Developmental Dyslexia and/or co-occurring Attention Deficit: Investigation of prevalence, underlying cognitive deficits, and family risk in a self-selected sample of parents and children.

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This thesis is dedicated to Kyle
Abstract

Reading acquisition is a major developmental achievement and plays a critical role in determining academic achievement. Developmental dyslexia is a heritable disorder with up to 66% of at-risk children later diagnosed with the disorder. Children with dyslexia represent a vulnerable group at high risk of underachievement at school, as well as for poorer socio-emotional outcomes. Dyslexia and Attention Deficit Hyperactivity Disorder (ADHD) commonly co-occur, and recent theories hypothesise that it is the inattentive (AD) symptom domain that is most closely associated with literacy problems. Despite an extensive overlap in symptoms, and a close genetic association, they have primarily been investigated as distinct disorders. However, recent theoretical models propose a multiple deficit framework to examine shared cognitive deficits as an explanation for their co-occurrence.

This research had seven aims: (1) investigate the prevalence of single and multiple developmental disorders, in a sample of children with a previous diagnosis of dyslexia or attention deficit, with or without hyperactivity disorder (ADHD, AD), or who had been identified by parents as underachieving in school; (2) determine whether the severity of the deficits found would be greater in children with more than one disorder compared to a single disorder; (3) validate an adult self-report protocol used to screen for dyslexia and ADHD, and use this measure to estimate family risk of each disorder; (4) determine the prevalence of symptoms of dyslexia, ADHD or AD in children with and without a family risk of dyslexia, ADHD or AD; (5) determine whether a single, double, multiple, or an intergenerational multiple deficit model is the best predictor of reading fluency; (6) examine the extent that difficulties with phonological awareness and rapid automatic naming differentiate dyslexia, and difficulties with interference control differentiate ADHD and AD; and (7) examine the Multiple Deficit Model as an explanation of the co-occurrence between dyslexia + ADHD, and dyslexia + AD using cognitive variables identified in the literature as common to both
disorders. Seventy-two children aged 9 to 11 years of age, were assessed on a broad range of cognitive, reading, language, motor and attention measures. In Study 1 (Chapter 3), prevalence rates were estimated for the single and multiple developmental disorders using the clinical and subclinical scores from the reading, attention, language and motor measures. The hypothesis that most children would show evidence of more than one developmental disorder was not met using clinical criteria, as the overall prevalence rates for single and multiple disorders were similar. However, when subclinical symptoms were included, most children diagnosed with one disorder had subclinical symptoms of at least one other disorder. Results from one-way ANOVAs showed the groups with co-occurring dyslexia + AD, or dyslexia + ADHD were not more impaired than the single disorder groups, indicating that the severity of each disorder was not influenced by the number of disorders experienced by each child.

The Adult Reading Questionnaire (ARQ) and Adult ADHD Self-Report Scale (ASRS) are self-report protocols designed to evaluate reading, writing and spelling proficiency, expressive language difficulties (word finding), as well as problems of organization, attention and hyperactivity in adults. One hundred and seventeen parents (64 mothers and 53 fathers) completed the self-report questionnaire and the data was used to replicate the factor structures of the ARQ and ASRS separately (Study 2, Chapter 4). The parent data was then used to estimate family risk of dyslexia and ADHD or AD in the child sample, and the results supported the hypothesis that children from a family with parent-reported reading difficulties were significantly more likely to have clinical symptoms of dyslexia than children without family risk. Similarly, children whose parents reported symptoms of ADHD were more likely to have clinical and subclinical symptoms of ADHD, than children coming from a family without parental report of these disorders. Correlation, regression, logistic regression analyses, and odds ratios indicated a strong influence of
parent-reported reading difficulties on child reading difficulties, as 70.4% of children at family risk of dyslexia had scores in the clinical range on the reading measure. The association between parent-reported symptoms and a child diagnosis was weaker for ADHD, as less than half (42.9%) the children at family risk met criteria for a clinical diagnosis, however, when subclinical symptoms were included, the children of parents with self-rated symptoms of ADHD were more likely to have clinical or subclinical symptoms than children from no-risk families. No significant association was found between parent-reported symptoms and a child diagnosis of AD.

In Study 3 (Chapter 5), correlation and multiple regression analyses were used to evaluate a number of cognitive variables (phonological awareness, rapid naming, and expressive language) identified in the research literature to be impaired in individuals with dyslexia. Single, double, and multiple deficit frameworks were used to determine which model best predicted reading fluency, with the results showing that children presented with a combination of deficits, indicating various pathways to dyslexia. Using the intergenerational multiple deficit model to examine the association between parent-reported reading difficulties and children’s reading fluency, it was shown that parent self-reported difficulties accounted for a substantial proportion (18.6%) of the variance in children’s reading fluency scores. After controlling for parent self-reported reading skills, phonological awareness and rapid letter naming each made additional contributions to the explanation of children’s reading fluency.

Finally, a multiple deficit model was used to achieve the final research aim (Study 4, Chapter 6). Mixed factorial ANOVAs, and moderated regression analyses were conducted to determine if impairments in phonological awareness and rapid automatic naming were unique to dyslexia, and if impairments in interference control were unique to ADHD and AD. The contribution of working memory and reading comprehension were examined as explanations
of the co-occurrence between dyslexia and ADHD or AD. The ability to inhibit distractors (interference control) was found to be impaired in each of the three groups, however some differences were noted. The group with dyslexia responded more slowly on the incongruent Flanker condition, and the ADHD group was less accurate on both the congruent and incongruent conditions, while the AD group were found to be both slower and less accurate on each of the conditions. Phonological awareness and reading comprehension were found to be unique predictors of dyslexia. The relationship between reading fluency and working memory was moderated by AD and ADHD. When symptoms of AD and ADHD were low, working memory improved as reading fluency improved, however this was not the case when symptoms of AD and ADHD were high, where an increase in reading fluency was not accompanied by increases in working memory capacity. Although weaker, reading fluency and rapid naming was moderated by AD, however no association was found for ADHD.

From a theoretical perspective, this research advances our understanding of the co-occurrence between dyslexia, ADHD and AD in school age children, as well as the associations between some of the cognitive variables associated with the disorders. This research highlights the high prevalence of dyslexia among children at family risk, and the important contribution of parent-reported difficulties to the explanation of their children’s reading fluency. At a practical level this research is expected to contribute to the better identification of children with reading and attention difficulties. The findings demonstrate the substantial overlap between the symptoms of dyslexia and inattention, and the importance of assessing for both disorders. The success of reading interventions has been found to be limited, and this may be due to the high prevalence of attention deficits which co-occur with dyslexia and that may interfere with remediation. When underlying attentional difficulties are identified, appropriate cognitive training programs can be implemented in conjunction with reading remediation to improve academic performance in school.
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Statement of Originality

I hereby certify that this thesis is the result of my original research. All sources and references quoted have been acknowledged in the text. The material contained in this thesis has not been submitted, in whole or in part, for a degree at this or any other University.

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The Griffith University Human Research Ethics Committee granted ethical clearance for the studies reported in this thesis (GU Ref: PSY/05/13/HREC). I confirm that the research was conducted in accordance with the approved protocols.

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Chapter 1: Introduction

1.1 Developmental Dyslexia

In alphabetic writing systems such as English, learning to read and write is a complex and integrative procedure. The reading process involves a multifaceted skillset that consists of two primary components: word recognition (the precise visual recognition of letters, letter combinations and words, together with the skill for converting the visual forms to their appropriate sounds) and reading comprehension (Aaron, Joshi, & Williams, 1999). To some extent these components develop independently, and difficulties with reading can result from either underdeveloped decoding skills or weak comprehension skills (Aaron, Joshi, Palmer, Smith, & Kirby, 2002). While most children acquire these skills with relative ease, for others, effortless and fluent reading is never fully achieved despite appropriate learning opportunities and normal intelligence. As reading is the foundation of learning at school, and a prerequisite for the development of other skills (Defior & Serrano, 2011), failure to achieve reading fluency has the potential to profoundly impact a child’s academic achievement, as well as future employment opportunities or choices (Boada, Willcutt, & Pennington, 2012; Gray & Climie, 2016). Reading fluency has been defined as “the ability to read connected text rapidly, smoothly, effortlessly, and automatically with little conscious attention to the mechanics of reading such as decoding” (Meyer & Felton, 1999, p.284).

Dyslexia is a broad term often used to define those children who have difficulty with accurate and/or fluent word recognition, poor decoding, and poor spelling abilities. Researchers have been attempting to uncover the underlying mechanisms which cause dyslexia for over a century, and for many years it has been the most intensely researched of all the developmental disorders. The phonological deficit hypothesis has been the dominant explanation of reading difficulty over the last 30 years (Vellutino, 1981), and holds that dyslexia is caused by a language-specific deficit within the phonological system. The
proposed phonological deficit impairs the representation, storage and/or retrieval of speech sounds which detrimentally affect the learning of grapheme-phoneme correspondences (Boets, Wouters, Van Wieringen, & Ghesquiere, 2007; Bradley & Bryant, 1978; Brady & Shankweiler, 1991; Frith, 1985), and results in phonological representations of speech sounds that are degraded and ill-specified (Elbro, 1996; Snowling, 2000). However, the heterogeneous nature of dyslexia is well documented in the literature, and while research indicates that a phonological difficulty is characteristic of many children with dyslexia (Gallagher, Frith, & Snowling, 2000) the disorder has also been noted to manifest as difficulty in areas such as orthographic coding (Bowers & Wolf, 1993; Vidyasagar & Pammer, 2010), deployment of attention (Hari & Renvall, 2001; McGrath et al., 2011; Vidyasagar & Pammer, 2010; Willcutt, Pennington, & DeFries, 2000), language disorders (Bishop & Snowling, 2004; McArthur, Hogben, Edwards, Heath, & Mengler, 2000; Melby-Lervåg et al., 2012; Melby-Lervag, Lyster, & Hulme, 2012) speech sound disorder (Pennington & Bishop, 2009), sensory difficulties in visual and auditory temporal processing (e.g., Livingstone, Rosen, Drislane, & Galaburda, 1991; Lovegrove, Martin, & Slaghuis, 1986; Wolf, 1991; Wright & Conlon, 2009) working memory deficits (Kibby & Cohen, 2008), problems with balance and motor control (Nicolson & Fawcett, 1990; Ramus, 2004), and slow naming speed (Wolf & Bowers, 1999). Consequently, there have been several competing lines of investigation, some with single, and some with multiple cognitive predictors. These are most notably the double-deficit hypothesis (Wolf & Bowers, 1999), the dual-route model of reading (Castles & Coltheart, 1993) the visual and auditory temporal processing deficit hypotheses (Stein & Walsh, 1997; Tallal, 1980) and the cerebellar deficit theory of dyslexia (Nicolson & Fawcett, 1990; Nicolson, Fawcett, & Dean, 2001).

Current research findings indicate that single deficit explanations of dyslexia are inadequate, and while such studies confirm that poor phonological processing skills may be a
primary risk factor for impaired reading, a child is more likely to be diagnosed with dyslexia when broader impairments (e.g., difficulties with language, grammar, and poor vocabulary) persist through the school years. Such findings contribute to a large body of evidence that suggests a phonological deficit is only one of the multiple risk factors that accumulate towards a threshold, and has led to the development of frameworks that propose a multifactorial etiology for complex disorders such as dyslexia (Pennington et al., 2012).

**Family Risk**

Dyslexia is recognised as a familial and heritable language disorder (Pennington & Gilger, 1996; Pennington & Olson, 2005). There are descriptions of high familial aggregation of the disorder, with reports of approximately 33% to 66% of children with at least one dyslexic parent, later diagnosed as having dyslexia (Blomert & Willems, 2010; Leavett, Nash, & Snowling, 2014; Pennington & Lefly, 2001; Pennington & Olson, 2005; Plomin & Kovas, 2005; Snowling & Melby-Lervåg, 2016), and there is substantial evidence to indicate that genetic variance explains between 20% and 80% of the total variation in reading skills (Carrion-Castillo et al., 2017). Little is known about the molecular underpinnings of the disorder, however recent research using genome-wide association studies have indicated that the heritability of complex traits such as dyslexia is largely due to many genes of small additive effect (Carrion-Castillo et al., 2016; Plomin, 2013). Furthermore, genes act through environmental factors, and it has been proposed that variables such as the home literacy environment impact children’s literacy development (Conlon, Zimmer-Gembeck, Creed, & Tucker, 2006; Hamilton, Hayiou-Thomas, Hulme, & Snowling, 2016; Hood, Conlon, & Andrews, 2008; van Bergen, Zuijen, Bishop, & Jong, 2017). The home literacy environment involves a range of factors associated with parents’ and children’s attitudes and dispositions toward reading, and these can influence children’s literacy skills when certain environmental experiences are provided, or alternatively are not available
For example, the availability of books and time (Bus, van Ijzendoorn, & Pellegrini, 1995; Molfese, Molfese, & Modgline, 2001) as well as parental practices such as direct teaching of reading concepts (e.g., Senechal & LeFevre, 2002), have been found to have a positive association with the development of literacy skills such as decoding, oral language and reading comprehension. Further, how long parents spend reading with their child, as well as parents’ own reading behaviour has been found to have a significant impact on the early development of children’s oral language skill (Bus et al., 1995). In addition, family demographic factors such as socioeconomic status and differences in parental education level have been associated with literacy attainment (Phillips & Lonigan, 2009; Rutter & Maughan, 2005).

