidity and d) risk to self and others, rates of deliberate self-harm, and previous suicide attempts. The relationships that were found to be significant will be considered for each separate group:

'ADHD and ODD' group

Children and adolescents in the 'ADHD and ODD' group were found to experience higher rates of loss and domestic violence compared to children and adolescents in the other groups. They

were also more likely to have a 'high' risk to others compared to the other groups. Children and adolescents in this group did not differ in terms of their average number of adverse events, diagnostic co-morbidity, risk to self, rate of deliberate self-harm, or previous suicide attempts compared to children and adolescents in the other four groups.

'CD and ODD' group

Children and adolescents in the 'CD and ODD' group did not differ in terms of their average number of adverse events, rates of individual adverse events, diagnostic co-morbidity, risk to self or others, rate of deliberate self-harm, or previous suicide attempts, compared to the other groups.

'ODD' group

Children and adolescents in this group did not differ in terms of their average number of adverse events, rates of individual adverse events, diagnostic com-morbidity, risk to self or others, rate of deliberate self-harm, or previous suicide attempts, compared to the other groups.

'Baseline' group

Interestingly, children and adolescents in the 'baseline' group were found to experience significantly lower levels of physical abuse and domestic violence, compared to children and adolescents in the other groups. However, children and adolescents in this group did not differ in terms of their average number of adverse events, diagnostic co-morbidity, risk to self and others, rate of deliberate self-harm, or previous suicide attempts.

'High all' group

There were significantly more males than females in the 'high all' group (88.9% were male). The 'high all' group had a significantly higher average number of adverse events compared

to children and adolescents in the other four groups, with children and adolescents in this group experiencing almost double the number of adverse childhood events than children and adolescents in the other groups. Furthermore, children and adolescents in the 'high all' group were found to experience higher rates of physical abuse, neglect, and exposure to domestic violence, compared to the other four groups. Children and adolescents in this group also had a higher rate of diagnostic co-morbidity, with 44.0% of this group having three or more formal diagnoses. In terms of risk, children and adolescents in this group were more likely to have 'medium' risk to others, compared to children and adolescents in the other groups. However, children and adolescents in this group did not differ in terms of their risk to self, rate of deliberate self-harm, or previous suicide attempts, compared to other groups.

Examining the 'complicated ADHD' group

In summary, the results indicate that the children and adolescents in the 'high all' group appear to experience a cluster of symptoms that could be considered as a 'complicated ADHD' group. The children in the 'high all' group were high across all of the inattentive and externalising symptoms of ADHD, ODD, and CD and had a higher rate of diagnostic co-morbidity with other *DSM-IV* disorders when compared to the children and adolescents in the other groups. The 'high all' group experienced a higher average rate of adverse events, along with higher rates of physical abuse, neglect, and exposure to domestic violence, compared to the children and adolescents in the other groups. These findings appear to support the hypothesis that there would be a group of children with symptoms of ADHD that may have a more 'complicated ADHD'; with a higher rate of co-morbidity with *DSM-IV* disorders, and more serious externalising symptoms. These findings also support the hypothesis that this group of children would have a higher average rate of childhood adverse events compared to other children and adolescents with symptoms of ADHD (Briscoe- Smith & Hinshaw, 2006; Glod et al., 1996; King et al., 1998). This group experienced

almost double the average number of adverse childhood events than other children and adolescents in the study with symptoms of ADHD.

It is also important to consider that the children and adolescents in the 'ODD and ADHD' group were consistently high across the inattentive and externalising symptoms of ADHD and ODD (just not CD). Therefore, the 'ODD and ADHD' group might also be considered a 'complicated' ADHD group'. This group also experienced higher rates of domestic violence compared to the children and adolescents in the other groups. However, the children and adolescents in the 'ADHD and ODD' group did not differ in terms of their average number of adverse events, diagnostic co-morbidity, risk to self, rate of deliberate self-harm, or previous suicide attempts, compared to children and adolescents in the other four groups. When referring to the main aims of the study, it was hypothesised that the 'complicated ADHD' group would have more serious externalising symptoms (i.e. a higher number of *DSM-IV* symptoms of the ADHD, CD and ODD disorders), along with a higher rate of co-morbidity with other *DSM-IV* disorders, and a higher average rate of childhood adverse events, compared to other children in the study with symptoms of ADHD (Briscoe- Smith & Hinshaw, 2006; Findling et al., 2007; Glod et al., 1996; King et al., 1998). The fact that the 'ADHD and ODD' group did not have a significantly higher rate of diagnostic co-morbidity or a higher average rate of adverse childhood events compared to the other groups, suggests that this group may not constitute a 'complicated ADHD' group. Therefore for the purpose of this study, the 'ADHD and ODD' group will not be included as a 'complicated ADHD' group. However, it is important to recognise that the research around what constitutes 'complicated ADHD' and what this group looks like is very limited. It could be hypothesised that there is more than one form of 'complicated ADHD'. It is clear there is a need for replication of these findings and further research that examines the symptom profile and characteristics of the 'complicated ADHD' group in a large population of children and adolescents.

An interesting finding in the current study, is that the children and adolescents in the 'high all' group were predominantly male. While research suggests that ADHD is more common in males (males are three times more likely to have a diagnosis), 88.9% of the 'high all' group were male (APA, 2000; Das Banerjee, Middleton, & Faraone, 2007; Hinshaw & Blackman, 2005). This finding supports previous research that suggests males with ADHD exhibit higher rates of disruptive behaviour than females (Gershon & Gershon, 2002). However, this finding is contradicted by research that has found girls with ADHD and histories of abuse also exhibit higher levels of problematic externalizing behaviour. It seems there is a need for further research that compares the symptom trajectories of males and females who have ADHD and histories of adverse childhood events.

In summary, there may be a group of children and adolescents with 'complicated ADHD' who have a history of adverse childhood experiences, particularly physical abuse and domestic violence, that present differently to children with ADHD that do not have such a pervasive history of adverse childhood experiences. This suggests that early adverse experiences may create a more complicated profile of ADHD symptoms and perhaps change the way that ADHD symptoms manifest. This supports the idea that there may be some kind of aetiological relationship where adverse childhood experiences place children at a significant risk of developing and/or exacerbating the symptoms of ADHD and other disruptive behaviour disorders. As mentioned earlier in this discussion, these findings further support the theory that ADHD is caused by an interaction of multiple biological, neurological, and environmental pathways that lead to various manifestations of the disorder (Connor, 2002; Nigg, 2006; Steinhausen, 2009).

Clinical implications

This study confirms previous findings that children and adolescents with symptoms and/or diagnoses of ADHD, ODD, and CD experience high rates of adverse childhood experiences. This is the first study to establish this relationship in a population of New Zealand CAMHS users. Furthermore, this study provides evidence to suggest that there are a group of children and adolescents with symptoms of ADHD that have more serious externalizing symptoms (i.e. high levels of ADHD, ODD and CD symptoms) and a higher rate of diagnostic co-morbidity with other *DSM-IV* disorders. This group of children and adolescents experienced a higher average rate of adverse events, along with higher rates of physical abuse, neglect, and exposure to domestic violence than other children and adolescents included in the study. This provides evidence to suggest that early adverse experiences may create a more complicated symptom profile of ADHD and perhaps change the way that ADHD symptoms manifest.

Clinical Assessment and staff training

The above findings point to the need for careful assessment and screening of adverse childhood experiences for children and adolescents in New Zealand, particularly those that are referred for an assessment and/or diagnosis of ADHD. This would include thorough assessment around the child's history of abuse, maltreatment, loss, and/or trauma (Weinstein, et al., 2000). These findings also highlight the need for highly comprehensive initial assessments that gather information from a variety of sources such as the child or adolescent, their family, school, and other relevant services involved with the child. Comprehensive assessment is crucial in identifying the complex needs, areas of risks, and available resources of these children, their families, and professionals involved in their wider system (Kisiel, Fehrenback, Small, & Lyons, 2009). Furthermore, a comprehensive assessment ensures that treatment is tailored to meet the needs of the child and their family.

Despite the clear need for comprehensive assessment around adverse childhood experiences, many mental health professionals do not routinely inquire around such topics (Agar & Read, 2002; Donohoe, 2010; Young, Read, Barker-Collo, & Harrison, 2001). Research in New Zealand has identified several factors related to mental health professional's reluctance to ask about abuse (Young et al., 2001). These factors include: more pressing issues, fear of disturbing clients, biological aetiology beliefs, poor rapport with the client, fear of the client disengaging from the service, and fear of inducing 'false memories' (Agar, Read, & Bush, 2002).

One way to overcome the lack of inquiry of adverse childhood events is by having mandatory and standardized procedures around the screening of early adverse experiences in the assessment of children and adolescents referred to New Zealand CAMHS. This may involve having a compulsory form in the electronic clinical records that screens for early adverse experiences. This type of information could be checked or reviewed in the MDT process to ensure that staff are enquiring around such events. Research in New Zealand adult mental health suggests that having an abuse section in the initial assessment form is associated with a significantly higher identification of abuse histories (Agar, Read, & Bush, 2002). The CAMHS used in this particular study did not appear to utilize a consistent form or checklist for adverse childhood experiences in the initial assessment process.

Implementation of mandatory inquiry of adverse childhood events should be accompanied by training for mental health professionals working in CAMHS's (Agar & Read, 2002; Read & Fraser, 1998). This would help in over-coming many of the factors listed above that relate to staff reluctance in asking about abuse (Young et al., 2001). Furthermore, adequate training is important so that staff know what to do when patients do disclose abuse (Donohoe, 2010). Many mental health professionals working in New Zealand CAMHS may not have received such training. Ideally, training would include education around how and when to inquire around adverse childhood experiences, how to respond to disclosures of abuse, and how to incorporate such

information into a psychological formulation and treatment plan (Agar & Read, 2002; Donohoe, 2010). It is also important to educate staff around the relationship between ADHD and adverse childhood experiences. Better understanding of this relationship will enhance mental health professional's abilities to identify and properly treat children that are at risk (Syzmanski, Sapanski, & Conway, 2011).

Similar training programmes have been implemented in New Zealand and England for staff working at adult mental health services (Donohoe, 2010; Read & Cavanagh, 2004). Research investigating the efficacy of such training programmes identified that staff felt more confident about asking about adverse events such as sexual abuse and had an overall increase in skills and knowledge relating to the relationships between abuse and mental health (Donohoe, 2010; Read & Cavanagh, 2004). It is clear there is a need for similar training programmes for staff working in CAMHS.

Diagnostic system for ADHD

As the findings from this study highlight, diagnosing ADHD in children with a history of adverse childhood events is complex (Weinstein, et al., 2000). The current understanding around the aetiology of ADHD is that there are multiple aetiological pathways that lead to various manifestations of the disorder (Nigg, 2006; Steinhausen, 2009; Taylor, 2009). This suggests that ADHD may manifest as different symptoms that vary in their severity and persistency among different children. The highly heterogeneous nature of ADHD as a diagnosis is problematic and these findings raise issues about the diagnostic system as a whole.

Furthermore despite having a consistent diagnostic system, research suggests that perceptions of ADHD symptoms vary not only between different countries, but also between mental health professionals in the same agency (Mann, et al., 1992; Sonuga-Barke, Minocha, Taylor, & Sandberg, 1993). It is clear that there are ongoing issues around what constitutes

'ADHD' and how the disorder is defined and diagnosed between various professionals (Radcliffe & Timimi, 2003; Taylor, 2009).

In addition, there is ongoing concern around the high overlap between ADHD and PTSD symptoms in some children, particularly for those children who may have a more 'complicated' form of ADHD (Ford et al., 2000). This raises a number of important clinical issues. Firstly, for children with a known history of adverse experiences and/or trauma, extra caution is required in making the diagnosis. For these children, a diagnosis of ADHD may not necessarily be helpful. This is mainly because a child who has experienced adverse events and trauma would require specific interventions to reduce the psychological impact of their trauma (Weinstein, et al., 2000), as opposed to the standard treatment that is provided for ADHD (pharmacological and behavioural interventions). There is perhaps a need for re-evaluation of the *DSM-V* criteria around the way ADHD and child-PTSD are defined and diagnosed. For example, PTSD could be included as a differential diagnosis for children with ADHD that have a known history of trauma.

As mentioned earlier in the discussion, it seems that the *DSM-IV* artificially categorises children into diagnostic groups which may wrongly imply biological (ADHD) or psychosocial/trauma (PTSD) aetiologies. Therefore, it would seem that a diagnosis of 'ADHD' may imply that it is a biologically based 'disorder', as a opposed to a problem that may have been caused by social factors. In contrast, a focus on specific problems or symptoms may be more useful clinically, and in terms of causation research. It is clear there an urgent need for clarification of the diagnostic system around what is ADHD and how we define, understand, and diagnose this disorder.