The risk for dyslexia is thought to be modulated by parental reading skills (Gilger, Hanebuth, Smith, & Pennington, 1996), which are shown to contribute unique variance to children’s reading fluency, even when controlling for phonological awareness (van Bergen, de Jong, Plakas, Maassen, & van der Leij, 2012), which is defined as "the ability to attend explicitly to the phonological structure of spoken words" (Scarborough, 1998). Children at family risk who were later diagnosed with dyslexia, have been shown to demonstrate deficits in reading (Snowling, Gallagher, & Frith, 2003; Wolff & Melngailis, 1994), language acquisition (McArthur et al., 2000; Spitz, Tallal, Flax, & Benasich, 1997), spelling (Schulte-Körne, Deimel, Müller, Gutenbrunner, & Remschmidt, 1996), and to have poorer phonemic recognition (the ability to perceive verbally presented basic units of language) than their typically developing peers (Pennala et al., 2010).

Studies have shown that literacy outcomes are distributed continuously among children at family risk of dyslexia, and many at-risk individuals who do not develop dyslexia show subclinical symptoms including poor reading fluency, spelling, phonological awareness, nonword repetition, short-term memory and reading comprehension (Hulme &
Snowling, 2016; Pennington & Lefly, 2001; Snowling et al., 2003). Family risk factors and the intergenerational transmission of the disorder have been found to play an important role in the manifestation of dyslexia, and the importance of identifying these pre-cursors has been highlighted, as early intervention has been found to result in better reading outcomes (Snowling et al., 2003; van Bergen, van der Leij, & de Jong, 2014). Consequently, recent studies investigating family risk of dyslexia have incorporated self-report measures of adult learning difficulties (e.g., Esmaeeli, Lundetræ, & Kyle, 2017) as self-report protocols have been found to be a good discriminator of adults with dyslexia, as well as a method for gathering multiple and reliable information (Gimenez, Luque, Lopez-Zamora, & Fernandez-Navas, 2015; Wolff & Lundberg, 2003).

1.2 Attention Deficit Hyperactivity Disorder (ADHD)

ADHD is diagnosed based on a set of behavioural indicators: maladaptive levels of inattention (predominantly inattentive type), hyperactivity and impulsivity (predominantly hyperactive/impulsive type) or the combined type, involving both inattention and hyperactivity/impulsivity (American Psychiatric Association, 2013; Eden & Vaidya, 2008). Children with the combined subtype (ADHD) are 1.5 times as likely to be identified in clinical samples than children with the inattentive subtype (AD), whereas children with AD are twice as likely to be identified in community samples than children with ADHD, suggesting that the disruptive behaviour exhibited by the combined groups leads to a greater likelihood of being clinically referred (Milich, Balentine, & Lynam, 2001).

The cognitive deficits underlying ADHD are not well understood (Shanahan et al., 2006), and a large body of research has repeatedly emphasized the complex and heterogeneous nature of the disorder (Boada et al., 2012; Castellanos, Sonuga-Barke, Milham, & Tannock, 2006; Nigg, 2006; Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005). Individuals with ADHD have been found to differ in core symptom combinations, severity of
impairment, as well as on individual, family, and social factors (Thapar & Cooper, 2016). There are also reports of a wide range (0.8% to 23.1%) in “subclinical” presentation of symptoms, which in the absence of a clinical diagnosis of ADHD may still result in impaired function (Balázs & Keresztény, 2014). Neuropsychological dysfunction has been explained with simple causal models of a single core impairment, such as an executive dysfunction due to deficient inhibitory control (Barkley, 1997), delay aversion (Sonuga-Barke, 2005; Sonuga-Barke, Taylor, Sembi, & Smith, 1992), state regulation (Sergeant, 2005) and slow speed of processing (Calhoun & Mayes, 2005; Clark, Prior, & Kinsella, 2002; Pennington, Groisser, & Welsh, 1993; Snow & Sapp, 2000). Executive functioning (EF) has received considerable attention; individuals with ADHD experience weaknesses in inhibition processes, planning, working memory and dual tasks (Rapport et al., 2008). Cognitive control or EF is a complex construct broadly defined as a collection of “top-down” controlled cognitive inputs that begin to develop in infancy and involves an array of processes such as attentional flexibility, inhibition, working memory and cognitive flexibility. These processes allow individuals to control their own behaviour, work toward goals, and manage complex cognitive processes. EF is described as conscious and effortful; it supports decision making by maintaining information about possible choices in working memory, and integrating this knowledge with information about the current context to identify an optimal course of action (Brosnan et al., 2002; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). Different components of the EF construct (i.e., inhibition/interference processes, processing speed, working memory etc.) are assumed by different theorists to be important in the etiology of ADHD (e.g., Nigg et al., 2005), however, inhibition processes and working memory are thought to be the two core elements that are critical for understanding EF (Pennington & Ozonoff, 1996; Verté, Geurts, Roeyers, Oosterlaan, & Sergeant, 2006). While the most consistent differences between groups with and without ADHD have been found on measures
of working memory, response inhibition and planning, none have been found necessary or sufficient to cause the disorder in isolation (Boada et al., 2012).

Some researchers propose a multifactorial perspective of ADHD that allows for multiple risk factors to exert effects at different ages (e.g., increase or decrease in symptomatology), with no defining separation of etiologic factors in childhood or adulthood (e.g., Faraone & Biederman, 2016). This perspective proposes that symptoms emerge only when the accumulation of genetic and environmental risk factors exceeds a threshold. However, two recent longitudinal, population studies have challenged this view, as they found that most cases of adult ADHD were not a continuation of childhood ADHD, and concluded that childhood onset ADHD, and adult onset ADHD may be two distinct conditions (Agnew-Blais et al., 2016; Caye et al., 2016; Moffitt et al., 2015).

1.3 Differentiating between AD and ADHD

One factor that has complicated interpretation of ADHD is the lack of distinction between symptoms of inattention and hyperactivity/impulsivity (Willcutt, Pennington, Olson, Chhabildas, & Hulslander, 2005). While studies that have investigated the cognitive differences between the three ADHD subtypes (e.g., Baeyens, Roeyers, & Walle, 2006; Bonafina, Newcorn, McKay, Koda, & Halperin, 2000; Chhabildas, Pennington, & Willcutt, 2001; Fair, Bathula, Nikolas, & Nigg, 2012; Faraone, Biederman, Weber, & Russell, 1998; Riccio, Homack, Jarratt, & Wolfe, 2006; Song & Hakoda, 2014) have reported differing results, there is evidence to indicate that groups with the combined and inattentive subtypes can be distinguished on several important variables (Milich et al., 2001). For example, Diamond (2005) proposed that rather than being distractible, the difficulty for children with AD may instead relate to a lack of motivation (under-arousal), rather than inhibitory control. Challenge or risk can be key to keeping their attention, and while adequate motivation may result in good performance, under-arousal of neural systems (primarily the striatum) makes it
difficult for them to sustain a sufficient level of performance or attention for mundane or boring tasks. Diamond (2005) hypothesised that ADHD and AD are two distinct disorders, with differing cognitive (working memory deficit in AD, and inhibitory control deficit in ADHD) and behavioural profiles, as well as different patterns of comorbidities. Several other researchers have also suggested that these subtypes are dissociable and should be viewed as distinct disorders (Baeyens et al., 2006; Barkley, 2001; Milich et al., 2001), and there has been some support from neurological research for differences in brain structure between the ADHD and AD subtypes. For example, a study by Li and colleagues (2014) found evidence for differential cortical wiring pathways between the two subtypes; AD was related to abnormalities in the temporo-occipital areas, while the combined subtype was related to abnormalities in the frontal-subcortical circuit, the fronto-limbic pathway, and the temporo-occipital areas, suggesting some neurophysiological distinctions between the subtypes (Li, He, et al., 2014).

1.4 Dyslexia and ADHD

In the past two decades, cognitive and developmental research has focussed on understanding the nature and underlying etiology of co-occurring dyslexia and ADHD (Boada et al., 2012), and these findings have demonstrated an extensive overlap between the symptoms of these disorders. For example, common deficits have been found in attention mechanisms (e.g., Facoetti & Molteni, 2001; Konrad, Neufang, Hanisch, Fink, & Herpertz-Dahlmann, 2006; Vidyasagar & Pammer, 2010), tasks that rely on speed of processing such as rapid automatic naming (Catts, Gillispie, Leonard, Kail, & Miller, 2002; Landerl et al., 2013; Shanahan et al., 2006; Willcutt, Pennington, et al., 2005; Wolf, 1997), poor reading comprehension (Ghelani, Sidhu, Jain, & Tannock, 2004; Miller et al., 2013), and problems with executive functions such as working memory (Moura et al., 2017; Moura, Simões, & Pereira, 2015; Swanson, Zheng, & Jerman, 2009). The evidence for the presence of an
overlap between the symptoms of dyslexia and ADHD is supported by studies of community
twin samples, which indicate that common genetic influences are involved in dyslexia and
ADHD, both together and separately (Gayán et al., 2005; Loo et al., 2004; Willcutt,
Pennington, Olson, & DeFries, 2007; Willcutt et al., 2002).

Attention is a multifaceted system comprising several attention networks (Posner &
Petersen, 1990; Tsal, Shalev, & Mevorach, 2005). Four semi-independent attentional
subsystems have been proposed; attention orienting, selective attention, sustained (or
focussed) attention and executive attention (Tsal et al., 2005). Sustained, or focussed
attention involves the ability to maintain attention over a long period of time (Stern & Shalev,
2013), and is thought to share a close association with reading, as there is evidence to
indicate that these attention mechanisms play a critical role in translating print to speech, and
for fluent reading (Reynolds & Besner, 2006). Studies that have separated the ADHD
symptom domains have found close associations, both phenotypically and genetically
(Willcutt, Betjemann, Wadsworth, et al., 2007) between reading and the inattentive (AD)
symptom domain of ADHD, and several studies have found that the heritability of dyslexia
and ADHD traits in normative samples is more pronounced for inattention (.39% to .60%)
than for hyperactivity/impulsivity (.05 to .35%; Greven, Harlaar, Dale, & Plomin, 2011;
Greven, Rijsdijk, Asherson, & Plomin, 2012; Paloyelis, Rijsdijk, Wood, Asherson, & Kuntsi,
2010; Plourde et al., 2015; Willcutt, Betjemann, et al., 2010). These associations are
supported by recent neurological research that used a functional magnetic resonance imaging
(fMRI) connectivity analysis to compare the connectivity profiles of younger (mean age 9
years) and older (mean age 20 years) dyslexic and typical readers. The study found that the
groups with dyslexia demonstrated decreased connectivity between prefrontal attentional
regions and posterior reading systems, relative to the typical readers, suggesting children with
dyslexia have more difficulty integrating visual information and modulating their attention to
visual stimuli (Finn et al., 2014). The inability to attend to visual stimuli would make it difficult for dyslexic readers to recognise words based on their visual properties, and is consistent with the many studies that have observed a closer association between reading and inattention, than between reading and hyperactivity (Boada et al., 2012; Miranda, Jesús Presentación, Siegenthaler, Colomer, & Pinto, 2011; Willcutt & Pennington, 2000; Willcutt et al., 2001; Willcutt, Pennington, et al., 2007). Accordingly, recent research studies investigating the associations between dyslexia and ADHD are more likely to separate the attention and hyperactive/impulsive symptom dimensions of ADHD, or to examine the results of each symptom dimension independently (e.g., Arnett et al., 2012; Lundervold, Meza, Hysing, & Hinshaw, 2017).