Treatment

In addition to having a comprehensive initial assessment and clear diagnostic criteria, a holistic treatment plan for ADHD is crucial. In terms of ADHD treatment, this does not appear to

be common practice. Over the last 15 years the use of pharmacological interventions to treat ADHD has increased dramatically (Faraone et al., 2006; Taylor, 2009; Mayes, Bagwell, Erkulwater, 2008; Olfson, et al, 2003; Zuvekas, Vitiello, & Norquist, 2006). It is estimated globally, that at least 85% of children diagnosed with ADHD are medicated with stimulants (Olfson, et al, 2003). It is generally agreed upon in the literature that the most effective treatment for ADHD is a multi-modal approach including pharmacological treatment and behavioural approaches (Brown et al., 2005; Jensen et al., 2001; Swanson et al., 2008; Van der Oord, Prins, Oosterlaan & Emmelkamp, 2008). However, there is concern that the growing use of medication to treat ADHD is side-lining other evidence based psycho-social treatments such as BPT, family based interventions, behavioural classroom interventions, school management strategies, and academic interventions (Barlow & Stewart-Brown, 2000; Chronis, Raggi, & Jones, 2006; DuPaul & Eckert, 1998; Goldman et al., 1998; Piquero, et al., 2009; Sereketich & Dumas, 1996; Thomas & Zimmer-Gembeck, 2007).

While there is an extensive body of research to support the short-term efficacy of using medication to treat core-ADHD symptoms, there are also many limitations (Chronis, Jones, & Raggi, 2006; Faraone et al., 2006; Van der Oord, Prins, Oosterlaan & Emmelkamp, 2008). Some of these include: adverse physical and psychological side effects (Cheng et al., 2007; Kratochvil et al., 2008; Radcliffe & Timimi, 2003), a paucity of research investigating long-term efficacy and safety of ADHD medication (Goldman et al., 1998; Kratochvil et al., 2008), and a lack of research that supports an improvement in social and academic functioning (Van der Oord et al., 2008). There is also the concern that children prescribed ADHD medication may be cultured into the attitude of 'a pills for life's problems' (Radcliffe & Timimi, 2003).

Furthermore, using medication to treat ADHD does not take into account the high number of children that have a history of adverse childhood experiences. The MTA study has revealed that a child's initial clinical presentation and social situation (i.e. severity of ADHD symptoms, conduct problems, intellectual ability, social advantage, and co-morbid problems) are more accurate

predictors of later functioning than type of treatment received in childhood (Molina et al., 2009). This research found that those children with ADHD, who had a lower social, demographic, academic, and behavioural advantage, were more likely to have a poor response to treatment and an overall negative long-term prognosis (Molina et al., 2009). Based on these findings, it would be fair to argue that the complicated ADHD group in the current study would have an overall poorer prognosis than other children in the study. This is supported by large body of research that suggests a co-morbid presentation of ADHD and CD and/or ODD is associated with an overall poorer prognosis than ADHD alone (Bauermeister et al., 2007; Barkley, 1998; Findling, Arnold, Greenhill, Kratochvil, & McGough, 2007; Spencer, Biederman, & Wilens, 1999).

The overall poor prognosis for this group highlights the need for holistic and individually tailored treatment approaches that go beyond medication, particularly those with a more complicated symptom presentation and a history of adverse childhood experiences. Most importantly, children that present with symptoms of ADHD that have a known history of abuse and/or trauma require specific interventions that reduce the psychological impact of their trauma, in addition to the standart ADHD treatment pathway. This may include: treatment of co-morbid psychological problems (including past trauma issues), BPT, family therapy to target dysfunctional patterns of interaction within the family and attachment difficulties, parent-child interaction therapy (PCIT), and ongoing liaison with relevant services and agencies such as school, and CYFS. There are also helpful services in New Zealand such as strengthening families and mentoring programmes that would be helpful for children with complicated ADHD.

Identifying early risk factors and prevention

From the findings in the current study, it is clear that children who experience adverse events early in life are at risk for developing ADHD and other disruptive behaviour problems. Furthermore, these children are at risk of falling into the 'complicated' ADHD group and are more

likely to have an overall negative prognosis (Molina et al., 2009). This highlights the need for early identification of children who are at risk and the importance of early intervention. In New Zealand there is a growing body of evidence to support the efficacy of early interventions that reduce early risk factors and increase protective factors (Mental Health Commission, 2011). This supports the use of evidence-based prevention programmes to increase resilience, positive parenting, and build skills for children at risk of developing problems (Mental Health Commission, 2011).

As found in the current study and in previous research, poor parenting skills, early parent-child attachment problems, parent mental health problems, divorce, domestic violence, and family conflict are some of the most important predictors for child behaviour problems and child disorders such as ADHD (Amato, 2000; Connor, 2002; DeVito & Hopkins, 2001; Das Banerjee et al., 2007; Erdman, 1998; Finzi-Dottan et al., 2006; Hjern, Weitoft, Lindblad, 2009; Johnston & Mash, 2001; Pierrehumbert et al., 2000). All of these factors constitute an area for early intervention for children who are at risk. Such interventions could include BPT, family therapy, referral to services such 'strengthening families', parent counselling, and/or support from adult mental health services. The IY and Triple-P BPT programmes are commonly used in New Zealand and have been associated with a reduction in a range of behaviour problems and ADHD symptoms (Bor, Sanders, & Markie-Dadds, 2002; Hartman, Stage, &Webster-Stratton, 2002; Sanders, 1999; Thomas & Zimmer-Gembeck, 2007; Webster-Stratton, Reid, & Hammond, 2004). Based on the efficacy of these programmes, it is important that mental health professionals, teachers, and other agencies actively participate in promoting these types of psychosocial interventions.

The introduction of Infant Mental Health Services (IMHS) in some district heath boards in New Zealand may prove to be helpful in targeting some of these early risk factors. IMHS's aim to provide specialist treatment for families with high needs, parent-child attachment difficulties, and parent mental health issues in order to prevent the child from developing ongoing mental health

difficulties. Depending on satisfactory efficacy evaluation, maybe there is a need for IMHS in all of the DHB's in New Zealand.

At a societal level, education around the relationship between adverse childhood experiences and child mental health problems is essential. Along with continuation of programmes that target child abuse, the de-stigmatization of mental health problems, and family violence to help reduce the occurrence of these events in the community. Some current programmes in New Zealand include the 'campaign for action on family violence' and the 'it is ok to ask for help campaign' (New Zealand Government, 2010).

Future Research

Ideally, there is a need for longitudinal research which follows a large population of children and adolescents from birth, into adolescence, and eventually adulthood. There is a need for well-designed studies that control for possible confounding factors. A key aspect of this longitudinal research would be documenting the timing of adverse childhood events, where possible. This way, the aetiological relationship between ADHD and adverse childhood experiences could be examined in closer detail.

Another avenue for future research would be to replicate the finding of a 'complicated ADHD' group. It would also be useful to include the symptoms of PTSD in the latent class analysis to see how the PTSD symptoms cluster with the ADHD, ODD, and CD symptoms. It would be helpful to examine a larger population of children with potential complicated ADHD as the sample size of the current study was relatively small. There is a need for research that looks closely at risk factors for developing complicated ADHD and how these may be identified.

Furthermore, it would also be helpful to follow-up the complicated ADHD group and the other groups identified by the latent class analysis to see if they differ in terms of treatment response, symptom trajectory, and long-term prognosis.

Following on from this study, the most helpful step would be to consider the efficacy of treatment for children with 'complicated ADHD', if replicated. It would be helpful to consider whether or not these children respond to common ADHD treatment used in New Zealand, such as pharmacological treatments and behavioural interventions. Due to the complexity of this group of children it can be predicted that these children may not respond to these standard ADHD treatments. These children most likely require a more holistic treatment approach which may include: treatment of co-morbid psychological problems, treatment of past trauma issues, parenting programmes, family therapy to target dysfunctional patterns of interaction within the family, and ongoing liaison with relevant services.

There is also a need for further research in CAMHS in New Zealand around the inquiry of adverse childhood events. Similar to adult chart review studies in New Zealand, it would be helpful to investigate the rates at which mental health professionals in CAMHS ask about abuse and respond to such disclosures (Agar & Read, 2002). It would also be helpful to look at what is done with this information and how abuse histories are incorporated into psychological formulations and treatment plans. Adding to this research, it would be helpful to introduce an initial assessment form into CAMHS that includes a mandatory section on adverse childhood experiences. The efficacy of introducing this new form could be investigated in the research.

Finally, there is a need for training of staff in CAMHS around the relationship between adverse childhood experiences and child mental health problems, how to inquire about adverse childhood experiences, and what to do with such disclosures. Further research could look at designing a training programme for staff working at New Zealand CAMHS that educates around these issues. Furthermore, it would be helpful to examine the efficacy of implementing such a training programme.

Limitations of Study

There are limitations of the current study that are important to keep in mind when interpreting the results. Most of the limitations are related to the study being a file audit study. This means that the findings from this study are based on information obtained from pre-existing clinical records.

The information gathered from each file was based on the clinical notes written by mental health professionals involved with the child or adolescent. The mental health professionals that wrote the clinical notes varied in their professional backgrounds and levels of expertise. For example, in the particular CAMHS investigated in this study some of the mental health professionals who worked at the service included: clinical psychologists, nurses, occupational therapists, social workers, psychiatrists, and 'clinical team members'. The variation of mental health professionals writing the clinical notes suggests that the quality, quantity, and nature of information recorded would have been highly variable from case to case. This suggests that the data collected from the clinical files may not have been consistent for each child and adolescent included in the study. Despite this inconsistency, there are factors that may have contributed to a certain degree of reliability between each clinical file. Each child or adolescent admitted to the service was reviewed in a multi-disciplinary team (MDT) meeting. In order to review a child or adolescents case in an MDT, the clinician could be expected to have information regarding the child or adolescents presenting problems, symptoms, tentative diagnosis, some developmental history and relevant family background. This suggests that the majority of files included in the present study would have had a certain degree of information covering these general areas.

Another potential limitation of the study is that the files would probably not have represented the true rates of adverse childhood events experienced by the children and adolescents included in the study (Agar & Read, 2002). It is likely that important information about adverse childhood events was missing from some of the files. This could be for a number of reasons. The

mental health professional may not have enquired about certain adverse life events. There may have also been some cases where the mental health professional did enquire about adverse life events, but the child, adolescent, or their family may not have disclosed this information. Disclosure of adverse childhood events may place the parent or caregiver at risk of a legal issue, or even loss of custody. This is particularly relevant for families with a history of involvement with CYFS and/or family court. Furthermore, there may have been some cases where the child, adolescent, or their family did not know of the adverse childhood event due to a lack of knowledge, or the child or adolescent not remembering the event. Finally, there may have been some cases where the mental health professional was aware of an adverse childhood experience, but chose not to document this information in the clinical file. Therefore, it can be hypothesized that the information gathered from the files regarding the rates of abuse and adverse life experiences was an under representation of the actual rates of childhood adverse events. This is similar to previous research investigating the relationship between adverse experiences and mental health problems, which has consistently concluded that experiences of abuse and trauma are unreported (Briscoe-Smith & Hinshaw, 2006; Read, 2007; Read & Fraser, 1998; Read, Van Os, & Morrison, 2006; Wurr & Partidge, 1996).

Despite research demonstrating low rates of enquiry, clinicians working in New Zealand CAMHS are generally expected to ask about abuse and adverse life experiences. Furthermore, clinicians are obligated by their various codes of ethics to involve agencies such as CYFS when a child is an unsafe situation and/or at risk of being harmed. This obligation suggests that clinicians working in CAMHS are aware of the importance of enquiring about past and current adverse childhood experiences. This suggests that for the many of the cases included in the present study, the mental health professionals involved would have at least enquired about adverse childhood experiences and included this information in the child or adolescents clinical file.

Another important issue to consider is the potential lack of consistency among mental health professionals around what is deemed 'abuse' or an 'adverse experience'. The information provided

in the notes may have been biased by the subjective opinion of each clinician around what they viewed as 'abusive' or 'non-abusive'. For example, one clinician may have viewed a 'smack on the hand' as 'physical abuse', where as another clinician may not see a 'smack on the hand' as harmful and may choose to exclude this information from the notes. While it was impossible to control for these factors, it is possible they may have impacted on the data that was recorded in the clinical files and thus the outcomes of the current study.

Another limitation in the study was that some important information was unavailable in the files. For example, information regarding the age of the child when adverse events took place was not always available in the file. Therefore, the time-line of abuse could not be included as a variable. This made it difficult to make any causal links regarding adverse life experiences and aetiology of ADHD.

Another issue that is important to consider is the generalisablity of the results. Firstly, the study only included children and adolescents that were clients at a New Zealand CAMHS. This means that the findings were not compared to the general population and cannot by generalised beyond the CAMHS sample. Furthermore, the sample excludes children and adolescents that did not have any 'face to face' contact with the service and that may have been difficult to engage. These particular children and adolescents may have differed in their symptom profiles and rates of adverse events. Furthermore, it can be assumed that children in community populations or psychiatric inpatient populations may differ in their symptom profiles and rates of adverse life experiences. This suggests that the findings of from this study may not be representative of all children and adolescents.

A final limitation of the study is the small number of children and adolescents in the study with a diagnosis of ADHD, and the small size of the complicated ADHD group. In order to improve on this limitation, further research could investigate a larger sample of children and adolescents with symptoms and/or diagnoses of ADHD.

Despite the various methodological limitations, the study methodology included some considerable strengths. The study may have been limited by the personal subjectivity of what the principal researcher and her supervisor included or excluded as an adverse childhood experience. However to help limit this bias, the researchers in the study went through a stringent inter-rater reliability check process. This helped to reduce any potential rater bias of what was included in the study as a symptom or adverse event.

Furthermore, the files used in the study offered very detailed and comprehensive information regarding the child or adolescents presenting problems, history, and treatment. Every file was read in its entirety, this included clinical notes, forms, and assessment sheets. This provided a very rich and comprehensive source of data. Additionally, the fact that there was no face to face contact with clients had its strengths. This meant that the results were not affected by interviewer bias or self-report bias (Podsakoff, MacKenzie, Lee, & Podsakoff, 2003).

CONCLUSIONS

ADHD is currently the most commonly diagnosed psychiatric disorder among school aged children and one of the most researched (APA, 2000; Brown et al., 2001; Hodgkins, et al., 2012; Mayes, Bagwell, & Erkulwater, 2008). Not only is the disorder wide-spread among school aged children, the number of cases worldwide appear to be dramatically increasing (Brown et al., 2001; Coleman, 2007; Hodgkins, et al., 2012; Mayes, Bagwell, & Erkulwater, 2008; Radcliffe & Timimi, 2004; Taylor, 2009; Timimi & Taylor, 2004). ADHD is of particular concern due to the significant negative sequelae associated with the disorder, including psychiatric, occupational, social, and academic problems later in life (Briscoe-Smith & Hinshaw, 2006; Goldstein & Ellison, 2002).

Despite the high prevalence rates and overall poor prognosis of ADHD, there are on-going issues around what constitutes 'ADHD' and how the disorder is defined and diagnosed (Nevid, et al., 2006; Radcliffe & Timimi, 2004; Rappley, et al., 1995; Taylor, 2009; Timimi et al., 2004).

Further complicating a diagnosis of ADHD is the fact that individuals with ADHD are a highly heterogeneous group who show great variance in not only their types of symptoms, but in the perseverance and severity of their ADHD (Connor, 2002; Nigg, 2006). This creates a complex aetiological picture and the cause of the disorder remains a topic of controversy (Barkley, 1997; Goldstein & Ellison, 2002; Mash & Barkley, 2003; Nigg, 2006; Steinhausen, 2009).

Various studies have found an association between child maltreatment, neglect, physical abuse, sexual abuse and the symptoms of ADHD (Briscoe-Smith and Hinshaw, 2006; Endo, Sugiyama, & Someya, 2006; Famularo, Fenton, & Kinscherff, 1993; Kendall-Tackett, et al., 1993; McLeer et al., 1994). Despite many studies finding an association between adverse childhood events and ADHD, this relationship is complex and there are many conflicting hypotheses about the nature and direction of this relationship (Findling, et al., 2007; Szymanski, et al., 2011).

The current study found a significant relationship between the symptoms of ADHD and adverse childhood experiences, which supports a substantial number of international studies (Conway, Oster, & Szymanski et al., 2011; Endo, et al., 2006; Famularo, Fenton, & Kinscherff, 1993; Famularo, et al., 1996; Kendall-Tackett, et al., 1993; McLeer et al., 1994; McLeer, et al., 1998; Merry & Andrews, 1994; Stern, et al., 1995). The current study also found a significant relationship between the symptoms of other externalising behaviour problems (ODD and CD), PTSD, and adverse childhood experiences.

Although the design of the current study doesn't facilitate definite conclusions about causality, the findings support that early adverse childhood experiences place children and adolescents at significant risk of developing ongoing issues with inattentiveness, hyperactivity and impulsivity. In the context of the many previous, similar findings, it seems reasonable to conclude that adverse childhood experiences play an aetiological role in the development of ADHD in children and adolescents.

This study also provides preliminary evidence that there may be a group of children and adolescents with 'complicated ADHD', who have more serious externalizing symptoms (i.e. high levels of ADHD, ODD and CD symptoms) and a higher rate of diagnostic co-morbidity with other *DSM-IV* disorders. This group experienced a higher average rate of adverse childhood events, along with a higher rate of physical abuse and exposure to domestic violence, compared to the other children and adolescents with symptoms of ADHD. This suggests that early adverse experiences may create a more complicated profile of ADHD symptoms and perhaps impact the way that ADHD symptoms manifest.

It is hoped that this study will help to increase awareness of the relationship between ADHD and adverse childhood experiences. It is also hoped that the findings from this study will help to influence the current predominantly medical understanding of ADHD and draw attention to the many psychosocial factors that play a role in the aetiology of the difficulties that result in a diagnosis of ADHD.

Having a clearer understanding of the cause of ADHD will hopefully lead to more comprehensive initial assessments that carefully screen for adverse childhood experiences in children and adolescents referred for an assessment/diagnosis of ADHD. Despite the clear relationship between adverse life experiences and mental health problems in children and adults, many mental health professionals do not commonly enquire around such topics. This is alarming when considering that over half of the children and adolescents in the current study experienced three or more adverse childhood experiences.

It is also hoped that having a clearer understanding of the cause of ADHD will help steer towards a clearer diagnostic system. The highly heterogeneous nature of ADHD as a diagnosis is problematic. In addition, there is ongoing concern around the high overlap between ADHD and PTSD symptoms in some children, particularly for those children who may have a more 'complicated' form of ADHD. For these children, a diagnosis of ADHD may not necessarily be

helpful. This is mainly because a child who has experienced adverse events and/or trauma would require specific interventions to reduce the psychological impact of their trauma, as opposed to the standard treatment that is provided for ADHD. It seems that the *DSM-IV* artificially categorises children into diagnostic groups which may wrongly imply biological (ADHD) or psychosocial/trauma (PTSD) aetiologies. Therefore, it would seem that a diagnosis of 'ADHD' may imply that it is a biologically based 'disorder', as a opposed to a problem that may have been caused by social factors. In contrast, a focus on specific problems or symptoms may be more useful clinically, and in terms of causation research. It is clear that there is an urgent need for clarification around how we define, understand, and diagnose ADHD.

Finally, it is hoped that having a clearer understanding of the relationship between adverse childhood experiences and ADHD will highlight the need for holistic and individually tailored treatment plans that go beyond pharmacological options. This is particularly relevant for children with a pervasive history of adverse experiences who require specific interventions to help in reducing the psychological impact of these adverse experiences.

APPENDIX A:

DSM-IV-TR DIAGNOSTIC CRITERIA FOR ATTENTION-DEFICIT/HYPERACTIVITY DISORDER

(American Psychiatric Association, 2000)

Attention-Deficit/Hyperactivity Disorder

A. Either (1) or (2):

1) Six (or more) of the following symptoms of *inattention* have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

Inattention:

   a) Often fails to give close attention to details or makes careless mistakes in schoolwork, work, or other activities.
   b) Often has difficulty sustaining attention in tasks or play activities.
   c) Often does not seem to listen when spoken to directly.
   d) Often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (not due to oppositional behaviour or failure to understand instructions).
   e) Often has difficulty organizing tasks and activities.
   f) Often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (such as schoolwork or homework).
   g) Often loses things necessary for tasks or activities (e.g., toys, school assignments, pencils, books, or tools).
   h) Is often easily distracted by extraneous stimuli.
   i) Often forgetful in daily activities.

2) Six (or more) of the following symptoms of *hyperactivity-impulsivity* have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

Hyperactivity:

   a) Often fidgets with hands or feet or squirms in seat.
   b) Often leaves seat in classroom or in other situations in which remaining seated is expected.
   c) Often runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults, may be limited to subjective feelings of restlessness).
   d) Often has difficulty playing or engaging in leisure activities quietly.
   e) Is often "on the go" or often acts as if "driven by a motor".

f) Often talks excessively.

Impulsivity :

g) Often blurts out answers before questions have been completed.
h) Often has difficulty awaiting turn.
i) Often interrupts or intrudes on others (e.g., butts into conversations or games).

B. Some hyperactive-impulsive or inattentive symptoms that caused impairment were present before age 7 years.

C. Some impairment from the symptoms is present in two or more settings (e.g., at school [or work] and at home).

D. There must be clear evidence of clinically significant impairment in social, academic, or occupational functioning.

E. The symptoms do not occur exclusively during the course of a Pervasive Developmental Disorder, Schizophrenia, or other Psychotic Disorder and are not better accounted for by another mental disorder (e.g., Mood Disorder, Anxiety Disorder, Dissociative Disorder, or a Personality Disorder).

Code based on type:

> **314.01 Attention-Deficit/Hyperactivity Disorder, Combined Type:** if both Criteria A1 and A2 are met for the past 6 months

> **314.00 Attention-Deficit/Hyperactivity Disorder, Predominantly Inattentive Type:** if Criterion A1 is met but Criterion A2 is not met for the past 6 months

> **314.01 Attention-Deficit/Hyperactivity Disorder, Predominantly Hyperactive-Impulsive Type:** if Criterion A2 is met but Criterion A1 is not met for the past 6 months

> *Coding note: For individuals (especially adolescents and adults) who currently have symptoms that no longer meet full criteria, "In Partial Remission" should be specified.

APPENDIX B:
DATA COLLECTION FORM

DATA COLLECTION FORM

*This information is **strictly** confidential. If found **please** contact: Dr John Read on 373 7599 ext 85011 or Rachael Mayne on 027 2180764 or 3737599 ext 86755*

NHI No#	Date of Birth	Age at Admission	Ethnicity	
Date Opened	**Date Closed**	**No# days at service**	**Gender**	
			M F	
Case Manager		**Stream at Kari Centre**		
1. Job Title:	M F	**1.**		
2. Job Title	M F	**2.**		

Diagnosis	
Previous Diagnosis?	NO YES:
Recent Diagnosis?	

DSM-IV-R	
Axis I	
Axis II	
Axis III	
Axis IV	**GAF:**

Alcohol	Drugs	Medication

Suicide Risk				
Self	Low	Medium	High	?
Others	Low	Medium	High	?
Deliberate Self-harm	Y N ?	**Suicide Attempt**	Y N M ?	

Family Assessment? Y N		Individual Assessment? Y N	
1. Job Title	M F	**1. Job Title**	M F
1. Job Title	M F	**1. Job Title**	M F

ADHD		
Inattention:	**Hyperactivity:**	**Impulsivity:**

Conduct Disorder	
Aggression to people and animals:	**Deceitfulness or Theft:**
Destruction of Property:	**Violations of Rules:**

Oppositional Defiant Disorder
Negative, Hostile and Defiant Behaviour:

Post Traumatic Stress Disorder	
Traumatic Event?	**Re-experienced?**
Avoidance and numbing?	**Increased arousal?**

Mood:	**Anxiety/Phobias**
Unipolar Bipolar	Panic Phobia Obsessions Compulsions

Eating	**Learning Difficulty/ Pervasive Developmental Dis**
Binging Compensatory Restricting	

Psychosis	**Other Significant Symptoms**
Hallucinations Delusions	

Aversive childhood Experiences		Treated
Sexual Abuse	Y N ?	✔ X ?
Assessment?		
Physical Abuse	Y N ?	✔ X ?
Assessment?		
Emotional Abuse	Y N ?	✔ X ?
Assessment?		
Emotional Neglect?	Y N ?	✔ X ?
Assessment?		
Physical Neglect	Y N ?	✔ X ?
Assessment?		
Chaotic Family Environment	Y N ?	✔ X ?
Assessment?		
Greif/Loss	Y N ?	✔ X ?
Assessment?		
Parental Discord	Y N ?	✔ X ?
Assessment?		
Parental Mental Illness?	Y N ?	✔ X ?
Assessment?		
Impoverishment	Y N ?	✔ X ?
Assessment?		
Other Aversive Childhood Event	**Specify:**	✔ X ?
Assessment?		