1.5 Single versus multiple deficit explanations of dyslexia and ADHD or AD

Despite strong evidence for the overlap between dyslexia and ADHD, as well as the heterogeneity of symptom expression within each disorder, the neuropsychological models used to investigate the disorders have typically involved a single deficit framework (Willcutt et al., 2013). However, single deficit models cannot explain the pervasive heterogeneity found within each disorder, or the co-occurrence between the developmental disorders. The accumulation of evidence from multiple levels of analysis has seen researchers argue in recent theoretical models that these disorders result from the additive and interactive effects of multiple genetic and environmental weaknesses and strengths (e.g., McGrath et al., 2011; Pennington et al., 2012; Willcutt, Sonuga-Barke, Nigg, & Sergeant, 2008). It is generally accepted that the proximal cognitive cause of dyslexia is a phonological deficit (Vellutino, Fletcher, Snowling, & Scanlon, 2004), whereas the predominant account of ADHD sees it as arising from an impairment in inhibitory functions (Barkley, 1997). However, the frequent co-occurrence of reading and AD or ADHD raises the issue of whether common causal mechanisms at either the cognitive or biological levels may be involved (Light, Pennington,
Gilger, & DeFries, 1995; Stevenson et al., 2005). The multiple deficit model (MDM: Pennington, 2006) is a recent framework designed to explain these associations. This model supports the heterogeneous nature of neurodevelopmental disorders and proposes that individuals with disorders such as dyslexia or ADHD will show evidence of deficits (both unique and common) in multiple neurocognitive domains (e.g., reading, attention and language and/or motor deficits), which are the result of multiple genetic and environmental risk (and protective) factors. It has now been proposed that in order to capture the heterogeneity of symptomatology inherent in developmental disorders such as dyslexia and ADHD, impairments should be viewed as varying along a single dimension that spans a spectrum, with the identification of a primary impairment as a starting point for further investigation, rather than being the endpoint of diagnosis (e.g., Heidbreder, 2015).

1.6 Dyslexia and ADHD or AD as dimensional versus categorical disorders

Researchers investigating the distribution of reading disorder (Shaywitz, Shaywitz, Escobar, Fletcher, & Makuch, 1992) and ADHD (Greven et al., 2016) have noted that these are continuously distributed disorders that vary in severity. This proposal is supported by substantial research that indicates high rates of clinical as well as sub-clinical difficulties for children assessed for dyslexia (e.g., Hulme & Snowling, 2016; Pennington & Lefly, 2001; Snowling et al., 2003) and ADHD (see Balázs & Keresztény, 2014 for a review). Moreover, studies investigating the comorbid condition have found that even when individuals do not meet clinical criteria for both disorders, they often exhibit subclinical symptoms of the other disorder (Willcutt, Betjemann, Pennington, et al., 2007). Consequently, the use of categorical groupings to diagnose dyslexia and ADHD or AD relies upon the use of an arbitrarily appointed cut-off, and its use may result in a substantial number of children failing to be diagnosed for a reading or behavioural difficulty. Without access to remedial services these
children may be at risk for poorer academic and social outcomes, therefore, clinical and subclinical symptoms were included in the current research project.

1.7 Aims and Overview

This research project had several aims (see Figure 1.1 for the aims and how the integrated group of studies achieved those aims). The primary aim in Chapter 3 was to investigate the prevalence of single and multiple developmental disorders in a group of children predominantly identified with symptoms of dyslexia, AD or ADHD, and to demonstrate that the patterns of disorder in the current sample was consistent with previous research that has found most children with one developmental disorder will meet clinical or subclinical criteria for at least one other developmental disorder. It was hypothesised that the majority of participants in the sample would show evidence of more than one disorder, particularly dyslexia + AD or dyslexia + ADHD (e.g., Dittman, 2016; Kaplan, Wilson, Dewey, & Crawford, 1998; Pitcher, Piek, & Hay, 2003). The second aim was to examine whether the severity of deficits would be greater for children with multiple disorders, compared to children with just a single disorder, as some research has reported children with multiple developmental disorders to have more severe impairments than those with a single disorder (e.g., Willcutt, Betjemann, Pennington, et al., 2007). In addition, some studies report higher prevalence rates between certain developmental disorders (e.g., dyslexia + AD or ADHD, dyslexia + SLI, ADHD + DCD), suggesting children with the combination of these disorders may exhibit more specific weaknesses in certain developmental and learning domains than children without this combination of deficits. Based on these stronger associations the following predictions were made regarding the expected severity of difficulties. It was hypothesised that the group with dyslexia + AD or ADHD would show greater evidence of reading, attention, language, and motor deficits than the groups with a single disorder (e.g., Helland, Posserud, Helland, Heimann, & Lundervold, 2016; Oliver,
Johnson, Karmiloff-Smith, & Pennington, 2000). Further, the group with dyslexia alone was expected to be more impaired on the language measures than the groups with AD or ADHD alone, who would perform more poorly than the control group (e.g., Helland et al., 2016). In contrast, the groups with AD or ADHD were expected to show more evidence of motor skill impairment than the group with dyslexia alone, who were not expected to differ from the control group (e.g., Kaplan, Dewey, Crawford, & Wilson, 2001). This study is unique in that it is the first study to investigate the prevalence of clinical and subclinical dyslexia, ADHD, AD, language and motor impairment in a single sample of children.

Familial risk has been identified as a primary risk for developing dyslexia and ADHD or AD, and early identification is paramount for optimal remediation. However, there are few adult self-report protocols designed to screen for dyslexia or ADHD, thus in Chapter 4 we aimed to validate a recent self-report questionnaire and use the measure to identify those children at family risk of the disorders. This was the first Australian study to validate the ARQ/ASRS self-report scales independently of the authors, and the investigation of the ADHD and AD subtypes separately using this protocol is novel to this study. The second aim was to determine the prevalence of symptoms of dyslexia and ADHD in the adult sample, and then to examine whether parent-reported symptomatology was associated with the prevalence of single or multiple disorders in their children. Based on previous research that found children with dyslexia (Snowling & Melby-Lervåg, 2016; van Bergen et al., 2011) and ADHD (Smalley et al., 2000; Starck, Grünwald, & Schlarb, 2016; Thissen, Rommelse, Altink, Oosterlaan, & Buitelaar, 2014) are more likely to have a parent with the same disorder, it was hypothesised that children from families with parents reporting high symptomatology of dyslexia, ADHD or AD would be more likely to exhibit these disorders than children without a family risk. In Chapter 5 we investigated children with developmental dyslexia only. The aim of this study was to replicate and extend the study by
Pennington and colleagues (2012), to determine which developmental framework (e.g., a single, double, multiple, or intergenerational multiple deficit model of dyslexia) was the best predictor of reading fluency, using measures of phonological decoding, language skills, and rapid automatic naming, in a group of children with and without a family risk of dyslexia. If the single deficit model was supported, it was expected that all children with dyslexia would have poor phonological processing. In this model, phonological processing was expected to explain more variance in reading fluency than either rapid naming or language skills, which would not add significant variance to the explanation of reading fluency. If the double deficit model was supported, phonological processing and rapid naming were each expected to make unique and independent contributions to the explanation of reading fluency. It was expected that children would be impaired on phonological processing, rapid naming or both. If the multiple deficit model was supported, phonological processing, language skills and rapid naming were each expected to make unique and independent contributions to the explanation of reading fluency. It was expected that children would show different patterns of phonological, language, and rapid naming impairments that could explain their reading difficulties. Finally, if reports of reading difficulties in mothers and fathers influenced their children’s reading skills, a significant association was expected between parent-reported difficulties and child reading fluency. After controlling for parent self-reported reading skills, it was expected that phonological processing, language skills and rapid naming would make additional contributions to the explanation of children’s reading fluency. This study is the first to include the intergenerational model within this framework.

The research findings for the co-occurrence between dyslexia and ADHD have been mixed. In order to establish which variables are unique or common to the disorders the study presented in Chapter 6 was designed to replicate previous research that has found (a) poor phonological processing and rapid automatic naming are associated with dyslexia only (e.g.,
Boada & Pennington, 2006), (b) poor interference control is associated with ADHD and AD only (e.g., Berger & Posner, 2000), and (c) poor working memory (e.g., Gathercole, Alloway, Willis, & Adams, 2006; Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005), slow rapid naming (e.g., Shanahan et al., 2006) and poor reading comprehension (e.g., Blachman, 2000; Brock & Knapp, 1996) are deficits common to dyslexia, ADHD and AD, and which explain the co-occurrence between the disorders. Using a continuous variable approach, the study allowed for the investigation of symptom severity, independent of diagnostic groupings. In addition, the unique influence of AD alone has not been previously investigated within this framework. The first aim of the study was to examine the extent to which poor phonological processing skills are unique to dyslexia, and the extent that impaired interference control is unique to ADHD and AD. Based on previous findings it was expected that difficulties in phonological processing would be associated with dyslexia only, and an impairment in interference control would be associated with ADHD and AD only. Using the multiple deficit framework, the second aim was to determine the extent to which difficulties with working memory, rapid naming, and reading comprehension explain the co-occurrence between dyslexia and AD or dyslexia and ADHD. Three hypotheses were proposed. First, that there would be a significant association between reading fluency, phonological processing and rapid naming. Regardless of symptoms of ADHD or AD, children with poorer reading fluency were expected to perform more poorly on phonological processing and rapid naming measures. Second, it was expected that children with ADHD or AD (with or without dyslexia) would make more errors and have slower response times on the incongruent interference control condition than the other groups. Third, that children with more symptoms of dyslexia and ADHD or AD would show evidence of poorer working memory and reading comprehension than those with fewer symptoms. Children with very
severe symptomatology were not expected to differ from one another because of the shared nature of the deficit.

Finally, in Chapter 7 the key findings of this thesis and its conclusions are discussed. From a theoretical perspective, this research contributes to our understanding of the association between dyslexia and attention deficit disorders. It highlights the high rates of subclinical presentations and contributes to the case for conceptualising the symptoms of dyslexia, ADHD and AD as continuously distributed disorders, as the use of categorical groupings leaves many children with unidentified disorders that negatively impact their academic performance. This research is expected to contribute to the better identification of children with reading and attention difficulties, which will ensure that the appropriate treatments and support (both reading interventions and cognitive training for inattention) are implemented to aid academic achievement. It also highlights the transfer of parental liability for reading difficulties, and the importance of the identification of children at family risk. This is critical for two reasons. First, there is a higher chance of ameliorating the symptoms of the disorder if remedial strategies are implemented before reading becomes delayed, and second, the majority of at-risk children without clinical symptoms still display subclinical difficulties that impact academic performance, and these children are at high risk of being overlooked for intervention.

This was a small self-selecting community sample of children who were classified with dyslexia based on reading fluency scores and this makes it difficult to generalise the findings to other studies. Future studies should use a larger sample size and include other classification variables such as word identification. Further, additional measures of phonological ability should be included, such as phonological memory (e.g., short-term memory for phonologically coded material) and orthographic coding (e.g., visual representations to form, store and recall words).
<table>
<thead>
<tr>
<th>Study 1 (Chapter 3)</th>
<th>Study 2 (Chapter 4)</th>
<th>Study 3 (Chapter 5)</th>
<th>Study 4 (Chapter 6)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Purpose:</strong> To estimate the prevalence of single and multiple developmental disorders in a sample of academically underperforming children, and to compare the severity of the symptoms of the different disorders in the single and multiple disorder groups.</td>
<td><strong>Purpose:</strong> To validate a self-report protocol for parents designed to screen for the presence of dyslexia and/or ADHD. To use the validated protocol to estimate the prevalence of dyslexia and/or ADHD in the parent sample. Finally, to determine if parent-reports of symptoms of dyslexia, ADHD, or AD were associated with the prevalence of single (dyslexia, ADHD, AD, Specific Language Impairment) or multiple disorders in the children assessed in the current study.</td>
<td><strong>Purpose:</strong> To determine whether a single, double, multiple, or Intergenerational Multiple Deficit Model is the best predictor of reading fluency.</td>
<td><strong>Purpose:</strong> To examine the extent that difficulties with phonological processing and rapid naming are unique to dyslexia, and the extent that interference control is unique to ADHD and AD. Determine the extent that difficulties with working memory and reading comprehension explain the co-occurrence between dyslexia + ADHD or AD using the Multiple Deficit Model.</td>
</tr>
<tr>
<td><strong>Design:</strong> Cross-sectional. N = 72</td>
<td><strong>Design:</strong> Cross-sectional. Parent group N = 117, Child group N = 64.</td>
<td><strong>Design:</strong> Cross-sectional. N = 72 children, as per study 1.</td>
<td><strong>Design:</strong> Cross-sectional. N = 72 children, as per study 1.</td>
</tr>
<tr>
<td><strong>Framework:</strong> A series of one-way ANOVA’s and post-hoc analyses. Percentage of children with dyslexia, ADHD or AD alone, and in combination with SLI/DCD.</td>
<td><strong>Framework:</strong> Percentage of parents classified with symptoms of dyslexia and/or AD, ADHD. Percentage of children at family risk/no family risk with dyslexia and/or AD.</td>
<td><strong>Framework:</strong> A combination of correlation, standard multiple regression and hierarchical regression analysis was used to evaluate the hypotheses posed.</td>
<td><strong>Framework:</strong> Two mixed factorial ANOVAs assessed the performance of the groups on the interference control task. A 2 × 2 × 2 analysis was conducted for each the mean correct response time and percentage correct for the interference control task. In the second analyses, the AD group was used, instead of the ADHD group. Moderated regression analyses were conducted to assess the contribution of phonological processing, as well as all variables potentially shared between reading and attention difficulties.</td>
</tr>
<tr>
<td><strong>Thesis Aim:</strong> 1 and 2</td>
<td><strong>Thesis Aim:</strong> 3 and 4</td>
<td><strong>Thesis Aim:</strong> 5</td>
<td><strong>Thesis Aim:</strong> 6 and 7</td>
</tr>
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**Figure 1.1** Outline of thesis aims and corresponding empirical studies
Chapter 2: Sample Description and Procedure

2.1 Sample Description

The sample was obtained from several community sources including three local private schools ($n = 34$), two private psychology practices ($n = 16$), an education support practice ($n = 2$), a private Facebook group for dyslexia support ($n = 25$) and by word of mouth ($n = 5$). All children had English as a first language. Two children spoke another language at home, however information was not provided to indicate when the other language was learnt. All children in the sample were considered by parents either to be academic underperformers in school, have dyslexia, ADHD or both. On this basis, the recruitment process was self-directed, and not the result of a mass screening process. A total of 82 children aged between 9 and 11 years (64 boys and 18 girls) who were attending local primary schools (Grade 3 to 6) were assessed over a two-year period. This age range was chosen for greater stability in abilities, and for more homogenous groups, as norms for standardised tests may differ across a broader age range (Dyck, Piek, Kane, & Patrick, 2009).