NOTES

APPENDIX C:

DEFINITIONS OF SYMPTOMOLGY

Definitions of Symptomology

The definitions for the symptoms recorded in the present study will be based on those in the *DSM-IV-TR* (APA, 2000). Due to the limited information available in many of the files, the present study assumes the clinician's judgment is correct and statements and descriptions in the file that clearly suggest any of the following symptoms will be included. For example, in the 'inattentiveness' criteria the following would be included as a symptom: "X has difficulty concentrating and is often preoccupied in class" or "X always losing things and unorganized: he loses clothes, laptop and homework". However, due to the nature of the present study, many of the symptoms listed in the files are unclear or vague. For these symptoms, at least two are needed to fulfil the criteria. For example, in the 'inattentiveness' criteria, "difficulty concentrating", "distractible" or "fidgety" do not clearly fulfil the 'inattentive' criteria and at least two of the these symptoms are needed in order to be included under this criterion. This rule applies to all of the symptoms listed below.

All of the following symptoms are considered relative to the age of the participant. For example, a five year old child who talks to an imaginary friend is perhaps not included under the symptom 'hallucinations'. Comparatively a 17 year old presenting with the same behaviour would be included under this symptom.

Inattentiveness (ADHD1)

The child/adolescent fails to give close attention to detail and makes careless mistakes in school work or other activities. The child/adolescent has difficulty sustaining attention in tasks or play activities and often doesn't listen when spoken to directly. The child/adolescent often has difficulty organizing tasks and activities and is reluctant to engage in tasks that involve sustained mental effort such as homework. Often inattentive children lose things that are necessary for activities, are easily distracted by extraneous stimuli, and forgetful in daily activities.

Examples that were considered symptoms of 'inattentiveness':

"Fidgety, easily distracted, and struggles to finish tasks".

"Low concentration level in class and has trouble listening when spoken to directly".

"Short concentration span. Can't focus on tasks at school and has trouble following instructions".

Examples that were not considered symptoms of 'inattentiveness':

"Difficulty concentrating".

"Inattentive".

"Limited attention span".

Hyperactivity (ADHD2)

The child/adolescent often fidgets with hands, squirms in seat, and leaves seat in classroom or at dinner table. The child/adolescent runs about excessively in situations which are inappropriate and often acts as though they are 'driven by a motor'. The child/adolescent talks excessively and has difficulty playing quietly.

Examples that were considered symptoms of 'hyperactivity':

"Very active and can't keep still".

"Full on, one hundred miles an hour as though he is driven by a motor"

"Run's around and yells around the classroom. A very disruptive child".

"Always hyperactive at home and at school".

"Challenging child – interrupts class".

Examples that were not considered symptoms of 'hyperactivity':

"Talks very fast".

"Full on child".

Impulsivity (ADHD3)

Impulsive behaviour characterized by blurting out answers before the question has been completed, has difficulty awaiting turn, often interrupts or intrudes on others. The child/adolescent often does not think before they act out.

Examples that were considered symptoms of 'impulsivity':

"Poor impulse control and no concept of danger".

"Often impulsive, does things without thinking and often hurts himself".

"Extremely impulsive".

Examples that were not considered symptoms of 'impulsivity':

"Poor decision making".

"Impulsive".

Aggression to people and animals (CD1)

The child/adolescent often bullies, threatens, intimidates, or physically hurts others. This includes initiating fights, using weapons, and being physically cruel to people or animals. The child/adolescent may have stolen while confronting a victim or forced someone into sexual activity.

Examples that were considered symptoms of 'aggression to people or animals':

"Offences of a sexual nature".

"Picks fights, bully's others at school, and kicks and punches other students".

"Anger outbursts – swears at staff at school. Has assaulted staff members in the past".

"Gets into fights – police involvement".

"Kicks and bites his mum, is angry and throws things, goes 'berserk' – is out of control and threatens to jump out of the window".

"Physically violent to mum – bruised her arm and swears at her. Has a lack of empathy and remorse".

"Aggressive and violent to peers – scratches and pinches"

Examples that were not considered symptoms of 'aggression to people or animals':

"Hit's other children".

"Anger control problems".

Destruction of Property (CD2)

The child/adolescent has deliberately engaged in fire setting with the intention of causing serious damage. Or the child/adolescent has deliberately destroyed others property.

Examples that were considered symptoms of 'destruction of property':

"Goes out and tags buildings".

"Smashed a chair through his parents ranch slider".

"Has smashed a window at home and trashed his own room – he goes berserk".

"Went through a phase of pyromania".

Examples that were not considered symptoms of 'destruction of property':

"Tagging".

"Breaks toys and throws food (age 5 years)".

Deceitfulness or theft (CD3)

The child/adolescent has broken into someone's house, building, or car. The child/adolescent often lies to obtain goods or favours to 'con' people. The individual has stolen items of non trivial value without confronting a victim, for example shoplifting or forgery.

Examples that were considered symptoms of 'deceitfulness or theft':

"Stole from a teacher this year and stole his friend's i-pod".

"Frequently telling lies and stealing from others".

"Often lies and is manipulative".

Examples that were not considered symptoms of 'deceitfulness or theft':

"Steels lunchboxes from peers".

"Ran away from his mother for a day".

"Lies to his parents".

Serious violation of rules (CD4)

The child/adolescent often runs away from home or stays out all night despite parental prohibitions before 13 years of age. The individual is often truant from school.

Examples that were considered symptoms of 'serious violation of rules':

"Frequently runs away from school and goes out for the day and won't come home".

"Doesn't follow rules and no social justice. Has no respect for authority".

"Frequently run's out of the classroom. At home she runs out of house and mum has to chase her".

"Sneaks out at night to go out".

Examples that were not considered symptoms of 'Serious violation of rules':

"Fails to adhere to medication".

"History of truancy from school".

Non-compliant/defiant (ODD1)

The child/adolescent often actively defies or refuses to comply with adult requests or rules. The child/adolescent often argues with adults and won't listen. The child/adolescents parents cannot control him/her.

Examples that were considered symptoms of 'non-compliant/defiant':

"He is non-compliant with adults, argues with authority, and won't listen to his parents".

"X is non compliant at school – he has no respect for authority".

"Demanding to parents – talks back to them. Has tantrums when mum is leaving".

"He is uncontrollable".

"Always getting into trouble".

"Back-chatting and defiant".

Examples that were not considered symptoms of 'non-compliant/defiant':

"Breaks rules".

"Shouts and has tantrums when he doesn't get his own way".

"Very bossy".

Oppositional (ODD2)

The child/adolescent often loses their temper, deliberately annoys others, often blames others for his/her mistakes, and is often touchy or annoyed by others. This individual is often angry, resentful, vindictive, and spiteful towards others.

Examples that were considered symptoms of 'oppositional':

"Has a low tolerance for frustration, is often angry and aggressive, and shows little remorse after getting into fights at school".

"Very angry. Shouts and gets angry at friends".

"Angry, aggressive, and oppositional at school and at home".

Examples that were not considered symptoms of 'oppositional':

"Easily annoyed".

"Yells at and bullies younger sister".

Re-experiencing (PTSD1)

The child/adolescent has experienced a traumatic event and is re-experiencing the incident. This includes flashbacks, nightmares, intrusive thoughts and recollections, and acting or feeling as

though the event were recurring. The individual may experience intense psychological distress in response to external stimuli that are associated with the traumatic event. The symptoms of 're-experiencing' must be associated with a traumatic event in order to be included in the present study under this criterion.

Examples that were considered symptoms of 're-experiencing':

"Remembers painful events, has violent nightmares and wakes up shaking".

"Toss's and turns in sleep – dreams of sexual abuse".

"Flashbacks of father's death".

Examples that were not considered symptoms of 're-experiencing':

"Lot's of bad dreams".

"X has intense nightmares".

Avoidance/numbing (PTSD2)

The adolescent/child is persistently avoiding stimuli associated with the traumatic event and experiencing a numbing of general responsiveness. This includes avoidance of associated thoughts, feelings, conversations, activities, places, or people. Individuals may not remember an important aspect of the trauma. Individuals may feel detached or estranged from others, have a restricted range of affect, or a sense of a foreshortened future.

Examples that were considered symptoms of 'avoidance/numbing':

"Feels 'disconnected' in relation to being beaten severely".

"X is avoiding men".

"X cannot remember being assaulted".

Examples that were not considered symptoms of 'avoidance/numbing':

"X has not been herself".

Increased arousal (PTSD3)

The child/adolescent experiences persistent symptoms of increased arousal. This includes an inability to fall or stay asleep, irritability, anger outbursts, difficulty concentrating, hyper vigilance, or an exaggerated startle response. For the purpose of the present study, symptoms of 'increased arousal' must be associated with an experienced traumatic event in order to be included under these criteria.

Examples that were considered symptoms of 'increased arousal':

"High irritability and agitation".

"Feels increasingly irritable, angry, and frustrated. Is having problem concentrating (since being raped)".

Examples that were not considered symptoms of 'increased arousal':

"Feels restless".

"Problems concentrating".

Low Mood (MOOD1)

The individual is depressed most of the day, nearly every day, for at least two months. This includes feeling of emptiness, sadness, hopelessness, worthlessness, and tearfulness. For the purpose of the present study, this criterion requires either two descriptors of 'low mood' such as: 'feels low', 'tearful' or 'sad' or an additional descriptor suggestive of severity, duration, or frequency such as: 'sad all of the time', 'very depressed', or 'tearful every day'.

Examples that were considered symptoms of 'low mood':

"Very low mood – rates as 0/100".

"Tearful, depressed, and sad all of the time".

"Low mood, sad, and depressed".

"Cries at school and is withdrawn".

Examples that were not considered symptoms of 'low mood':

"Feels low".

"Negative thinking".

"Upset by small things".

Other Depressive Symptoms (MOOD2)

The child/adolescent experiences serious symptoms in relation to a change in mood. This includes suicide ideation/intent, diminished interest in previously enjoyable activities, lack of motivation, sleep difficulty, significant weight loss or gain relating to a change in mood, fatigue, and a diminished ability to concentrate. For the purpose of the present study, this criteria requires at least two other symptoms of depression in order to be included under this criterion. However, if the file mentions suicide ideation or intent, this is sufficient alone in order to be included under this category.

Examples that were not considered symptoms of 'other symptoms of depression':

"Sleeps for over 12 hours – has problems with his sleep cycle. He also has had an increase in appetite".

"Can't get to sleep until 2 am and stays in bed all day. Feels low on energy".

"Poor sleep and change in eating".

"X feel suicidal".

"Stopped all activities and decreased appetite".

Examples that were not considered symptoms of 'other symptoms of depression':

"Trouble sleeping – wakes up shaking".

"Low self esteem".

"X hates school and says he wants to die if he has to go".

Emotional Dys-regulation (MOOD3)

The child/adolescent experiences affective instability due to a marked reactivity of mood (e.g. dysphoria, irritability, or anxiety that usually lasts for a few hours and only rarely more than a few days). The individual experiences chronic feelings of emptiness, along with intense anger, and difficulty controlling emotions. This includes frequent displays of temper, anger, and arguments. Due to the nature of the present study and the limited detail available in many of the files, descriptors such as 'emotionally dys-regulated', 'difficulty controlling emotions' and 'intense emotions' will be included under this criteria. However, symptoms that are unclear and perhaps vague, such as 'mood swings' require an additional descriptor of severity, duration, or frequency, such as 'frequent mood swings' or 'severe mood swings' in order to be included.

Examples that were considered symptoms of 'emotional dys-regulation':

"Emotional deregulation".

"Mood regulation problems".

"Overwhelmed by emotions – distraught and unhappy. Has emotional outbursts. Marked affective dys-regulation".

Examples that were not considered symptoms of 'emotional dys-regulation':

"Mood swings".

"Changeable moods".

"Mood is up and down".

Mania (MOOD4)

The child/adolescent experiences manic symptoms including: an inflated sense of self, grandiosity, decreased need for sleep, and distractibility. The individual is also more talkative than normal, has flights of ideas, has excessive involvement in pleasurable activities (e.g. shopping sprees, sexual behaviour, or foolish business investments), and an increase in goal directed activity.

Examples that were considered symptoms of 'mania':

"Manic symptoms – grandiose ideas and periods of excessive energy".

"Periods of going at 100 miles per hour. Reduced need for sleep, high physical activity and very intense conversations".

Examples that were not considered symptoms of 'mania':

"Grand ideas".

Excessive and persistent worry (ANX1)

The child/adolescent experiences excessive worry about a number of events or activities, that occurs more days than not. The individual finds it difficult to control the worry and experiences associated symptoms such as restlessness, sleep difficulty, muscle tension, and difficulty concentration. 'Excessive and persistent worry' also has an additional requirement similar to 'low mood'. This criterion requires either: two descriptors of 'excessive and persistent worry' such as: 'anxious', 'fearful', 'worries', or 'ruminates', or an additional descriptor suggestive of severity, duration, or frequency such as: 'anxious every day', 'has many excessive fears', or 'has severe anxiety'.

Examples that were considered symptoms of 'excessive and persistent worry':

"Pronounced anxiety and high stress 100% of the time. Has a high heart rate, sweaty palms, and anxious thoughts ruminating through his mind".

"Has many fears. Is scared of the dark and has to sleep with a night light on (X is 17 years)".

"Anxious and worried a lot about death and going to jail. Worries in bed and this makes him cry".

"Always scared. Won't sleep in the dark".

Examples that were not considered symptoms of 'excessive and persistent worry':

"Anxiety about going to school".

"Anxiety symptoms".

"Worrying/ catastrophises about future".

Phobia (ANX2)

The child/adolescent experiences excessive fear in relation to a specific object of situation. Exposure to the feared object or situation evokes an immediate anxiety response which may involve a panic attack. The individual recognizes that the fear is excessive or unreasonable. The phobic stimuli is avoided or endured with intense distress.

Examples that were not considered symptoms of 'phobia':

"Is scared of spiders, snakes and animals".

"School phobia – won't play sport"

"Phobia of being spoken to or touched by strangers. Can't talk to other people outside of the family. Has a sore tummy when tries to talk to strangers".

"Phobia of vomiting – very scared of vomiting. Not attending school due to this anxiety".

Examples that were not considered symptoms of 'phobia':

"Scared of the dark" (age 8 years).

"Fearful of everything"

Compulsions (ANX3)

Compulsive behaviour including repetitive behaviours (e.g. hand washing, ordering, checking), or mental acts (e.g. counting, praying) that the person feels driven to act out in response to an obsession or in accordance to rigid rules. The acts and rituals are aimed at reducing distress or preventing a dreaded event or situation, even though the behaviours are not connected in a realistic way. The individual recognizes the behaviours are excessive or unreasonable.

Examples that were considered symptoms of 'compulsions':

"Ritual behaviours – at bedtime has to have the light on, door open and used blanket as a barrier. Has done this as long as she can remember. X also covers all of the plugs in the bathroom with towels".

"Rubs hands often. Picks up football with sleeves over hands".

"Washes hands until raw. Stacks and organizes shampoos. Is always checking – has rituals such as turning the lights on and off".

"Compulsions to twitch. Cleans all of the time and feels agitated if she doesn't tidy".

Examples that were not considered symptoms of 'compulsions':

"Counts his steps".

Obsessions (ANX4)

The adolescent/child experiences recurrent and persistent thoughts, impulses or images that are experienced as intrusive and cause distress. These thoughts are not simply excessive worries about real life problems. The individual attempts to ignore or neutralize the thoughts with some other action or thought. The individual recognizes the thoughts are impulses are a product of their own mind.

Examples that were considered symptoms of 'obsessions':

"Obsessive about doors being locked and fussy about cleanliness".

"Obsessed with cleanliness, order, and has rituals around this".

Examples that were not considered symptoms of 'obsessions':

 "Obsessed with her hair".

"Obsessed with political injustices".

Panic Symptoms (ANX5)

The individual experiences intense periods of fear and discomfort with some of the following symptoms present: increased heart rate, palpitations, shaking, short of breath, chest pain or tightness, nausea, light headedness, depersonalization, or hot flushes. The individual also has an intense hear of losing control or dying during the panic.

Examples that were considered symptoms of 'panic':

"Has frequent panic attacks. Has 6-7 every fortnight. Describes feeling shaky, short of breath and has headaches".

"Panic attacks – can't breathe and shakes".

Has anxious feelings – and feels tight in chest, and increase in heart rate and short of breath"

"Frequent panic attacks".

Examples that were not considered symptoms of 'panic':

"Panicky at his hostel".

"Stressed 100% of the time".

"Heart races when anxious".

Separation Anxiety (ANX6)

The child/adolescent exhibits developmentally inappropriate and excessive anxiety when separated from a primary attachment figure. This includes persistent worry and anxiety around the anticipation of separation, losing the attachment figure, the person being harmed, an event will occur that leads to separation, and reluctance to go to school due to separation. The individual may experience nightmares relating to separation anxiety and physical symptoms such as headaches and vomiting when separated from the person.

Examples that were considered symptoms of 'separation anxiety':

"Separation anxiety – took three weeks to settle at school".

"Doesn't like leaving mum – he has tantrums when she leaves. He is clingy to his mum."

"Separation anxiety with mum".

"Scared to go to kindy and has separation anxiety with mum".

Examples that were not considered symptoms of 'separation anxiety':

"Clingy to parents'.

Social Anxiety (ANX7)

The child/adolescent has a persistent fear of one or more social or performance situations. The individual fears that they will act in a particular way that will be humiliating or embarrassing. Exposure to the feared situation evokes an intense anxiety response.

Examples that were considered symptoms of 'social anxiety':

"X has social anxiety".

"Social phobia – group work and hates going to school. Has problems socializing".

"Social anxiety – extreme shyness and very quiet. Doesn't like meetings, school and public places".

Examples that were not considered symptoms of 'social anxiety':

"Shy – lack of communication".

"Quiet and shy".

Restrictive eating (EAT1)

The child/adolescent is engaging in recurrent and inappropriate restrictive behaviours in order to reach or maintain a lower than average body weight. This includes starvation, fasting, strict dieting, calorie counting, and exercise that is excessive and persistent.

Examples that were considered symptoms of 'restrictive eating':

"Restricts eating. Periods of food refusal and over-exercising".

"Fluctuates between restricting and binging. Exercises excessively".

Examples that were not considered symptoms of 'restrictive eating':

"Often unwell and can't eat for days at a time".

"Eating has increased".

Compensatory and purging behaviour (EAT2)

The individual engages in appropriate compensatory behaviour in order to prevent weight gain. This includes self induced vomiting, the use of laxatives, diuretics, enemas, or other medications.

Examples that were considered symptoms of 'compensatory and purging behaviour':

"Bulimic – causing medical problems".

"Purging behaviour".

Examples that were not considered symptoms of 'compensatory and purging behaviour':

"Erratic appetite".

"Patchy eating".

Binging behaviours (EAT3)

The individual engages in recurrent episodes of binge eating. An episode of binge eating is characterized by eating an amount of food in a discrete period of time that is definitely larger than most people would eat in a similar time period. There is a sense of lack of control over the eating during the binging.

Examples that were considered symptoms of 'binging behaviour':

"Binging four times a day".

"Spends all day eating and binging".

"Has started eating excessively".

Examples that were not considered symptoms of 'binging behaviour':

"Over – eating".

Distorted body image/intense fear of gaining weight (EAT4)

The child/adolescent experiences an intense fear of gaining weight or becoming fat, despite being underweight. The individual's perception of their body weight, size and shape is distorted and sometimes there is denial of the seriousness of the low body weight. The individual's self evaluation is unduly influenced by their body shape and size.

Examples that were considered symptoms of 'Distorted body image/intense fear of gaining weight':

"Body dysmorphia – doesn't like any part of her body".

"Extremely negative body image".

Examples that were not considered symptoms of 'Distorted body image/intense fear of gaining weight':

"Poor body image".

"Worried about appearance".

"Unhappy with body weight".

Mental Retardation (LEARN1)

Mental retardation is characterized by significantly sub-average intellectual functioning before the age of 18 years. The individual also experiences concurrent deficits in adaptive functioning.

Examples that were considered symptoms of 'mental retardation':

"Cannot dress herself and still not properly toilet trained at the age of 5 years old. Cannot use a knife or fork".

Examples that were not considered symptoms of 'mental retardation':

"Engages in childlike play".

Communication problems (LEARN2)

The individual experiences difficulty in speech or language and this includes problems in expressive language, articulation of words, and stuttering.

Examples that were considered symptoms of 'communication problems':

"Speech and language delay".

"Speech problems".

Examples that were not considered symptoms of 'communication problems':

"Problems with handwriting".

Learning difficulties (LEARN3)

The individual experiences academic functioning that is substantially below what is expected of the child/adolescents age, measured intelligence, and age appropriate education. This includes problems in reading, mathematics, written expression, and other learning areas.

Examples that were considered symptoms of 'learning difficulty':

"Academic delay and delayed language development".

"Borderline cognitive functioning"

"Learning disability – dyspraxia".

"Delayed cognitive ability and dyspraxia"

Examples that were not considered symptoms of 'learning difficulty':

"Mum reports a learning difficulty".

"Mum questions whether X has dyslexia".

"Behind at school" .

Deficits in social interaction (ASD1)

The child/adolescent exhibits a qualitative impairment in their interaction with others. This is manifested by deficits in eye to eye gaze, facial expression, and gestures to regulate social interaction. The individual fails to develop appropriate peer relationships and shows a lack of spontaneity to share enjoyment, interests, or achievements with other people and a lack of social reciprocity.

Examples that were considered symptoms of 'deficit in social interaction':

"Relationship difficulties and egocentrism".

"No empathy or social skills. Has difficulty making friends and has no friends at school".

"Likes to play alone and won't play interactively".

"Isolated, has limited peer interactions and no imaginative play. X doesn't make eye contact and won't initiate conversation".

Examples that were not considered symptoms of 'deficit in social interaction':

"Isolates himself – no friends".

"Reduced eye contact".

Qualitative impairments in communication (ASD2)

The individual experiences a delay or total lack of the development of spoken language. In children/adolescents with adequate speech, there is a marked impairment in the ability to initiate or sustain a conversation with others. The individual shows a stereotyped and repetitive pattern of language or idiosyncratic language.

Examples that were considered symptoms of 'qualitative impairments in communication':

"Repetitive loud noises".

"Speech and language delay".

"Delayed language development".

Examples that were not considered symptoms of 'qualitative impairments in communication':

"Not very talkative".

"Poor eye contact".

Stereotypical and repetitive patterns of behaviour (ASD3)

The child/adolescent has an encompassing pre-occupation with one or more stereotyped and restricted patterns of interest that is abnormal either in intensity or focus. The individual is highly inflexible to changes in routines or rituals and may have preoccupation with parts of objects. The individual may exhibit repetitive motor mannerisms.

Examples that were considered symptoms of 'stereotypical and repetitive patterns of behaviour':

"Jerks his head, hand flapping activity, unusual gait, and bangs things".

"Fussy with food and eating and particular about mess. Is ritualistic in routines".

"Particular and inflexible. Used to line up his cars at preschool. He likes order".

"Hand flapping. Is fixated on animals and vehicles".

"Sensitive to loud sounds, doesn't cope well with change".

"Repetitive speech and behaviour. Likes a set routine and can't cope with change".

Examples that were not considered symptoms of 'stereotypical and repetitive patterns of behaviour':

"Black and white thinking".

"Fixated with gardening".

Hallucinations (PSYCHOSIS1)

The child/adolescent experiences hallucinations that can occur in any sensory modality (e.g. visual, auditory, olfactory, gustatory, and tactile). For example, this can be experienced as hearing voices that are perceived as distinct from the persons own thoughts.

Examples that were considered symptoms of 'hallucinations':

"Hearing the devils voice".

"Hearing voices in his head about suicide".

"X see's 'spirits' around the house at night".

"Responding to non apparent stimuli – acutely psychotic".

Examples that were not considered symptoms of 'hallucinations':

"X talks to herself and plays with an imaginary friend" (age 5 years).

"Talks to himself frequently".

Delusions (PSYCHOSIS2)

The child/adolescent experiences erroneous beliefs that usually involve a misinterpretation of perceptions or experiences. Their content may include a variety of themes (e.g., persecutory, referential, somatic, religious, or grandiose). The distinction between a delusion and a strongly held idea is sometimes difficult to make and depends in part on the degree of conviction with which the belief is held despite clear contradictory evidence regarding its veracity.

Examples that were considered symptoms of 'delusions':

"Paranoia, fear, and guilt, around the idea that he may have raped someone. Believes that food contains human flesh".

"Very superstitious – believes that 'evil' comes at 3 am and wakes up every night at this time".

"Thinks everything is not a coincidence and looks for hidden meaning. Always looks for patterns. Believes he can recreate people as holograms and thinks his energy interferes with electrical appliances".

Examples that were not considered symptoms of 'delusions':

"Paranoia".

Disorganized speech (PSYCHOSIS3)

The child/adolescent exhibits disorganized speech that may be disorganized in a variety of ways. The person may 'slip off the track' from one topic to another, their answers to questions may be obliquely related or completely unrelated and, rarely, speech may be so severely disorganized that it is nearly incomprehensible, and resembles receptive aphasia in its linguistic disorganization. Examples that were considered symptoms of 'disorganized speech':

"Disorganized thoughts and bizarre speech".

"Speech incomprehensible and thoughts are disordered".