Based on parental responses to a developmental questionnaire (parent-completed measure), the data of 4 children was excluded based on the presence of other neurological or pervasive developmental disorders which may have impaired the child’s ability to undertake or complete measures. The data of a further 6 children were excluded as there was no associated behavioural information (i.e., no completed Parent or Teacher Conners-3 Rating Scales), leaving a total of 72 children (57 boys and 15 girls) who were included in the study. Of the final 72 participants, 39% had a previous diagnosis of dyslexia, 6% had a previous diagnosis of co-occurring dyslexia + ADHD, and only one child had a previous diagnosis of ADHD alone. All diagnoses of dyslexia were made by a trained psychologist, while ADHD diagnoses were made by a paediatrician. Although more than half the child sample had no previous diagnosis, they were recruited on the basis that their parents had concerns regarding
their academic performance, and consequently over half (65%) the participants were receiving remedial help for literacy and/or numeracy, either at school or privately. Further, many of the parents personally reported symptoms of reading and attention difficulties, and these parents may have been particularly vigilant in identifying subtle, subclinical difficulties in their children.

The parent sample included 117 parents (64 mothers and 53 fathers) who completed the Adult Reading Questionnaire (ARQ), and the short-form of the Adult ADHD Self-Report Scale (ASRS: Kessler et al., 2005). Ethical clearance for the study was granted by the Griffith University Human Ethics Committee.

### 2.2 Assessment Procedure

Caregivers, schools, and teachers provided written consent, and child participants gave written assent prior to the commencement of testing. There was one data collection phase which was conducted over a two-year period. The entire assessment battery required approximately three hours to complete. Participants were allocated three 1 hour and 20 minute sessions, or two 2 hour sessions to allow for numerous breaks between tasks, to reduce test fatigue, and ensure that concentration was maintained. Consequently, actual testing times varied depending on each child’s capacity and needs. Testing was administered on an individual basis in a quiet room free from visual and acoustic distractions at the participant’s school or at a private psychology clinic.

All child participants were administered the tests in the following order:

1. The subtests from the Wechsler Intelligence Scale for Children (WISC-IV; Weschler, 2003): digit span forward, digit span backward, and letter-number sequencing;  
2. subtests from the Wechsler Individual Achievement Test Second Edition (WIAT-II; Wechsler, 2007): written comprehension and pseudoword decoding;  
3. subtests from the Clinical Evaluation of Language Fundamentals-Fourth Edition (CELF-IV; Semel, Wiig, & Secord, 2006):
Concepts and Following Directions, Recalling Sentences, Formulated Sentences, and Word Classes 2; (4) the Sight Word Efficiency (SWE) and Phonemic Decoding Efficiency (PDE) subtests (Form A) from the Test of Word Reading Efficiency-2 (TOWRE-2; Torgesen, Wagner, & Rashotte, 2012); (5) the Elision subtest from the Comprehensive Test of Phonological Processing (CTOPP; Wagner, Torgesen, & Rashotte, 1999) (6) the Rapid Automatic Naming (RAN; Denckla & Rudel, 1976) subtests: RAN digits and RAN letters; and finally (7) the computerised Eriksen Flanker task (Eriksen & Eriksen, 1974). All caregivers received a written summary of their child’s performance on all neuropsychological measures, and when parental consent was given, this information was also provided to the child’s school.

Parents were asked to complete the Conners-3 (Conners, 2008) parent-report, as well as the Adult Reading Questionnaire (ARQ) and the short-form of the Adult ADHD Self-Report Scale (ASRS; Snowling, Dawes, Nash, & Hulme, 2012). Those teachers who provided consent were asked to complete the Conners-3 teachers’ version. Parents and teachers received, completed and returned the Conners-3 in a sealed envelope. The reports were completed separately; a child’s parent and teacher did not each see the other’s assessment. The full description of each assessment measure is provided in the following study chapters as they are introduced or first used.
Chapter 3: Prevalence of single and multiple developmental disorders in a sample of underperforming children

3.1 Single versus multiple developmental disorders

The study of developmental disorders is an important part of cognitive neuroscience (Oliver et al., 2000); however, over recent decades there has been considerable theoretical debate regarding the best approach to use. Traditional models posit a direct association between damage to a specific cortical brain region and a specific cognitive deficit (e.g., a single phonological deficit in dyslexia). In contrast, others propose that damage to a specific cortical region may have cascading effects that result in multiple cognitive weaknesses (e.g., phonological, language and attention deficits in dyslexia) that may differ in severity, and could occur because of the connectivity between brain regions (Oliver et al., 2000). Cascade effects imply impairment in a range of cognitive functions, rather than a single isolated deficit (Dyck & Piek, 2014), and this notion of multiple cognitive weaknesses is supported by the developmental research literature that has established the common co-occurrence between neurodevelopmental disorders. Current evidence indicates that approximately 40% of school-aged children with one developmental disorder will also meet diagnostic criteria for at least one other developmental disorder (e.g., Kadesjo & Gillberg, 1999; Kaplan, Crawford, Wilson, & Dewey, 1997; McArthur et al., 2000; Pitcher et al., 2003; Rasmussen & Gillberg, 2000; Rochelle & Talcott, 2006; Sergeant, Piek, & Oosterlaan, 2006; Willcutt & Pennington, 2000). For example, children with dyslexia are also likely to meet clinical criteria for a combined Attention Deficit Hyperactivity Disorder (ADHD) or an Attention Deficit (AD) Disorder (Kaplan et al., 1998; Pitcher et al., 2003), a Specific Language Impairment (SLI; e.g., Bishop & Snowling, 2004; McArthur et al., 2000; Melby-Lervåg & Lervåg, 2012), and/or a Developmental Coordination Disorder (DCD; e.g., Brookes, Nicolson, & Fawcett, 2007; Ramus, Pidgeon, & Frith, 2003). The rates of co-occurrence among dyslexia, ADHD,
SLI and DCD are high, and have been reported to range from 30% to 90% (Dewey, Cantell, & Crawford, 2007; Hill, Bishop, & Nimmo-Smith, 1998; Kirby & Sugden, 2007; Taurines et al., 2010; Taurines et al., 2012; Willcutt, Pennington, et al., 2010). Consequently, there is growing awareness that developmental disorders are nonspecific and heterogeneous, and that a diagnosis of one disorder significantly increases the likelihood of meeting criteria for at least one other disorder (Dewey & Bernier, 2016). Some researchers have now proposed that “specific” or pure disorders are rare in development (Gooch, Hulme, Nash, & Snowling, 2014), suggesting prevalence estimates would be higher for co-occurring, rather than single disorders. The focus of the current study was to investigate the prevalence and severity of single and multiple developmental disorders in a sample of school-aged children with symptoms of dyslexia and/or ADHD or AD.

3.2 Co-occurrence between dyslexia and ADHD or AD

Developmental dyslexia and ADHD are two of the most prevalent and complex childhood disorders (Gooch, Snowling, & Hulme, 2011). Dyslexia accounts for approximately 80% of the learning disorders, and is reported to occur in approximately 5% to 10% of the population (Germanò, Gagliano, & Curatolo, 2010; Willcutt, Pennington, et al., 2005), although some estimates are as high as 17.5% (Eden & Vaidya, 2008; Shaywitz & Shaywitz, 2008). According to the Diagnostic and Statistical Manual of mental disorders (DSM-IV; American Psychiatric Association, 2000), the prevalence of ADHD is reported to be between 3% and 7% in school-aged children, and a recent review of the worldwide ADHD prevalence literature (Polanczyk, de Lima, Rohde, Horta, & Biederman, 2007; Thomas, Sanders, Doust, Beller, & Glasziou, 2015; Willcutt, 2012) estimated between 6.7% to 7.8% of children are affected, with an overall pooled estimate of 7.2%. The prevalence of ADHD is influenced by the technique used in making a diagnostic decision. Clinician based assessment produces the lowest estimates, with teachers reporting more symptomatology in
students than do parents. Consequently, there are reports of over and under identification of the presence of the disorder, based on the criteria used (Angold, Erkanli, Egger, & Costello, 2000; Thomas et al., 2015).

There is substantial literature documenting a significant comorbidity between dyslexia and ADHD (Friedman, Chhabildas, Budhiraja, Willcutt, & Pennington, 2003), with estimates of co-occurrence reported to range from approximately 15% to 60% (Bental & Tirosh, 2007; Czamara et al., 2013; Del’Homme, Kim, Loo, Yang, & Smalley, 2007; Miranda et al., 2011), depending on the criteria for inclusion (August & Garfinkel, 1990; Dykman & Ackerman, 1991; Shaywitz, Shaywitz, Fletcher, & Escobar, 1990; Willcutt & Pennington, 2000). This rate is substantially higher than would be expected by chance, which is around 5%. Studies have shown that within samples of children with dyslexia, the rate of ADHD ranges from as low as 9% to as high as 60%, while 15% to 45% of individuals with ADHD also meet criteria for dyslexia (Carroll, Maughan, Goodman, & Meltzer, 2005; Sexton, Gelhorn, Bell, & Classi, 2012; Willcutt & Pennington, 2000; Willcutt et al., 2001). A substantial number of children with the ADHD inattentive subtype also exhibit reading difficulties (e.g., Dally, 2006; Dittman, 2016), and this is evident in clinical as well as community samples (Mayes & Calhoun, 2006; Willcutt & Pennington, 2000). A recent study that investigated the overlap between dyslexia and AD found that of the 78 children in a sample with AD, 64% also had dyslexia, and of the 158 children in the sample with dyslexia, 31.6% also had co-occurring AD (Heikkilä, Torppa, Aro, Närhi, & Ahonen, 2016). The co-occurrence between these disorders is often stable over time, so even when individuals with dyslexia or ADHD do not meet full criteria for both disorders, they often show evidence of subclinical symptoms of the other disorder (Willcutt, Betjemann, Pennington, et al., 2007). The prevalence data on subclinical ADHD in children and adolescents is wide-ranging (0.8% to 23.1%), partly explained by differences in the criteria used to define the subclinical range (Balázs &
Keresztény, 2014). Although there are many reports that children with ADHD have poorer reading skills than expected, few studies have evaluated the rate of sub-clinical estimates of dyslexia and ADHD.

3.3 The overlap of dyslexia and ADHD with other developmental disorders

A large body of research has demonstrated the common co-occurrence between dyslexia and ADHD (Friedman et al., 2003), however, there is also considerable evidence to indicate strong associations between dyslexia and SLI (Bishop, 2008; McArthur et al., 2000; Pennington & Bishop, 2009), as well as ADHD and SLI (McGrath et al., 2008; Mueller & Tomblin, 2012), although it has been argued that the prevalence estimates between ADHD and SLI may be inflated (Redmond, 2002). An association between ADHD and DCD has also been demonstrated (Kaplan et al., 2001), however, some research has reported no significant co-occurrence between dyslexia and DCD without the presence of ADHD (Kaplan et al., 2001; Rochelle & Talcott, 2006).

The overlap of dyslexia and SLI

Specific language impairments reflect oral language competency and involve both receptive and expressive language functioning (e.g., understanding spoken language and the ability to communicate through oral language) as well as pragmatics (e.g., the ability to use language for social communication). The disorder commonly occurs with dyslexia (Bishop, 2008; McArthur et al., 2000; Pennington & Bishop, 2009) and some researchers have posited that dyslexia and SLI represent variants of the same underlying cognitive deficit, rather than distinct disorders (Kamhi & Catts, 1986; Tallal, Allard, Miller, & Curtiss, 1997). However, others have concluded that despite close behavioural similarities, a unidimensional model underestimates the independent influence of the semantic and syntactic deficits that characterise SLI (e.g, Bishop & Snowling, 2004). For example, although Bishop and Snowling (2004) propose that children with dyslexia and SLI share similar phonological
processing deficits, they hypothesize there are additional cognitive deficits associated with SLI that result in the oral language difficulties.