Examples that were not considered symptoms of 'disorganized speech':

"Talks about odd topics".

Disorganized/catatonic behaviour (PSYCHOSIS4)

Grossly disorganized behaviour may manifest itself in a variety of ways, ranging from childlike silliness to unpredictable agitation. This may include problems in goal-directed behaviour, difficulties in performing activities of daily living, appearing markedly dishevelled, dressing in an unusual manner, or displaying clearly inappropriate sexual behaviour. The child/adolescent exhibits catatonic motor behaviours. These include a marked decrease in reactivity to the environment, sometimes reaching an extreme degree of complete unawareness, maintaining a rigid posture and resisting efforts to be moved, active resistance to instructions or attempts to be moved, the assumption of inappropriate or bizarre postures, or purposeless and un-stimulated excessive motor activity.

Examples that were considered symptoms of 'disorganized /catatonic behaviour':

"Masturbating in front of hostel residents, plays with own faeces, touches own anus, and urinates in public".

"Bizarre behaviour - Washing face with toilet water, kissing walls, exposing genitals, and tearing up clothing".

"Walking outside in her underwear".

Examples that were not considered symptoms of 'disorganized /catatonic behaviour':

"X doesn't like showering".

Negative symptoms (PSYCHOSIS5)

The child/adolescent experiences negative symptoms of schizophrenia. These included affective flattening, alogia, and avolition. Affective flattening is characterized by the person's face appearing immobile and unresponsive, with poor eye contact and reduced body language. Alogia is manifested by brief, laconic, empty replies. Alogia is a diminution of thoughts that is reflected in decreased fluency and productivity of speech. Avolition is characterized by an inability to initiate and persist in goal-directed activities. The individual may sit for long periods of time and show little interest in participating in work or social activities.

Selective Mutism (OTHER1)

The child/adolescent exhibits a consistent failure to speak in specific social situations which there is an expectation for speaking (e.g. school), despite having the ability to speak in other situations. This disturbance interferes with educational or occupational achievement or with social communication.

Examples that were considered symptoms of 'selective mutism':

"Can't talk to other people outside of the family. Has a sore tummy when tries to talk to strangers".

Examples that were not considered symptoms of 'selective mutism':

"X is shy and quiet".

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"X doesn't say much in the classroom".

Somatoform (Other2)

The child/adolescent experiences physical symptoms that suggest a general medical condition, however the symptoms are not fully explained by a general medical condition, the effects of a substance, or by another mental disorder. There is no medical condition to fully account for the physical symptoms.

Examples that were considered 'Somatoform' symptoms:

"Somatic symptoms such as headaches and vomiting".

"Experiences consistent headaches and tummy pains".

Examples that were not considered 'Somatoform' symptoms:

"Gets headaches".

"Feels sick when anxious".

APPENDIX D:

DEFINITIONS OF ADVERSE CHILDHOOD EVENTS USED IN STUDY

<center>**Definitions of Adverse Childhood Events**</center>

The *DSM-IV-TR* defines 'trauma' as someone witnessing, or being confronted with an event or events that involve actual or threatened death, serious injury, or a threat to the physical integrity of self or others (APA, 2000, p. 463). Furthermore, the *DSM-IV-TR* includes neglect of a child, physical abuse of a child, and sexual abuse of a child in the chapter "Other Conditions that may be a Focus of Clinical Attention" and states that these diagnoses should be used "when the focus of clinical attention is severe mistreatment of one individual by another through physical abuse, sexual abuse, or child neglect" (APA, 2000).

However, adverse childhood events represent a wider category than traumatic events described in the *DSM-IV-TR* (APA, 2000; Rosenberg, Weili, Mueser, Jankowski, & Cournos, 2007). Adverse childhood events may also include non-traumatic, however stressful life events such as parental divorce, household substance abuse, and imprisonment of a parent (Kessler, Davis, & Kendler, 1997; Rosenberg, 2007).

The adverse childhood events defined below will be based on a New Zealand context. In accordance to the *Convention of the Rights of the Child*, children and adolescents in New Zealand under the age of 18 years have the same basic general human rights as adults (New Zealand Human Rights Commission, 1993). Children's rights fall into three categories: *Provision rights* include the right to an adequate standard of living, free education, adequate health resources, and legal and social services. *Protection rights* include protection from abuse and neglect, bullying and discrimination, and the right to safety within the justice system. *Participation rights* include the right to freedom of expression and the right to participate in public life (New Zealand Human Rights Commission, 1993).

Based on a review of the literature, 13 adverse childhood events were included in the present study. These included; childhood sexual abuse, childhood physical abuse, childhood emotional/psychological abuse, physical /emotional neglect, exposure to domestic/family violence,

grief/loss, parental discord/conflict, parental mental illness, parental substance use, and impoverishment. The cut-off date for the occurrence of these adverse events was 18 years of age which is consistent with the cut-off age for the *Convention of the Rights of the Child* (New Zealand Human Rights Commission, 1993).

Child Physical Abuse

Defining 'child physical abuse' can be problematic due to a definition being largely influenced by cultural and individual factors (Agar & Read, 2002). For example, in New Zealand there is currently large dispute around section 59 of the *New Zealand Crimes Act* which relates to using reasonable force to discipline children (New Zealand Ministry of Justice,1961). The *New Zealand Crimes* Act currently states that it is a criminal offence for a parent to use reasonable force to correct their child's behaviour. However, a parent is justified to use reasonable force for the particular purposes such as; "preventing harm to the child, preventing the child from engaging or continuing to engage in conduct that amounts to a criminal offence; or preventing the child from engaging or continuing to engage in offensive or disruptive behaviour" (The New Zealand Ministry of Justice, 1961, p. 61). Therefore, it is important to consider these distinctions when defining 'physical abuse' in a New Zealand context.

It is generally agreed upon in the literature that the act of child physical abuse involves a deliberately inflicted injury, or risk of injury, to a child or adolescent under the age of 18 years, as a result of being hit by a hand or object, kicked, poisoned, burnt, bitten, shaken, stabbed, or pushed (Anda et al., 2006; Kessler, Davis, & Kendler, 1997; Kolko, 2002). Some argue that a 'resulting injury' is necessary in the definition of child physical abuse (Bonner, Logue, Kaufman, & Niec, 2001; Bernet, 2000) For example, Bonner et al. (2001, p.991) describe childhood physical abuse as "an act of commission on the part of a parent or caregiver involving excessive discipline, beatings, or some other form of overt physical violence that results in injury to a child that may include

fractures, bruises, lacerations, burns, or internal injuries". This definition can be problematic because some acts that are 'physically abusive' may not necessarily result in a physical or visible injury (Malinosky-Rummell &Hansen, 1993). For example, a 6 year old child that is deliberately shoved or kicked by his father may not result in an injury, although may be in pain or feel endangered. In context of the New Zealand crimes act this 'deliberate shove or kick' is considered a criminal offence and 'abusive'. Therefore, in a New Zealand context child physical abuse does not have to result in injury and rather that the risk of injury, pain, or the endangerment of a child is sufficient to constitute 'abuse'.

Child physical abuse can be a one-off incident that fits the definition of 'physical abuse', or a repetitive behaviour (Carr, 2006; Kessler et al., 1997). Physical abuse may be intra-familial, extra-familial, or institutional, and may occur alone or in conjunction with other childhood adversities (Carr, 2006). Some argue physical abuse perpetrators are confined to 'parents or caregivers', however, such a definition would exclude acts of physical abuse carried out by older siblings, school teachers, extended family, and adult family friends (Carr, 2006).

In consideration of relevant literature and the *New Zealand Crimes Act*, for the purpose of this study 'child physical abuse' can be defined as:

"Deliberately inflicted injury, pain, or risk of injury, as a result of being, kicked, suffocated, poisoned, burnt, bitten, shaken, hit by a hand or object, stabbed, or pushed. This includes intra-familial, extra-familial, and institutional perpetrators of physical abuse. This includes physical assault by adolescent peers and older siblings, although does not include child-like rough and tumble of pre-adolescent children".

In cases that are vague, for example "X was slapped by his father", there must be an additional descriptor such as "slapped frequently", "slapped hard", or "slapped across the face" to

be included as physical abuse. This is in order to prevent misdemeanours such as a one-off slap on the hand for misbehaviour being included as 'child physical abuse'.

Examples that were considered 'child physical abuse' in the present study:

"X is often slapped by his father".

"X was physically abused by her Mother who tried to strangle her as a child".

"X was stabbed through the hand with a pen by a 13 year old classmate".

"X (adolescent) was bullied at school; she was kicked repeatedly by some older boys"

"Mum smacks X when he is naughty".

"Older brother often bullies X – he has hit her on more than one occasion".

"Physical and verbal bullying at intermediate school – racist comments"

"Beaten by 4 boys at school – gang members. Often bullied at school and physically assaulted".

Examples that were not considered 'child physical abuse' are:

"Bullied at school since intermediate".

"Suspected history of abuse and neglect and specifics are unknown".

"X was hit and pushed by older sister at times as a child".

"X was slapped by his father".

Child Sexual Abuse

There is no general consensus between practitioners, professionals, and researchers, as to what exactly constitutes 'child sexual abuse' (Bonner et al., 2001; Finkelhor, 1999). Similarly to child physical abuse, such a definition is largely influenced by cultural, societal, and individual

factors. The lack of consensus around a clear definition has a negative impact on research findings, prevalence rates, assessment, and treatment relating to sexual abuse (Bonner et al., 2001).

Many researchers base definitions on age differentials between the perpetrator and victim, the relationship between the victim and perpetrator, the use of coercion, the reaction of the victim and the level physical contact (Bonner et al., 2001; Holmes & Slap, 1998). Some researchers specify that the perpetrator must be an 'adult' or 'at least 5 years older than the victim' (Felitti et al., 1998; Kessler et al., 1997; Webster, 2001). For example, Anda et al. (2006, p.177) define child sexual abuse as "an adult or person at least 5 years older touching or fondling a child in a sexual way, or touching their body in a sexual way, or attempted oral anal or vaginal intercourse with the child, or actually had oral anal or vaginal intercourse with the child". However, others argue this age specification is problematic as the use of force and the exploitation of power and authority should be considered 'abusive' despite age differentials (Bernet, 2000; Finkelhor, 1999; Russel, 1983; Wyatt & Peters, 1986, Wyatt, 1985). Therefore, the use of force, exploitation of power or authority, regardless of age differentials should always be considered abusive despite age differentials.

The level of physical contact involved in the act of sexual abuse also various among the definitions used by researchers (Holmes & Slap, 1998; Wyatt, 1985). However, most studies agree that genital touching and fondling in a sexual way, touching a child/adolescents body in a sexual way, or attempting oral, vaginal, or anal intercourse all constitute 'child sexual abuse' (Finkelhor, 1999; Kessler et al., 1997, Wyatt, 1985). Some studies include exhibitionism and socialisations to engage in sexual behaviour as 'sexual abuse' due to the potentially exploitive and derogatory nature of these acts (Wyatt, 1985).

Based on relevant literature, for the purpose of this study 'child sexual abuse' can be defined as (Bernet, 2000; Bonner et al., 2001; Finkelhor, 1999; Webster, 2001; Wyatt, 1985):

"Sexual experience in which the child/adolescent is subjected to sexually based exploitation, humiliation, or degradation, or unable to fully comprehend the situation or give informed consent. Non-physical sexual experience such as exhibitionism is included as a second defining factor provided this subjected the child/adolescent to exploitation, humiliation, or degradation. The perpetrator of sexual abuse can be of the same or opposite sex, is generally older, and generally has coercive or authoritative power over the child or adolescent. However, for the purpose of this study it is also important to have a third defining characteristic of 'exploitive, forceful or unwanted sexual experience' regardless of age difference. This includes sexual experiences of this nature with peers that are exploitive or forceful, despite the lack of age or power discrepancy".

The majority of cases in the present study that referred to 'sexual abuse' did not include extensive detail. For example, many cases stated "X was sexually abused by her cousin when she was 12 years old". Therefore, for the purpose of the present study, cases that state the participant was "sexually abused" will be included as sexual abuse. This is based on the assumption that incidences that are judged by the clinician as 'sexual abuse' in the participants file are consistent with the definition above.

Examples of what were considered 'child sexual abuse' in the present study:

"X disclosed sexual abuse during therapy and told her school teacher. She was referred for ACC counselling".

"X had abuse issues at school – he was made to show his penis in the toilet, he was forced to remove his clothes and the other children touched him inappropriately".

"X was touched around his bottom by a mentally unwell man".

"X was exposed to sexual behaviour between her father and step mother. She saw them having sexual intercourse at the age of 9 years".

Examples that were not included as 'child sexual abuse are:

"Suspected sexual abuse as a child by a male painter who came into the home".

"X had an unwanted sexual experience with male friend while intoxicated".