Estimating the prevalence of children with SLI is hindered due to lack of agreement regarding key inclusion and exclusion criteria (Bishop, 2014). Rates of persistence vary widely from 11% to 92% (Snowling, 2000; Tomblin et al., 1997; Tomblin, Zhang, Buckwalter, & O'Brien, 2003), with a recent population-based survey estimating the total population prevalence at 9.92% (Norbury et al., 2016). Further, age has been found to be an important variable when estimating language impairment in children, as research has observed both resolving and persisting patterns of development among children with language difficulties. For example, although Hayiou-Thomas and colleagues (2014) found that having an expressive language impairment at 4 years of age was a risk factor for a language impairment (or at least below-average language skills) at 12 years, there was also a large amount of variability in outcome. The study found that approximately one third of children identified with an early language impairment went on to have average or above-average language skills at 12 years, (Hayiou-Thomas, Dale, & Plomin, 2014), and this is in accord with other studies that have observed the resolution of language difficulties in the early years of language acquisition (e.g., Rescorla, 2002). However, they also found that one third of the children who met criteria for a language impairment at 12 years had no reported language difficulties at 4 years of age.

Similarly, an earlier study by Bishop and colleagues followed a clinically referred sample of children at 4, 5.5, and 8.5 years, with a further follow-up in adolescence at 15.5 years of age (Bishop & Adams, 1990; Snowling, Bishop, & Stothard, 2000; Stothard, Snowling, Bishop, Chipchase, & Kaplan, 1998). This study found that of the children identified with a language impairment at 4 years, 35% of those who appeared to have resolved their language difficulty later relapsed in adolescence. Several explanations have
been proposed to explain the variability in the developmental trajectory of SLI. For instance, it has been suggested that the language difficulties of children who receive effective treatment or interventions may be ameliorated (Hayiou-Thomas et al., 2014), or that early language difficulties later manifest as literacy problems, rather than obvious deficits in oral language (e.g., Stothard et al., 1998).

**The overlap of ADHD and SLI**

Although language difficulties are not included in the core diagnostic criteria for ADHD (Helland, Biringer, Helland, & Heimann, 2010), children with an attention deficit and/or hyperactivity disorder are also more likely to be identified with SLI than their typically developing peers (McGrath et al., 2008; Mueller & Tomblin, 2012; Willcutt & Pennington, 2000). The rates of co-occurrence between SLI and ADHD is reported to be between 30% and 50% (Cohen et al., 2000; Sciberras et al., 2014), although it has been argued that these rates are inflated, and reflect the overlap of symptoms from distinct causal pathways (Redmond, 2002, 2016). Indeed, Redmond (2002) cautions that the relationship between behavioural and language difficulties can be an artefact of behavioural rating scales which incorporate indicators of language difficulties as items to show behavioural difficulties. For example, many ADHD rating scales include items that assess academic difficulties that could reflect a receptive language impairment, or an attention deficit (e.g., does not follow through on instructions; does not seem to listen to what is being said; Redmond, 2002). Further, most studies on language difficulties in children with ADHD are based on clinical samples, and it has been suggested these findings are likely to represent the most severe cases (Sexton et al., 2012). However, an early population-based study found a substantial overlap between AD and language difficulties. Tirosh and Cohen (1998) examined AD and language impairment in a sample of 3,208 children aged 6 to 11 years. The study found that 45% of participating children fulfilled the criteria for a diagnosis of AD plus at least one language
impairment (i.e., vocabulary, syntax, receptive, expressive, reading fluency, text recall or pragmatic). The disorders were diagnosed using self-report (teacher’s behavioural questionnaire), as well as objective measures (language and attention tests). Children with comorbid behavioural disorders were excluded from these estimates.

Other non-clinical studies have also demonstrated that it is common for children with symptoms of dyslexia and/or ADHD to exhibit receptive and expressive language impairment (Bignell & Cain, 2007; Helland et al., 2016; Sciberras et al., 2014). For example, Helland and colleagues (2016) used a questionnaire to investigate language impairment in a population sample of 5,672 children aged 7 to 9 years who had symptoms of ADHD, dyslexia, or ADHD + dyslexia. The study aimed to examine whether the groups could be differentiated from one another, as well as from a control group on different aspects of language (e.g., language composite, pragmatics, phonology, expressive and receptive language). In the total sample of children with ADHD (regardless of dyslexia status), 58.5% were identified with a language impairment, with a similar pattern of 55.7% for children with dyslexia. The majority (80.7%) of children with ADHD + dyslexia, and almost half of both the dyslexia (46.0%) and ADHD (42.6%) groups were identified with a language impairment, compared to a minority in the control group (5.7%). The study used a language composite as a continuous measure to compare the groups. The language composite was comprised of four items relating to language impairments: (1) cannot pronounce certain words or sounds; (2) cannot elaborate, explain, or express himself or herself; (3) has difficulties understanding things that are being said; and (4) has difficulties having a conversation with others, and which were scored on a 3-point scale (0 = not true, 1 = somewhat true, and 2 = certainly true). The language composite score was calculated by summing the scores of items 1 to 4 and was then dichotomized. Language impairment was defined as a total score of two or more across these four language items. A child was defined as having a language impairment
if both a parent and teacher both checked “somewhat true” on at least one of the four language items, or if either teacher or parent checked “certainly true” on at least one item. The results showed that the group with co-occurring ADHD and dyslexia was the most impaired across all the language measures. The groups with either dyslexia or ADHD alone could not be differentiated from one other on the language composite and pragmatic language variables. However, children in the group with dyslexia exhibited more expressive language problems than the ADHD group, while receptive language problems were more prominent in the ADHD group. The control group outperformed the other groups across all the different aspects of language. The results of the study support findings from clinical studies that have also found children with dyslexia and ADHD to have high rates of language difficulties. However, an important methodological limitation is that the study used no objective measures of performance, and the evaluation of the participants’ reading, language and behavioural status relied solely on parent and teacher reports.

The overlap of DCD with dyslexia and ADHD

Developmental Coordination Disorder is a neuromotor disorder that impairs a child's motor coordination, and significantly interferes with daily activities and/or academic achievement. Children typically have difficulty with gross and/or fine motor skills, with motor abilities that are slower, less accurate, and more variable than their peers (Zwicker, Missiuna, Harris, & Boyd, 2012). Depending on the selection criteria, prevalence estimates for DCD are reported between 1.8% (5th percentile cut-off) to 6% (15th percentile cut-off), with 2% found to be severely affected (Lingam, Hunt, Golding, Jongmans, & Emond, 2009). Over the last 25 years, evidence from several researchers (e.g., Kaplan et al., 1997; Kooistra, Crawford, Dewey, Cantell, & Kaplan, 2005; O'Hare & Khalid, 2002; Pitcher et al., 2003; Sugden, Chambers, & Snowling, 2005; Tervo, Azuma, Fogas, & Fiechtner, 2002) has shown an overlap of DCD with the other developmental disorders, however it has been found to
occur most commonly with ADHD (Dewey, Kaplan, Crawford, & Wilson, 2002; Kadesjo & Gillberg, 1999; Piek & Dyck, 2004). For example, an early study by Kaplan and colleagues (2001) examined the overlap of developmental disorders in a population and clinical sample of 170 children aged 8 to 16 years who were assessed for dyslexia, ADHD, and DCD. The Diagnostic Interview Schedule for Children (Costello, Edelbrock, & Costello, 1985) was used to classify children with ADHD. In addition, parents completed the Child Behaviour Checklist (CBCL: Achenbach, 1991) and the Abbreviated Symptom Questionnaire (Goyette, Conners, & Ulrich, 1978). Based on these measures, 107 children met diagnostic criteria for ADHD. Children were classified with dyslexia at the phonological level if they scored at or below the 24th percentile on the word Attack subtest of the Woodcock-Johnson Psychoeducational Battery-Revised (Woodcock, Johnson, & Mather, 1989), and scored at or below the 16th percentile on the Spelling subtest of the Wide Range Achievement Test-Revised (Jastak & Wilkinson, 1984) or the WJ-R Spelling subtest, and scored less than 17 on the Auditory Analysis Test (Rosner & Simon, 1971). Children were also assessed for higher level reading deficits and were classified with dyslexia if they scored at or below the 16th percentile on either the WJ-R Basic Reading or Reading Comprehension measures.

The children were also assessed on a battery of sensorimotor tests, and DCD was defined as performance below a designated cut-off on at least two of the following measures: the Bruininks-Oseretsky Test of Motor Proficiency (BOTMP; Bruininks & Bruininks, 2005), the Movement Assessment Battery for Children (M-ABC; Henderson, Sugden, & Barnett, 1992), and/or the Developmental Coordination Disorder Questionnaire (Wilson, Kaplan, Crawford, Campbell, & Dewey, 2000). No child had DCD alone, as by definition they would not have been included in the sample. The study found that the prevalence of dyslexia was 74.1%, ADHD 62.9%, and DCD 17.1%. When the children with dyslexia were considered, 50% had dyslexia alone, and 50% had co-occurring dyslexia and ADHD. When considering
ADHD, only 20% were found to have ADHD alone, while 59% met criteria for co-occurring dyslexia and ADHD. When DCD was considered in combination with dyslexia and/or ADHD, 31% of children had co-occurring ADHD and DCD, while the majority (69%) of children were found to have co-occurring ADHD, DCD and dyslexia. However, the study found no evidence of children with DCD without ADHD, indicating DCD did not occur with dyslexia alone (Kaplan et al., 2001). Similarly, a meta-analysis by Rochelle and Talcott (2006) found that the association between dyslexia and DCD is most strongly influenced by variables other than reading skills. The analyses included 17 studies that compared balance function between groups with dyslexia and controls, and effect sizes were obtained for each. There was wide variation in the size of the effects, and the results indicated that studies which pre-screened and excluded participants with ADHD produced the smallest effect sizes. The strongest predictor of balance effects across studies was the proportion of participants that had been screened for symptoms of ADHD.

The authors suggested the association between literacy impairments and motor deficits was unlikely to be unique, and may relate to the high co-occurrence of dyslexia with other developmental disorders such as ADHD, which is known to be highly prevalent with motor impairments (Kadesjö & Gillberg, 2001; Kaplan et al., 2001; Piek, Pitcher, & Hay, 1999), and indeed, some evidence suggests a genetic link between DCD and ADHD (Martin, Piek, & Hay, 2006). The review was unable to estimate the performance of the group with dyslexia relative to controls, as significant heterogeneity of effect-sizes revealed that the standardised mean differences between the dyslexia and control groups across the different studies were inconsistent, and unlikely to have been sampled from the same underlying population.

The occurrence of DCD in children with language impairments is also well documented (Hill, 2001), and the prevalence estimates for DCD in children with SLI are
reported to vary between 20% and 75% (Cheng, Chen, Chen, Tsai, & Cherng, 2009; Visscher, Houwen, Scherder, Moolenaar, & Hartman, 2007; Webster et al., 2006; Webster, Majnemer, Platt, & Shevell, 2005). There is also evidence for shared genetic liability between language impairment and DCD (Bishop, 2002), as well as between ADHD and DCD (Fliers, 2009; Martin et al., 2006). In addition, some children with ADHD and DCD have been noted as more likely to be diagnosed with the ADHD combined subtype, and more likely to have impaired language functioning, particularly oral expression (Tervo et al., 2002). Overall, the research evidence suggests a closer association between language disorders, ADHD and DCD in school-aged children (Gooch et al., 2014; Kadesjo & Gillberg, 1999; Kaplan et al., 2001; Rochelle & Talcott, 2006; Willcutt & Pennington, 2000), as the evidence for an association between DCD and dyslexia alone is not strong (Rochelle, Witton, & Talcott, 2009).

The above reviewed the substantial literature highlighting the high rates of co-occurring clinical and subclinical developmental disorders. The current evidence indicates that a substantial number of children diagnosed with dyslexia will also exhibit clinical, or at least sub-clinical symptoms of ADHD or AD (and vice-versa). In addition, research has noted the common co-occurrence between dyslexia and SLI (Bishop, 2008; Pennington & Bishop, 2009), as well as ADHD and SLI (McGrath et al., 2008; Mueller & Tomblin, 2012). However, it has been suggested that the relationship between ADHD and SLI may be inflated due to an artefact of behaviour rating scales (Redmond, 2002). It has also been noted that most studies investigating ADHD + SLI are based on clinical samples and represent the most severe cases (Sexton et al., 2012). These findings may indicate the association between dyslexia and SLI may be stronger than the association between ADHD and SLI, suggesting children with dyslexia may present with more severe language difficulties than those with AD or ADHD. In contrast, DCD has been found to be more strongly related to ADHD and is
not thought to be uniquely associated with dyslexia (Kaplan et al., 2001; Rochelle & Talcott, 2006), suggesting the motor skills of children with ADHD may be poorer than those with dyslexia. There has been little investigation of the prevalence of AD alone and other developmental disorders. Consequently, one aim of the current research was to investigate these potential associations, and to examine the severity of difficulties among the disorders.

While the common co-occurrence between the developmental disorders has been established, a broad assessment of ability domains is not yet standard practice, and it is likely that many children have undiagnosed clinical and subclinical difficulties that negatively impact their academic performance. The issue also poses a methodological limitation in research practices, as children who are not assessed across a broad range of abilities may not provide a pure representation of their allocated group (i.e., ADHD), as they may also have clinical or subclinical reading, language or motor impairments. The current literature supports the use of continuous groupings in developmental research, and this study is novel in that it is the first to investigate different ways that a sample of children can be classified as having a condition(s) or not using clinical and subclinical criteria.

3.4 Aims and Hypotheses

Previous research has found that most children with one developmental disorder will meet clinical or subclinical criteria for at least one other developmental disorder. Thus, the current study investigated the prevalence of single and multiple developmental disorders in this sample of school-aged children to establish whether the patterns of disorder are consistent with those reported in previous research. The children were recruited based on a previous diagnosis of dyslexia and/or ADHD, or whose parents thought they may have had symptoms of dyslexia or ADHD. Of the 72 children in the sample, 46% had a previous clinical diagnosis. Of these, 39% had a diagnosis of dyslexia, 6% had a diagnosis of co-occurring dyslexia + ADHD, and 1 child had a previous diagnosis of ADHD alone. In
addition, 65% of the sample was receiving remediation for literacy and/or numeracy either at school and/or privately. Based on research highlighting the substantial overlap between dyslexia and ADHD or AD, it was hypothesised that a high proportion of participants in the sample would show evidence of more than one disorder, particularly dyslexia + AD, or dyslexia + ADHD, at a clinical or sub-clinical level.

Based on the reported associations that have been found in the research literature between the developmental disorders, the following hypotheses were made regarding the expected severity of difficulties: a) the group with dyslexia + AD or dyslexia + ADHD would show greater evidence of reading, attention, language, and motor deficits than the groups with a single disorder b) the group with dyslexia alone was expected to be more impaired on the language measures than the groups with AD or ADHD alone, who were expected to perform more poorly than the control group. It was also predicted that the groups with AD or ADHD would perform more poorly on the measures of motor skill than the group with dyslexia alone, who were not expected to differ from the control group in motor performance.

3.5 Method

Participants

Participants were a community sample who were recruited as part of a larger investigation that has been described in detail in Chapter 2. This sample consisted of 72 children aged between 9 and 11 years (57 boys and 15 girls) who were attending local primary schools (Grade 3 to 6). Caregivers, schools, and teachers provided written consent, and child participants gave written assent prior to the commencement of testing.

3.6 Group Classification

Dyslexia Classification

Children were classified with dyslexia using a mean composite score from the Sight Word Efficiency (SWE) and Phonemic Decoding Efficiency (PDE) subtests (Form A) from
the TOWRE-2 (Torgesen et al., 2012). Participants in the current study who scored at or below the 15th percentile on the average score on both the SWE and PDE were classified with dyslexia. This method has been used by some studies that used alternative measures of word reading and phonological processing to determine the presence of dyslexia (e.g., Gooch et al., 2011; Wright & Conlon, 2009; Wright, Conlon, & Dyck, 2012). This is consistent with the method used by Pennington et al. (2012).

**AD and ADHD classification**

Children were classified as having AD and ADHD using the Conners-3 (Conners, 2008) parent and teacher reports. T-scores from the parent and teacher measures were combined to produce a mean T-score adjusted for age and gender, with higher T-scores indicating more symptoms of inattention or hyperactivity. These criteria have been adopted in recent studies (e.g., (Arnett, Pennington, Willcutt, DeFries, & Olson, 2015), as averaging scores from multiple raters has been found to be the optimal way to increase the validity of ADHD diagnosis (Martel, Schimmack, Nikolas, & Nigg, 2015). There were 10 participants with only one completed measure, and in these cases, classification was based on T-scores from either a Parent or Teacher measure. Children with a mean T-score at or above 65 on the attention component only were included in the group with AD. Children with a mean T-score at or above 65 on both the attention and hyperactivity components were included in the group with ADHD. These criteria have been adopted in several studies (see Gooch et al., 2011; Willcutt et al., 2001). The clinical cut-off was a T-score ≥ 65; subclinical 60 – 64; and average range ≤ 59. These guidelines have been determined by Conners (2008).

**SLI classification**

Four core subtests of the Clinical Evaluation of Language Fundamentals-Fourth Edition (CELF-IV; Semel, Wiig, & Secord, 2003) were used to measure core areas of Receptive and Expressive language ability, and as a standardised measure of general
language skill. Receptive language was assessed with Concepts and Following Directions which measures the ability to follow oral directions of increased procedural and linguistic complexity; and Word Classes 2 which assesses knowledge of semantic relationships between words. Expressive language was assessed with Recalling Sentences which measures the ability to repeat sentences increasing in syntactic complexity; and Formulated Sentences, that requires the use of certain words and phrases in full sentences. The CELF-IV was included as it has Australian norms for 5- to 21-year-olds. It has good test-retest reliability coefficients ($r = 0.88 – 0.92$), and good validity, as evidenced by high correlations with other independent language measures (correlations of 0.80 - 0.87: Conti-Ramsden, Durkin, & Walker, 2010). Scoring is based on the correctness of syntactic structures and the meaningful use of the words/phrases presented. The Core language score was used to determine the presence of SLI and is derived from the four core subtests. It is considered the most representative measure of a child’s language skills and provides a reliable way to quantify a student’s overall language performance. The clinical cut-off was $\leq 85$; subclinical $86 - 89$; and average $\geq 90$. These cut-offs are based on the authors’ guidelines.

**DCD Classification**

The Movement Assessment Battery for Children 2 (M ABC-2: (Henderson, Sugden, & Barnett, 2007) was used to provide quantitative and qualitative data about a child’s performance of age-appropriate tasks within 3 subsections: Manual Dexterity, Ball Skills, and Static and Dynamic Balance. The measure is designed to screen for motor impairment rather than provide a profile of a child’s motor performance. Quantitative performance of each item (e.g., time of completion) is scored from 0 (best) to 5 (worst) and qualitative aspects of performance (e.g., body posture) are recorded using standard cues. The M-ABC is the most commonly reported norm-ranked assessment used to determine the presence of DCD in
school-aged children and has been evaluated and found useful for identifying children with DCD in Australia (Mon-Williams, Pascal, & Wann, 1994). Test-retest reliability is good ($r = .92$; Croce, Horvat, & McCarthy, 2001) and moderate concurrent validity ($r = 0.50, p < 0.01$) has been demonstrated with the Bruininks-Oseretsky Test of Motor Proficiency (Bruininks & Bruininks, 2005). Children in the current study fell into one of two age assessment bands: 7 to 10 years, or 11 to 16 years. The performance index was the scaled scores for each motor domain which are compared to normative tables to determine whether subsection performance is typical, suspect, or definitely impaired. Based on the authors guidelines the severity range was classified as: Clinical $\leq 5$; Subclinical (at risk) 6; and Average $7 - 19$.

3.7 Other measures

**Executive Dysfunction**

Symptoms of executive dysfunction were identified using the Conners-3 (Conners, 2008) parent and teacher reports. T-scores from the parent and teacher measures were combined to produce a mean T-score adjusted for age and gender, with higher T-scores indicating more symptoms of executive dysfunction. The clinical cut-off was a T-score $\geq 65$; subclinical $60 - 64$; and average range $\leq 59$. These guidelines have been determined by Conners (2008).

**General Cognitive Ability**

The Picture Concepts and Matrix Reasoning subtests from the Wechsler Intelligence Scale for Children (WISC-IV; Weschler, 2003) were used as measures of non-verbal intellectual ability and to estimate general cognitive ability because verbal IQ scores are confounded with reading scores (Sternberg & Grigorenko, 2002). The Picture Concepts subtest requires the child to select one picture from each of two to three rows of pictures to form a group with common characteristics (maximum score = 28; $\alpha = .86$ for internal
consistency; $r = .76$ for test–retest reliability). Matrix Reasoning requires the child to identify the missing portion of an incomplete visual matrix (maximum score = 35; $\alpha = .89$ for internal consistency; $r = .85$ for test–retest reliability). The scaled scores are combined to form a nonverbal fluid reasoning index (Flanagan & Kaufman, 2009). The measure is reported to have excellent internal consistency, test–retest reliability, criterion validity and construct validity (Sattler, 2001). In a review of 16 nonverbal IQ measures (DeThorne & Schaefer, 2004), the WISC-IV was one of four tests reported to be particularly strong. It was one of only three tests to report test-retest coefficients of 0.83 to 0.93. The clinical cut-off was a standard score $\leq 85$, which is based on the author’s guidelines.

3.8 Procedure

The procedure is described in detail in Chapter 2. With regards to the current study, the order of administration of the relevant measures was as follows: the WISC-IV subtests (matrix reasoning and picture concepts), the CELF-IV subtests (Concepts and Following Directions, Recalling Sentences, Formulated Sentences, and Word Classes 2) the TOWRE-2 (Sight Word Efficiency and Phonemic Decoding), and finally the M ABC-2 (manual dexterity, aiming and catching, and balance). Parents and teachers each completed the Conners-3 report measures for the child participants.

Analysis strategy

The prevalence rates for the single and multiple developmental disorders among the groups on the reading, attention, language and motor measures were obtained by using clinical and subclinical symptoms of each disorder. To determine whether the severity of each disorder was influenced by the number of disorders experienced by each child, a series of one-way ANOVAs and post-hoc analyses were conducted. To account for Type 1 error, in the multiple testing, a $p$ value of .01 was adopted. For the reading, language, non-verbal
ability and motor measures, higher scores indicated better performance. For the measures of AD, ADHD and executive function, higher scores indicated greater symptomatology.

3.9 Results

**Overall Prevalence Rates**

Each of the 72 participants was classified using the clinical and sub-clinical criteria for dyslexia, AD, ADHD, SLI and DCD. Based on the *clinical* cut-off scores for the presence of each disorder, there were 36 children with dyslexia (50.0%), and a total of 46 children with AD (64.0%). Of the 46 children with AD, 23 had no hyperactivity (AD only: 32%) and 23 had clinical symptoms of attention and hyperactivity (ADHD: 32%). There were 5 children with SLI (7.0%), no child with a clinical diagnosis of DCD, and 13 children in the sample with no evidence of any disorder (18.0%). When *subclinical* diagnoses were included, there were 48 children who met criteria for dyslexia (66.7%), and 55 who met criteria for AD (76.4%). Of the 55 children with AD, 19 had no hyperactivity (AD only, 26.4%) and 36 had symptoms of attention and hyperactivity (ADHD, 50.0%). There were 5 children who met criteria for SLI (6.9%), 2 children who met criteria for DCD (2.8%), and 6 children with no clinical or subclinical evidence of any disorder (8.3%).

**Presence of single and multiple disorders**

Of the 59 children who met diagnostic criteria for one or more disorders, there were 30 children who met the *clinical* criteria for a single disorder (41.7%), 25 who met criteria for two disorders (34.7%), and 4 children who met criteria for three disorders (5.5%). When *subclinical* symptoms were included, there were 20 children with a single disorder (27.8%), 39 with two disorders (54.2%), and 8 children identified with three disorders (11.1%).

3.10 Disorders that overlap with dyslexia
Table 3.1 shows the proportion of children with dyslexia (clinical and subclinical symptoms) and the presence or absence of AD, SLI, and DCD. Consistent with the hypothesis that there would be greater evidence of multiple disorders, a small proportion of the sample showed evidence of dyslexia only, particularly when sub-clinical features of AD, and SLI were considered. Regardless of the classification criteria used, more than two-thirds of the sample showed evidence of two disorders, predominantly dyslexia and AD. The proportion of children showing evidence of SLI (either at clinical or subclinical levels) as well as dyslexia was relatively low. DCD was not associated with a diagnosis of dyslexia, and this is consistent with the literature which indicates DCD is more closely associated with ADHD than dyslexia. Consistent with the hypothesis, dyslexia was more likely to occur in combination with at least one other disorder than to appear alone.