"Mum questions sexual abuse by X's 22 year old boyfriend".

Childhood neglect

The definition of childhood neglect is particularly problematic due to the varying levels of contact between children and their caregivers and what is deemed as 'acceptable' among different cultures (Carr, 2006). It is therefore important to take into account cultural and societal beliefs and values when defining 'child neglect'. From a western perspective, Bernet (2000, p.321) defines child neglect as "the failure to provide adequate care and protection for children by malicious or ignorant withholding of physical, emotional, and educational necessities". Unlike child physical, sexual and emotional abuse, child neglect is generally not intentional on the parent's behalf. Rather it typically arises as a result of inadequate parenting resources and a lack of awareness of their child's needs (Carr, 2006; Glaser, 2002).

Childhood neglect is commonly divided into discrete categories of 'emotional neglect' and 'physical neglect' (Glaser, 2002; Kessler et al., 1997). For the purpose of this study 'childhood neglect' is inclusive of both emotional and physical neglect. Based on relevant literature, 'childhood emotional neglect' can be defined as:

"The unavailability of parents/caregivers to respond to the emotional needs of their child/adolescent due to a preoccupation with parental difficulties or responsibilities, such as

parental mental health problems, substance abuse, or overwhelming work commitments"

(Carr, 2006; Glaser, 2002; Kessler et al., 1997). The emotional unavailability of the

primary caregiver leads the child to feel unloved, unsupported, weak, and unimportant".

In addition, 'childhood physical neglect' can be defined as:

"A passive ignoring of the child or adolescents needs including: physical needs of clothing,

shelter, and food, safety needs and protection, emotional needs such as nurturance,

intellectual needs of stimulation, social interaction and conversation; and the need for age

appropriate discipline and boundary setting" (Carr, 2006; Kessler, 1997). Similarly to

emotional neglect, physical neglect is generally not intentional on behalf of the parent;

moreover a lack of resources or lack of awareness of their child's needs (Carr, 2006").

In order to fit the definition above, it must mention in the file that the parent is emotionally or physically neglectful. Therefore, statements such as "X feels lonely and isolated" do not fit the criteria to be included as 'childhood neglect'.

Examples of childhood neglect that were included in the present study are:

"Father works very long hours, not around much - and refuses to talk to X".

"CYFS are involved due to neglect".

"No boundaries from parents".

"Invisible dad - he is always at work. Both parents are disengaged from X and he lives in an isolated family environment".

Examples of childhood neglect that were not included in the present study are:

"X feels lonely – her family are never around. X's mum and grandmother are moving to Australia."

"X feels isolated and lonely".

Childhood emotional and psychological abuse

The problem of definition is perhaps the most pronounced when defining 'emotional or psychological child abuse'. One major discrepancy in the literature relates to weather 'psychological abuse' and 'emotional abuse' can be regarded as synonymous (Glaser, 2002; O'Hagen, 1995). O'Hagan (1995) suggests that emotional and psychological abuse are usefully distinguished from each other. However, other researchers argue such a distinction is not helpful as emotional processing impacts on psychological processing and vice versa (Carr, 2006; Glaser, 2002). Due to the mutual consensus that psychological and emotional processes are not mutually exclusive, this study will define psychological and emotional abuse as synonymous (Carr, 2006; Glaser, 2002; Peterson, Borrego, Terao, & Urquiza, 2003).

There is also inconsistency in the research relating to whether emotional and psychological abuse can occur outside of the parent-child relationship. Some researchers define psychological and emotional abuse as only carried out by a parent or caregiver (Bernet, 2000; Bonner et al., 2001; Carr, 2006). However, other researchers include psychological and emotional abuse outside of the parent-child relationship, including: siblings, adolescent dating relationships, in schools, and adults outside of the family (Anda et al., 2006; Kessler et al.,1997; O'Hagan, 1995). For the purpose of the present study, psychological and emotional abuse outside of the parent-child relationship will be included due to the high levels of rejection, bullying, and discrimination reported in adolescent school relationships in this particular population that fit the definition for 'emotional and psychological abuse' outlined below.

Based on a review of relevant literature, childhood emotional/psychological abuse can be defined as:

"A repeated pattern or extreme instances of inappropriate emotional response to a child or adolescent's experience of emotions and its accompanying expressive behaviour. This includes: rejection, terrorizing, isolating, exploiting/corrupting, racism, discrimination, denying emotional responsiveness, having unrealistic or excessive expectations of child, deception, and frequent punishment for minor misdemeanours (Anda et al., 2006; Bernet, 2000; Boner et al., 2001; Carr, 2006; Kessler et al., 1997). Emotional/psychological abuse leads the child/adolescent to feel they are worthless, humiliated, unloved, unwanted, endangered, vulnerable, or only of value only in meeting someone else's needs (Bonner et al; Carr, 2006; O'Hagan, 1995").

Examples of what was considered 'child psychological/emotional abuse' in the present study are:

"X's parents are verbally abusive. They say they hate her and that she is a demon child".

"Mum is verbally abusive and negative to X".

"X is persistently bullied and ostracized by friends at school".

"Dad was volatile, mean, and rude to X, he doesn't care about her and not interested in her".

"Father tells X that he doesn't want to know him".

"Physical and verbal bullying at intermediate school – racist comments".

Examples of 'child psychological/emotional abuse' that were not included in the present study are:

"Mother was very negative and critical".

"Father's anger towards X is contributing to his depression".

"One teacher at daycare wouldn't let X go to the toilet and she wet her pants".

Bullying

Due to the high prevalence of 'bullying' among the participants in the present study, a separate criterion was included. The definition of "bullying' includes physical abuse and emotional abuse that occurs outside of the family home. This includes bullying at school, work, sport activities, or between peers. In order to be included as 'bullying' the example must fit either the definition of 'physical abuse' or 'emotional and psychological abuse' outlined above. Due to potential overlap between 'bullying' and other categories (physical abuse and emotional abuse), those that fit more than one category are included in both.

Examples that were considered 'bullying':

"Physical and verbal bullying at intermediate school – racist comments".

"X was bullied at school".

"Bullying".

"Beaten by 4 boys at school – gang members. Often bullied at school and physically assaulted".

Examples that were not considered "bullying":

"X does not have many friends at school".

"X finds it difficult getting on with peers at school. She thinks no one likes her".

"Older brother often bullies X – he has hit her on more than one occasion".

Parental Separation or Divorce

The definition of 'parental separation or divorce' is perhaps more straight forward than other childhood adverse events (King, 2001; Rosenberg et al., 2007). The common definition in

research was a simple question: 'while growing up did your parents separate or divorce?' (Kessler et al., 1997; Rosenberg et al., 2007). For the purposes of this study, parental separation and divorce can be defined as"

"One parent leaving the household due to marital difficulties for 6 months or more (Kessler et al., 1997; King; 2001; Rosenberg et al., 2007; Thill, 1999). This includes the separation of a parent and a step-parent provided the step-parent was providing the role of a parent or caregiver to the child/adolescent".

Examples of what were considered 'parental separation or divorce' in the present study are:

"Parents are divorced and X has lived in a blended family for the last two years".

"Parents separated when X was 2 years old".

"X's parents have been separated since he was 9 months old and they both have remarried and had children".

Examples of 'parental separation or divorce' that were not included in the present study are:

"Parents separated when X was a child, however are together now".

"Dad left the family home six weeks ago".

"Parents broke up 10 years ago for a year, while X's mum was in a refuge".

Childhood grief/loss

Based on a review of the literature child grief/loss can be defined as:

"The death of a parent, sibling, or significant other person, or absence of a parent for six months or longer (Sayler & Koocher, 2001; Kessler et al., 1997). In order for a person to

be 'significant' the file must state that the individual was 'close' or 'important' to the

individual, or that the individual is experiencing 'grief', 'upset' or 'distress' due to their

departure. Furthermore, grief or loss can include personal loss of own function due to a

serious illness, or loss due to relocating living situation away from family, friends, and

school provided this relates to feelings of loss and grief (Sayler & Koocher, 2001)".

Grief and loss can lead to a range of complex emotions in children and adolescents including numbness, sadness, confusion, anger, and fear (Sayler & Koocher, 2001). For the purpose of this study, the absence of a parent for six months or longer is only included if there is no ongoing contact with the parent. Files that indicate any degree of ongoing contact are excluded from this criteria.

Examples of what was considered 'childhood grief/loss' in the present study are:

"X's father has recently moved to Australia with his new partner and children – he has not seen him this year".

"X has no contact with her father – he lives in China and she misses him".

"Uncle committed suicide in 2007 – he was very close to his uncle".

Examples of 'childhood grief/loss' that were not included in the present study are:

"There have been two recent suicides in the family – an uncle and an aunt"

"X's grandmother died during his referral to the service"

"Recent change of school"

"Grandfather died"

"Parents are divorced and there is not much contact with X's father".

"X see's his dad every two weeks".

Parental mental illness

For the purpose of this study, biological parents and/or caregivers with a mental illness will be included in the definition for 'parental mental illness' (Sivec, Waehler, Masterson, & Pearson, 2006). Based on previous research, 'parental mental illness' can be defined as:

"A parent or caregiver who has attempted suicide, committed suicide, has a known mental disorder, or has been hospitalized due to mental health problem' (Anda et al., 2006; Kessler et al., 1997). This includes parents/caregivers with previous and/or current mental health diagnosis, regardless of level of treatment".

Examples of that were considered 'parental mental illness' in the present study are:

"Father has an alcohol dependence and has been hospitalized for severe liver damage".

"Mum has schizophrenia and has periods of hospitalization. Dad has drug related mental health problems and a diagnosis of schizoaffective disorder".

"Mum had post natal depression following the birth of X".

"Mum has 'stress attacks', anxiety, and a history of rapes".

"Dad had a nervous breakdown".

Examples of 'parental mental illness' that were not included in the present study are:

"Mother believes that X's father is depressed".

"Mum was sad and cried a lot during pregnancy".

"Mum was burnt out while X was a toddler".

Exposure to domestic and family violence in the home

'Domestic violence' is violence committed by a heterosexual partner (generally between the parents or caregivers of the child/adolescent) including physical injury, intimidation, willful damage to property, indecent behaviour without consent , serious harassment or a threat to commit any of these acts (Hegarty, Hindmarsh, & Gilles, 2000). 'Family violence' can occur in other relationships within the household, including abuse between fathers, mothers, uncles, aunties, elders, children, and siblings. Domestic and family violence includes intentional pushing, hitting with a hand or object, kicking, burning, suffocating, poisoning, or otherwise being physically hurt, forcing of sexual activities, or threatened or hurt by a knife or gun (Anda et al. 2006; Hegarty, Hindmarsh, & Gilles, 2000; McCauley et al., 1995). Similarly to the definition of physical abuse, the perpetrator of family violence must be an adolescent or older in order prevent the inclusion of child-like 'play fighting'. This includes physical assault by adolescent peers and older siblings, although does not include child-like rough and tumble of pre-adolescent children.

Exposure to domestic and/or family violence can be defined as:

> *"A child or adolescent witnessing violence between family members in the home, being forced to watch or participate in violent behaviour, experiencing a police intervention due to violence in the home, or being taken to a shelter for 'battered woman' (Eleson, 1999; Hegarty, Hindmarsh, & Gilles, 2000)".*

Examples of 'exposure to domestic and family violence' that were included in the present study are:

> "Parents fight a lot and X feels unsafe. There have been care and protection issues and CYFS are involved for the last three months".

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"X exposed to domestic abuse, frequent arguments, and fights in family".

"X heard his father assault his mum's new partner. He is scared of his father. His father is aggressive with the car towards his mother".

"X's younger brother has a concussion from being hit by his father".

Examples of 'exposure to domestic and family violence' that were not included in the present study are:

"There is conflict between X's siblings and their step mother".

"Frequent arguments between family members".

"Lots of fighting in the family home".

Exposure to Parental Discord

Child and adolescent experiences of parental discord are commonly defined in research using measures such as the 'Family Conflict Inventory' and the 'Children's Perception of Interparental Conflict' (Davies & Lindsay, 2004; Jenkins, Simpson, Dunn, Rasbash, & O'Conner, 2005). Other researchers ask parents to rate how often they have had 'open disagreements' with their spouse in front of their children as a way of measuring marital discord (Musick & Meier, 2010; Vandewater & Lansford, 1998). Perhaps of most relevance to the present study, Davies and Lindsay (2004) measured children and adolescent's exposure to marital discord across four more domains: the frequency of parental arguments, the intensity of parental arguments, weather the arguments are resolved, and the content of parental disagreements. For the purpose of the present study, 'exposure to marital discord' can be defined as:

"A child or adolescent who is exposed to repetitive or extreme incidences of inter-personal parental conflict that is of a verbally aggressive nature' (Davies & Lindsay, 2004). This

includes open disagreements about household tasks, money, spending time together, sex, in-laws, and children (Musick & Meier, 2010; Vandewater & Lansford, 1998). The exposure to parental discord is less than optimal for the child or adolescent's development and negatively affects their wellbeing (King, 2001; Maccoby & Martin, 1983).