Table 3.1

Prevalence of single and multiple developmental disorders

<table>
<thead>
<tr>
<th></th>
<th>Clinical diagnosis only</th>
<th>Clinical Dyslexia, Clinical and subclinical AD and LI</th>
<th>Clinical/Subclinical Dyslexia and SLI, DCD (90 or less) and AD (60 or greater)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total with dyslexia</strong></td>
<td>36/72 (50%)</td>
<td>36/72 (50%)</td>
<td>48/72 (66.7%)</td>
</tr>
<tr>
<td><strong>Single Disorder</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dyslexia only</td>
<td>9/36 (25.0%)</td>
<td>5/36 (13.9%)</td>
<td>6/48 (12.5%)</td>
</tr>
<tr>
<td><strong>Two disorders</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dyslexia + AD</td>
<td>22/36 (61.1%)</td>
<td>25/36 (69.4%)</td>
<td>34/48 (70.8%)</td>
</tr>
<tr>
<td>Dyslexia + SLI</td>
<td>3/36 (8.3%)</td>
<td>2/36 (5.5%)</td>
<td>3/48 (6.3%)</td>
</tr>
<tr>
<td>Percentage with 2 disorders</td>
<td>69.4%</td>
<td>74.9%</td>
<td>77.1%</td>
</tr>
<tr>
<td><strong>Three disorders</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dyslexia/SLI/AD</td>
<td>2/36 (5.5%)</td>
<td>4/36 (11.1%)</td>
<td>5/48 (10.4%)</td>
</tr>
<tr>
<td>Percentage 2 or more disorders</td>
<td>74.9%</td>
<td>86%</td>
<td>87.5%</td>
</tr>
</tbody>
</table>

*Children who met clinical (n = 36) and subclinical (n = 48) criteria for dyslexia.*
3.11 Disorders that overlap with ADHD or AD

Table 3.2 shows the proportion of children with AD (clinical and subclinical symptoms) and the presence or absence of dyslexia, SLI and DCD. The analyses included all children with a diagnosis of inattention (including 23 who meet criteria for ADHD). When clinical criteria only were considered, there was a similar proportion of the sample with AD only and AD + dyslexia. When subclinical symptoms of dyslexia were included in the classification process, the proportion of children with AD + dyslexia increased to 63%, indicating that many children with clinical symptoms of AD also have poor reading skills.

The percentage of children with AD and at least one other disorder, was similar to the number of children found to have AD alone. This might have occurred because the full diagnostic classification of ADHD was not used. DCD and SLI co-occurred infrequently in the sample, and some explanations are proposed in the discussion.

Table 3.2

<table>
<thead>
<tr>
<th></th>
<th>Clinical diagnosis only</th>
<th>Clinical AD, Clinical and Subclinical Dyslexia, SLI and DCD</th>
<th>Clinical/Subclinical AD, Dyslexia, SLI and DCD.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total with AD</td>
<td>46/72 (65.3%)</td>
<td>46/72 (65.3%)</td>
<td>55/72 (76.4%)</td>
</tr>
<tr>
<td>Single disorder</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AD only</td>
<td>21/46 (45.7%)</td>
<td>12/46 (26.1%)</td>
<td>14/57 (25.5%)</td>
</tr>
<tr>
<td>Two disorders</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AD + Dyslexia</td>
<td>21/46 (45.7%)</td>
<td>29/46 (63.0%)</td>
<td>33/57 (60.0%)</td>
</tr>
<tr>
<td>AD + DCD</td>
<td>-</td>
<td>2/46 (4.2%)</td>
<td>2/57 (3.6%)</td>
</tr>
<tr>
<td>AD + SLI</td>
<td>2/46 (4.3%)</td>
<td>1/46 (2.1%)</td>
<td>2/57 (3.6%)</td>
</tr>
<tr>
<td>Percentage with 2</td>
<td>50%</td>
<td>69.3%</td>
<td>64.8%</td>
</tr>
<tr>
<td>disorders</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Three disorders

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Clinical (n=23)</th>
<th>Clinical and subclinical (n=36)</th>
</tr>
</thead>
<tbody>
<tr>
<td>AD/SLI/Dyslexia</td>
<td>2/46 (4.3%)</td>
<td>2/46 (4.2%)</td>
</tr>
<tr>
<td>Percentage 2 or more</td>
<td>53.3%</td>
<td>73.5%</td>
</tr>
<tr>
<td>disorders</td>
<td></td>
<td>71.9%</td>
</tr>
</tbody>
</table>

Note. Clinical (n = 46) and subclinical criteria (n = 57) for AD.

Given the high proportion of children in the sample with AD, prevalence rates were obtained for children meeting the criteria for ADHD and one or more additional developmental disorders (see Table 3.3).

Table 3.3

Prevalence of Single and Multiple Developmental Disorders in Children with ADHD

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Clinical diagnosis only</th>
<th>Clinical ADHD, Clinical SLI and DCD</th>
<th>Sub-clinical ADHD, reading, SLI and DCD.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total with ADHD</td>
<td>23/72 (31.9%)</td>
<td>23/72 (31.9%)</td>
<td>36/72 (50.0%)</td>
</tr>
<tr>
<td>Single disorder</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ADHD only</td>
<td>13/23 (56.5%)</td>
<td>7/23 (30.4%)</td>
<td>12/36 (33.3%)</td>
</tr>
<tr>
<td>Two disorders</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ADHD + Dyslexia</td>
<td>9/23 (39.1%)</td>
<td>13/23 (56.5%)</td>
<td>20/36 (55.6%)</td>
</tr>
<tr>
<td>ADHD + DCD</td>
<td>2/23 (8.6%)</td>
<td>2/36 (5.6%)</td>
<td></td>
</tr>
<tr>
<td>ADHD + SLI</td>
<td></td>
<td>1/36 (2.8%)</td>
<td></td>
</tr>
<tr>
<td>Percentage with 2 disorders</td>
<td></td>
<td>39.1%</td>
<td>65.1%</td>
</tr>
<tr>
<td>Three disorders</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ADHD/SLI/Dyslexia</td>
<td>1/23 (4.3%)</td>
<td>1/23 (4.3%)</td>
<td>1/36 (2.8%)</td>
</tr>
<tr>
<td>Percentage 2 or more</td>
<td>43.4%</td>
<td>69.4%</td>
<td>66.8%</td>
</tr>
<tr>
<td>disorders</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Clinical (n = 23) and subclinical criteria (n = 36)

Of the 46 children in the sample who met diagnostic criteria for AD, 23 (31.9% of the total sample) also met the criteria for ADHD combined type. Of these 9 (39.1%) also had dyslexia, with one child diagnosed with ADHD, dyslexia and SLI. When clinical and subclinical symptoms of dyslexia, SLI and DCD were included, the percentage with ADHD
only was reduced by almost half, with 56.5% showing evidence of dyslexia and ADHD. Two children had subclinical symptoms of DCD, with one child only being diagnosed with three disorders. When subclinical symptoms were considered, 50% of the sample had ADHD, and two-thirds of these experienced at least one other disorder, most frequently dyslexia. Proportionally, a higher percentage of the ADHD group than the AD group were found to have a single disorder, with a slightly higher percentage of individuals in the AD group observed to have more than one developmental disorder. However, the proportion of children with disorders additional to ADHD or AD remained lower than the proportion found for the group with Dyslexia.

3.12 Do children with single or multiple deficits differ in diagnostic severity?

The mean scores on the classification and other validation measures were compared to determine if children with more than one disorder had more severe difficulties than those with a single disorder. Groups were formed in three ways. First, based on the diagnosis of dyslexia or AD, children meeting only the clinical criteria for the different disorders were used and contrasted with a control group (see Table 3.4). Due to the small group numbers, children with a language disorder, and either dyslexia, AD or both were included in a single group. Second, children who met clinical and subclinical criteria for each of dyslexia and AD were included (Table 3.5). The third set of analyses used children clinically diagnosed with ADHD (Table 3.6). The same groups analysis was conducted, with a supplementary analysis included to determine if there were significant differences on the reading measures for children with AD or ADHD (Table 3.7).

3.13 Dyslexia

On the reading measures, the groups (single or multiple disorders) with a clinical or sub-clinical classification of dyslexia did not differ significantly on reading fluency (see
This indicates that the presence of more than one disorder had no influence on the severity of reading scores and failed to support the hypothesis that the multiple disorder groups would perform more poorly. The groups with dyslexia had significantly poorer reading fluency than either the control, AD or ADHD only groups, who showed no evidence of reading difficulties. When ADHD was used in the classification process (Table 3.6) the group with dyslexia alone had similar symptoms of attentional difficulties as the ADHD and dyslexia + ADHD groups, supporting the hypothesis that greater attentional difficulties were also found in children with dyslexia. The groups with dyslexia (single and multiple deficit) did not differ from the control group on measures of language, motor ability or non-verbal ability.

### 3.14 Attention Deficit

On the measures of AD, the AD groups (single or multiple deficit) did not differ in severity on either the attention or executive function measures, indicating that the presence of more than one disorder did not influence the severity of the reported difficulties (see Table 3.5). The groups with AD had significantly poorer performance on the attention and executive function measures than either the group with dyslexia alone or the control group, who did not differ. The AD group did not differ from the control group on the measures of language or motor ability.

### 3.15 Attention Deficit Hyperactivity Disorder

The ADHD groups (single or multiple deficit) did not differ in severity on the attention measure, with the control group showing significantly better performance on the attention measure than the other groups (see Table 3.6). The groups with ADHD, and ADHD + dyslexia, did not differ significantly on hyperactivity symptoms, and both groups had significantly more hyperactivity symptoms than all the other groups who did not differ significantly. On the measure of executive functioning the group with dyslexia + ADHD
scored significantly higher than the control group, indicating more executive function
difficulties for this group. There were no other group differences on this measure. There
were no significant differences in performance between the groups on the measures of motor
ability.
Table 3.4

**AD, Dyslexia, LI: Mean classification and validation scores using clinical criteria: severity of single deficit versus severity of multiple deficits**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control (n = 13)</th>
<th>AD (n = 21)</th>
<th>AD + Dyslexia (n = 22)</th>
<th>Dyslexia (n = 9)</th>
<th>SLI, and AD/Dyslexia (n = 6)!!</th>
<th>ANOVA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>( M )</td>
<td>95% CI’s</td>
<td>( M )</td>
<td>95% CI’s</td>
<td>( M )</td>
<td>95% CI’s</td>
</tr>
<tr>
<td><strong>Reading Fluency</strong></td>
<td>96.1</td>
<td>90.6 - 101.5</td>
<td>97.2</td>
<td>92.6 - 101.8</td>
<td>80.2</td>
<td>78.6 - 81.9</td>
</tr>
<tr>
<td>( F(4, 66) = 24.20, p &lt; .001 )</td>
<td>The three groups with dyslexia did not differ significantly on reading fluency. These groups had significantly poorer reading skills than the control or AD group, who were also equivalent.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>AD measures</strong></td>
<td>Mean AD (P/T)!</td>
<td>55.1</td>
<td>50.6 - 59.1</td>
<td>74.3</td>
<td>71.3 - 77.3</td>
<td>75.7</td>
</tr>
<tr>
<td></td>
<td>Mean EF (P/T)</td>
<td>56.4</td>
<td>50.6 - 62.3</td>
<td>66.8</td>
<td>63.3 - 70.3</td>
<td>72.2</td>
</tr>
<tr>
<td></td>
<td>For both AD measures, the groups with AD had significantly poorer performance than other groups. The groups with AD and AD + Dyslexia did not differ significantly.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Language Measures</strong></td>
<td>Core Language!</td>
<td>110.2</td>
<td>104.4 -116.1</td>
<td>105.2</td>
<td>100.5 - 109.9</td>
<td>102.8</td>
</tr>
<tr>
<td></td>
<td>Receptive</td>
<td>103.5</td>
<td>98.7 -108.2</td>
<td>98.5</td>
<td>94.3 - 102.6</td>
<td>96.5</td>
</tr>
<tr>
<td></td>
<td>Expressive</td>
<td>113.0</td>
<td>107.4 -118.6</td>
<td>109.2</td>
<td>103.9 - 114.5</td>
<td>107.2</td>
</tr>
<tr>
<td></td>
<td>For each of the language measures the group containing those with a language impairment scored significantly lower than all other groups who were not significantly different. There were no other group differences.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Non-Verbal Ability</strong></td>
<td>PRI</td>
<td>105.4</td>
<td>98.4 -112.4</td>
<td>101.2</td>
<td>97.4 - 104.9</td>
<td>101.4</td>
</tr>
<tr>
<td></td>
<td>Motor ability!</td>
<td>9.64</td>
<td>8.8 - 10.4</td>
<td>10.2</td>
<td>9.4 - 11.2</td>
<td>10.4</td>
</tr>
</tbody>
</table>

Note. ! indicates measured used in classification criteria. !! indicates all language impaired children with at least one other disorder.
Table 3.5

AD, Dyslexia, LI: Mean classification and validation scores using clinical and subclinical criteria: severity of single deficit versus severity of multiple deficits