In cases that are vague, for example "parents fight", there must be an additional descriptor such as "fight frequently", "have serious arguments", or "X is often distressed by parents fighting" to be included as 'parental conflict'. This is in order to prevent a one off misdemeanour between parents which is likely common in most families. In files that state the parents are 'divorced' or 'separated', it cannot be presumed that there is discord between the parents unless otherwise stated. In files that state there is domestic violence between the parents, it can be assumed there is parental discord and can be included under both 'parental discord' and 'domestic violence' (provided the file fits the definition of 'domestic violence').

Examples of 'parental discord' that were included in the present study are:

"Parents fight a lot".

"Parents arguments scared her – they argued a lot".

"Mum and dad continue to argue, dad is very angry and they often refuse to talk to each other".

"Lots of fighting between X's parents before they separated. They were often physically violent towards each other".

"X heard his father assault his mum's new partner. He is scared of his father. His father is aggressive with the car towards his mother".

Examples of 'parental discord' that were not included in the present study are:

"Relationship issues [parents]".

"Parental conflict".

"Friction in parent's relationship".

Parental substance use

The Children of Alcoholic's Screening Test (CAST) is commonly used in research to identify children who live with an alcoholic parent (Gance-Cleveland, Mays, & Steffen, 2008). Other researchers ask generic questions such as "does anyone living in the household have a problem with drinking or alcohol? Or does anyone in the household use or deal drugs or illegal substances?" (Anda et al., 2006, p.177).

As defined in the *DSM-IV,* alcoholism can be referred to as "a maladaptive pattern of alcohol consumption leading to clinically significant distress and impairment occurring over a 12 month period or more" (APA, 2000). For the purpose of the present study, 'parental substance use' can be defined as:

"Parent/caregiver alcoholism, parent/caregiver use of illegal substances, or the dealing of illegal drugs within the family. The substance use actually or potentially leads to a failure to fulfil major roles at home and work, legal problems, financial difficulties, and recurrent social and interpersonal problems (APA, 2000). In regards to parent/caregiver use of illegal drugs, this includes a single incident or repetitive use of illegal substances in the family household. The dealing of illegal substances in the household includes parents, siblings, and extended family or friends living in the household that deal illegal substances. This can be a one-off incident or repetitive dealing (Anda et al., 2006)".

Examples of 'parental substance use' that were included in the present study are:

"X's dad is a drug addict so X lives with his grandparents".

"X's mum is an alcoholic – X now lives with his dad".

"Mum abused substances while she was pregnant".

"Dad often 'comas' out after drinking. He was recently unemployed because of his drinking problems".

"Dad smokes cannabis in front of the children at home"

"Dad is violent when he drinks

Examples of 'parental substance use' that were not included in the present study are:

"Dad is regularly drunk".

Impoverishment

Based on relevant literature, impoverishment can be defined as:

"A position of financial hardship leading a family to operate on a scarce economy' (Townsed, 2010). This includes parental unemployment and ongoing financial hardship that leads to over-crowded living situations, lack of food supply, poor clothing, and lack of education (Young & Gately, 1988)".

Examples of 'impoverishment' that were included in the present study are:

"Significant financial stress recently. Dad was declared bankrupt".

"Mum doesn't proved proper meals".

Examples of 'impoverishment that were not included in the present study are:

"Lives with a solo mum".

"Mum recently lost her job".

"Mum lived on very little income when X was a baby".

APPENDIX E

CHI-SQUARE ASSOCIATIONS BETWEEN ADVERSE CHILDHOOD EVENTS AND

DIAGNOSES

		ODD	CD	ADHD	PTSD
Adverse childhood event					
Physical abuse	x^2	6.45	1.38	0.12	6.46
	P	.026	.236	.445	.011
Sexual abuse	x^2	0.16	6.78	0.08	32.23
	P	.528	.009	.534	.000
Neglect	x^2	2.44	1.57	0.01	0.12
	P	.167	.227	.549	.591
Emotional and psychological abuse	x^2	1.46	0.00	2.65	1.83
	P	.226	.657	.077	.160
Parental separation or divorce	x^2	9.30	0.01	1.93	0.02
	P	.002	.638	.122	.598
Loss	x^2	3.68	0.33	0.08	3.09
	p	.075	.429	.474	.081
Parent mental health problems	x^2	0.01	1.61	0.50	0.03
	p	.658	.235	.310	.556
Parental discord	x^2	4.43	0.50	0.53	0.00
	p	.069	.613	.330	.617
Exposure to domestic violence	x^2	10.63	2.47	0.01	0.82
	p	.015	.163	.622	.311
Parent substance use	x^2	0.01	4.09	0.05	1.39
	p	.643	.078	.505	.218
Impoverishment	x^2	0.33	8.191	0.20	0.48
	p	.732	.004	.456	.433
Bullying	x^2	0.38	0.00	0.00	0.47
	p	.472	.640	.565	.389
Illness or disability	x^2	0.35	0.59	0.37	0.90
	p	.714	.573	.378	.431
Family illness or disability	x^2	0.81	0.43	0.28	0.65
	p	.370	.667	.503	.542

Diagnosis (column span header over ODD, CD, ADHD, PTSD)

APPENDIX F

CHI-SQUARE ASSOCIATIONS BETWEEN ADVERSE CHILDHOOD EVENTS AND

SYMPTOM CLUSTERS

		Symptom clusters			
Adverse childhood event		ADHD	CD	ODD	PTSD
Physical abuse	x^2	9.58	12.19	12.49	4.44
	p	.022	.016	.002	.218
Sexual abuse	x^2	0.76	12.94	0.83	23.39
	p	.859	.012	.661	.000
Neglect	x^2	1.29	11.67	9.44	5.22
	p	.731	.020	.009	.157
Emotional and psychological abuse	x^2	1.46	3.90	3.68	8.39
	p	.692	.420	.158	.039
Parental separation or divorce	x^2	1.79	7.92	2.46	1.57
	p	.618	.095	.293	.667
Loss	x^2	2.33	7.12	1.82	17.56
	p	.506	.130	.404	.001
Parent mental health problems	x^2	2.85	8.05	1.52	5.37
	p	.416	.090	.469	.147
Parental discord	x^2	2.85	1.29	1.87	1.54
	p	.416	.863	.393	.673
Exposure to domestic violence	x^2	5.50	16.87	0.70	3.15
	p	.139	.002	.705	.370
Parent substance use	x^2	4.60	7.53	1.56	1.12
	p	.203	.111	.458	.772
Impoverishment	x^2	0.50	7.58	7.20	3.22
	p	.920	.108	.027	.359
Bullying	x^2	2.03	0.59	1.70	3.54
	p	.556	.964	.427	.316
Illness or disability	x^2	0.27	2.14	0.06	1.23
	p	.966	.709	.970	.745
Family illness or disability	x^2	2.60	6.07	0.58	0.62
	p	.458	.194	.747	.892

APPENDIX G

CHI-SQUARE ASSOCIATIONS BETWEEN ADVERSE CHILDHOOD EVENTS AND

INDIVIDUAL SYMPTOMS

Adverse childhood event		Inattentiveness	Hyperactivity	Impulsivity
			ADHD symptoms	
Physical abuse	x^2	1.78	6.19	7.20
	p	.126	.013	.010
Sexual abuse	x^2	0.43	0.00	0.03
	p	.351	.595	.544
Neglect	x^2	0.21	1.42	1.64
	p	.421	.172	.168
Emotional and psychological abuse	x^2	0.00	0.21	0.00
	p	.551	.389	.572
Parental separation or divorce	x^2	0.06	0.18	2.03
	p	.472	.411	.120
Loss	x^2	1.19	1.86	0.37
	p	.179	.120	.353
Parent mental health problems	x^2	1.75	2.90	0.02
	p	.125	.065	.543
Parental discord	x^2	0.68	0.76	0.04
	p	.263	.253	.556
Exposure to domestic violence	x^2	1.31	12.24	0.12
	p	.183	.002	.534
Parent substance use	x^2	2.27	5.81	0.04
	p	.100	.018	.556
Impoverishment	x^2	0.28	0.36	0.04
	p	.454	.387	.523
Bullying	x^2	1.96	0.53	0.63
	p	.112	.292	.341
Illness or disability	x^2	0.00	0.24	0.05
	p	.626	.412	.499
Family illness or disability	x^2	0.43	3.08	0.04
	p	.398	.067	.543

		Conduct Disorder symptoms			
Adverse childhood event		Aggression to people and animals	Destruction of property	Deceitfulness and/or theft	Serious Violation of Rules
Physical abuse	x^2	5.71	1.29	2.03	9.21
	p	.013	.197	.113	.003
Sexual abuse	x^2	0.07	2.99	3.56	2.56
	p	.482	.081	.060	.095
Neglect	x^2	2.87	6.50	0.00	5.29
	p	.069	.016	.615	.025
Emotional and psychological abuse	x^2	0.03	0.69	0.06	0.05
	p	.488	.265	.479	.486
Parental separation or divorce	x^2	0.87	1.28	2.87	0.72
	p	.216	.178	.069	.253
Loss	x^2	2.83	1.29	4.11	3.60
	p	.063	.175	.034	.045
Parent mental health problems	x^2	0.31	0.17	0.25	0.16
	p	.340	.419	.375	.414
Parental discord	x^2	0.64	0.12	3.13	0.00
	p	.263	.445	.066	.541
Exposure to domestic violence	x^2	7.61	2.09	7.86	6.25
	p	.006	.128	.010	.018
Parent substance use	x^2	0.44	1.87	2.02	15.50
	p	.313	.135	.121	.000
Impoverishment	x^2	6.88	3.62	2.59	2.01
	p	.012	.078	.116	.150
Bullying	x^2	0.06	0.40	0.79	0.09
	p	.461	.343	.244	.463
Illness or disability	x^2	0.02	1.18	0.55	0.05
	p	.540	.244	.322	.389
Family illness or disability	x^2	5.25	0.50	0.76	2.98
	p	.016	.416	.340	.072

		Oppositional Defiant Disorder symptoms	
Adverse childhood event		Non-compliance	Oppositional Behaviour
Physical abuse	x^2	1.78	5.92
	p	.126	.012
Sexual abuse	x^2	1.61	0.05
	p	.151	.491
Neglect	x^2	1.93	2.90
	p	.125	.067
Emotional and psychological abuse	x^2	0.10	0.62
	p	.449	.261
Parental separation or divorce	x^2	0.86	0.20
	p	.224	.379
Loss	x^2	0.17	2.83
	p	.404	.062
Parent mental health problems	x^2	0.42	0.03
	p	.315	.491
Parental discord	x^2	0.15	0.08
	p	.435	.466
Exposure to domestic violence	x^2	1.31	2.84
	p	.183	.063
Parent substance use	x^2	0.05	2.87
	p	.486	.066
Impoverishment	x^2	0.76	3.18
	p	.287	.070
Bullying	x^2	0.00	2.08
	p	.568	.098
Illness or disability	x^2	0.00	0.13
	p	.626	.448
Family illness or disability	x^2	0.00	0.59
	p	.602	.320

		PTSD symptoms		
Adverse childhood event		Re-experiencing of traumatic events	Avoidance and numbing	Increased arousal
Physical abuse	x^2	1.32	3.66	3.67
	p	.190	.056	.063
Sexual abuse	x^2	17.10	1.76	33.75
	p	.001	.182	.000
Neglect	x^2	0.17	2.08	0.08
	p	.505	.155	.520
Emotional and psychological abuse	x^2	0.22	0.02	0.61
	p	.430	.565	.317
Parental separation or divorce	x^2	0.00	1.84	0.50
	p	.605	.151	.342
Loss	x^2	5.25	13.31	5.65
	p	.022	.000	.021
Parent mental health problems	x^2	2.45	8.21	0.96
	p	.093	.002	.256
Parental discord	x^2	1.06	3.20	0.22
	p	.246	.063	.439
Exposure to domestic violence	x^2	2.08	0.12	0.35
	p	.144	.592	.408
Parent substance use	x^2	0.01	0.00	0.60
	p	.615	.671	.329
Impoverishment	x^2	0.00	0.74	0.20
	p	.730	.498	.502
Bullying	x^2	0.06	1.11	0.16
	p	.503	.241	.488
Illness or disability	x^2	0.19	1.11	1.11
	p	.548	.356	.356
Family illness or disability	x^2	0.86	0.13	0.80
	p	.353	.528	.472

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