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control (n=6)</th>
<th>AD (n=17)</th>
<th>AD + Dyslexia (n=34)</th>
<th>Dyslexia (n=6)</th>
<th>SLI + AD/dys (n=8)!!</th>
<th>ANOVA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M 95% CI's</td>
<td>M 95% CI's</td>
<td>M 95% CI's</td>
<td>M 95% CI's</td>
<td>M 95% CI's</td>
<td>F (4, 66) = 30.65, p &lt; .001</td>
</tr>
<tr>
<td>Reading Fluency!</td>
<td>99.0 86.9 - 111.1</td>
<td>101.5 97.1 - 105.9</td>
<td>82.35 80.66 - 84.0</td>
<td>82.8 76.9 - 88.7</td>
<td>76.2* 68.7 - 83.7</td>
<td></td>
</tr>
<tr>
<td>The three groups with dyslexia did not differ significantly on reading fluency. These groups had significantly poorer reading skills than the control or AD group.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AD Measures</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>F (4, 66) = 23.1, p &lt; .001</td>
</tr>
<tr>
<td>Mean AD (P/T)!</td>
<td>50.4 45.1 - 55.7</td>
<td>71.2 67.0 - 75.4</td>
<td>73.4 71.0 - 75.6</td>
<td>51.3 45.5 - 57.1</td>
<td>64.4 56.8 - 71.9!</td>
<td></td>
</tr>
<tr>
<td>Mean EF (P/T)</td>
<td>54.3 49.4 - 61.7</td>
<td>65.6 62.1 - 69.0</td>
<td>69.1 66.3 - 71.9</td>
<td>51.7 45.2 - 58.1</td>
<td>63.3 51.9 - 74.7</td>
<td></td>
</tr>
<tr>
<td>For both AD measures the groups with AD had significantly poorer performance than other groups. The group with AD and AD + dyslexia did not differ significantly. The control group and the group with dyslexia were not different.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Language Measures</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>F (4, 66) = 10.25 p &lt; .001</td>
</tr>
<tr>
<td>Core Language!</td>
<td>110.8 105.2 - 116.4</td>
<td>107.8 102.1 - 113.5</td>
<td>102.5 99.9 - 105.1</td>
<td>104.6 92.0 - 117.3</td>
<td>85.7 78.3 - 93.2</td>
<td></td>
</tr>
<tr>
<td>Receptive</td>
<td>102.5 96.4 - 108.6</td>
<td>100.0 95.4 - 104.6</td>
<td>97.2 94.5 - 99.9</td>
<td>99.5 85.5 - 113.5</td>
<td>77.9 73.2 - 82.5</td>
<td></td>
</tr>
<tr>
<td>Expressive</td>
<td>113.7 107.2 - 120.1</td>
<td>112.1 105.9 - 118.3</td>
<td>106.4 103.9 - 108.9</td>
<td>107.3 95.1 - 119.5</td>
<td>96.3 83.1 - 100.3</td>
<td></td>
</tr>
<tr>
<td>For each of the language measures the group containing those with a language impairment scored significantly lower than all other groups who were not significantly different. There were no other group differences.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-Verbal Ability</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>F (4, 66) = 2.65, p =.041</td>
</tr>
<tr>
<td>PRI</td>
<td>97.3 87.7 - 107.0</td>
<td>104.3 99.2 - 109.5</td>
<td>102.2 98.6 - 105.7</td>
<td>101.8 92.7 - 110.9</td>
<td>90.2 76.4 - 104.1</td>
<td></td>
</tr>
<tr>
<td>There were no significant group differences</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Motor ability!</td>
<td>9.64 8.8 - 10.4</td>
<td>10.2 9.4 - 11.2</td>
<td>10.4 9.8 - 11.0</td>
<td>10.2 9.0 - 11.5</td>
<td>9.2 7.9 - 10.4</td>
<td>F (4, 66) = 1.09, p = .370</td>
</tr>
</tbody>
</table>

Note. ! indicates measures used in classification criteria. !! indicates all language impaired children with at least one other disorder
Table 3.6

**ADHD, Dyslexia, LI: Mean classification and validation scores using clinical criteria: severity of single deficit versus multiple deficits**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control (n = 19)</th>
<th>ADHD (n = 13)</th>
<th>ADHD + Dyslexia (n = 9)</th>
<th>Dyslexia (n = 22)</th>
<th>SLI, ADHD and/Dyslexia (n = 6)!!</th>
<th>ANOVA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>95% CI's</td>
<td>M</td>
<td>95% CI's</td>
<td>M</td>
<td>95% CI's</td>
</tr>
<tr>
<td>Reading Fluency!</td>
<td>94.0</td>
<td>90.0 - 97.9</td>
<td>100.3</td>
<td>94.6 - 106.0</td>
<td>80.4</td>
<td>77.0 - 83.8</td>
</tr>
<tr>
<td></td>
<td>10.0</td>
<td>9.4 - 10.8</td>
<td>10.6</td>
<td>9.7 - 11.1</td>
<td>10.2</td>
<td>9.5 - 10.9</td>
</tr>
<tr>
<td></td>
<td>10.1</td>
<td>9.4 - 10.8</td>
<td>10.6</td>
<td>9.7 - 11.1</td>
<td>10.2</td>
<td>9.5 - 10.9</td>
</tr>
<tr>
<td></td>
<td>9.9</td>
<td>8.7 - 11.1</td>
<td>10.6</td>
<td>9.7 - 11.1</td>
<td>10.2</td>
<td>9.5 - 10.9</td>
</tr>
<tr>
<td></td>
<td>10.1</td>
<td>9.4 - 10.8</td>
<td>10.6</td>
<td>9.7 - 11.1</td>
<td>10.2</td>
<td>9.5 - 10.9</td>
</tr>
</tbody>
</table>

*ANOVA M* 95% CI's  

The groups with dyslexia did not differ significantly on reading fluency. These groups had significantly poorer reading skills than the control or ADHD group.

**ADHD Measures**

Mean Hyp (P/T)  

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control (n = 19)</th>
<th>ADHD (n = 13)</th>
<th>ADHD + Dyslexia (n = 9)</th>
<th>Dyslexia (n = 22)</th>
<th>SLI, ADHD and/Dyslexia (n = 6)!!</th>
<th>ANOVA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>95% CI's</td>
<td>M</td>
<td>95% CI's</td>
<td>M</td>
<td>95% CI's</td>
</tr>
<tr>
<td></td>
<td>54.6</td>
<td>50.6 - 58.6</td>
<td>74.4</td>
<td>69.6 - 79.2</td>
<td>72.8</td>
<td>67.3 - 78.2</td>
</tr>
<tr>
<td></td>
<td>60.2</td>
<td>55.3 - 65.1</td>
<td>75.5</td>
<td>71.9 - 79.2</td>
<td>76.4</td>
<td>73.6 - 79.3</td>
</tr>
<tr>
<td></td>
<td>60.0</td>
<td>55.1 - 65.0</td>
<td>66.6</td>
<td>62.2 - 71.1</td>
<td>73.7</td>
<td>70.0 - 77.4</td>
</tr>
</tbody>
</table>

The two groups with ADHD did not differ significantly on hyperactivity symptoms. Both groups reported significantly greater symptoms than all other groups who did not differ.

Mean AD (P/T)  

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control (n = 19)</th>
<th>ADHD (n = 13)</th>
<th>ADHD + Dyslexia (n = 9)</th>
<th>Dyslexia (n = 22)</th>
<th>SLI, ADHD and/Dyslexia (n = 6)!!</th>
<th>ANOVA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>95% CI's</td>
<td>M</td>
<td>95% CI's</td>
<td>M</td>
<td>95% CI's</td>
</tr>
<tr>
<td></td>
<td>60.2</td>
<td>55.3 - 65.1</td>
<td>75.5</td>
<td>71.9 - 79.2</td>
<td>76.4</td>
<td>73.6 - 79.3</td>
</tr>
<tr>
<td></td>
<td>60.0</td>
<td>55.1 - 65.0</td>
<td>66.6</td>
<td>62.2 - 71.1</td>
<td>73.7</td>
<td>70.0 - 77.4</td>
</tr>
</tbody>
</table>

The mean AD score for the two ADHD groups did not differ significantly. The group with dyslexia did not differ from either of these groups. The control group had significantly better performance than these groups. On executive functioning, the control group scored significantly lower than the group with ADHD + dyslexia. There were no other group differences.

**Language Measures**

Core Language!  

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control (n = 19)</th>
<th>ADHD (n = 13)</th>
<th>ADHD + Dyslexia (n = 9)</th>
<th>Dyslexia (n = 22)</th>
<th>SLI, ADHD and/Dyslexia (n = 6)!!</th>
<th>ANOVA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>95% CI's</td>
<td>M</td>
<td>95% CI's</td>
<td>M</td>
<td>95% CI's</td>
</tr>
<tr>
<td></td>
<td>107.9</td>
<td>103.5 - 112.3</td>
<td>106.1</td>
<td>99.6 - 112.6</td>
<td>101.9</td>
<td>98.6 - 105.2</td>
</tr>
<tr>
<td></td>
<td>101.7</td>
<td>97.9 - 105.5</td>
<td>98.7</td>
<td>93.0 - 104.3</td>
<td>95.3</td>
<td>90.8 - 99.8</td>
</tr>
<tr>
<td></td>
<td>111.0</td>
<td>106.9 - 115.1</td>
<td>110.2</td>
<td>102.7 - 109.3</td>
<td>106.9</td>
<td>102.9 - 100.1</td>
</tr>
</tbody>
</table>

For each of the language measures the group containing those with a language impairment scored significantly lower than all other groups who were not significantly different.

Receptive  

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control (n = 19)</th>
<th>ADHD (n = 13)</th>
<th>ADHD + Dyslexia (n = 9)</th>
<th>Dyslexia (n = 22)</th>
<th>SLI, ADHD and/Dyslexia (n = 6)!!</th>
<th>ANOVA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>95% CI's</td>
<td>M</td>
<td>95% CI's</td>
<td>M</td>
<td>95% CI's</td>
</tr>
<tr>
<td></td>
<td>107.9</td>
<td>103.5 - 112.3</td>
<td>106.1</td>
<td>99.6 - 112.6</td>
<td>101.9</td>
<td>98.6 - 105.2</td>
</tr>
<tr>
<td></td>
<td>101.7</td>
<td>97.9 - 105.5</td>
<td>98.7</td>
<td>93.0 - 104.3</td>
<td>95.3</td>
<td>90.8 - 99.8</td>
</tr>
<tr>
<td></td>
<td>111.0</td>
<td>106.9 - 115.1</td>
<td>110.2</td>
<td>102.7 - 109.3</td>
<td>106.9</td>
<td>102.9 - 100.1</td>
</tr>
</tbody>
</table>

Non-Verbal Ability

PRI  

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control (n = 19)</th>
<th>ADHD (n = 13)</th>
<th>ADHD + Dyslexia (n = 9)</th>
<th>Dyslexia (n = 22)</th>
<th>SLI, ADHD and/Dyslexia (n = 6)!!</th>
<th>ANOVA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>95% CI's</td>
<td>M</td>
<td>95% CI's</td>
<td>M</td>
<td>95% CI's</td>
</tr>
<tr>
<td></td>
<td>104.7</td>
<td>99.9 - 109.5</td>
<td>100.3</td>
<td>95.2 - 105.4</td>
<td>102.8</td>
<td>94.7 - 110.8</td>
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<tr>
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<td>97.9 - 105.5</td>
<td>98.7</td>
<td>93.0 - 104.3</td>
<td>95.3</td>
<td>90.8 - 99.8</td>
</tr>
<tr>
<td></td>
<td>111.0</td>
<td>106.9 - 115.1</td>
<td>110.2</td>
<td>102.7 - 109.3</td>
<td>106.9</td>
<td>102.9 - 100.1</td>
</tr>
</tbody>
</table>

The group with a language disorder performed more poorly than the control group. There were no other group differences.

Motor ability!  

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control (n = 19)</th>
<th>ADHD (n = 13)</th>
<th>ADHD + Dyslexia (n = 9)</th>
<th>Dyslexia (n = 22)</th>
<th>SLI, ADHD and/Dyslexia (n = 6)!!</th>
<th>ANOVA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>95% CI's</td>
<td>M</td>
<td>95% CI's</td>
<td>M</td>
<td>95% CI's</td>
</tr>
<tr>
<td></td>
<td>10.1</td>
<td>9.4 - 10.8</td>
<td>9.9</td>
<td>8.7 - 11.1</td>
<td>10.6</td>
<td>9.7 - 11.1</td>
</tr>
<tr>
<td></td>
<td>10.1</td>
<td>9.4 - 10.8</td>
<td>9.9</td>
<td>8.7 - 11.1</td>
<td>10.6</td>
<td>9.7 - 11.1</td>
</tr>
<tr>
<td></td>
<td>10.1</td>
<td>9.4 - 10.8</td>
<td>9.9</td>
<td>8.7 - 11.1</td>
<td>10.6</td>
<td>9.7 - 11.1</td>
</tr>
</tbody>
</table>

*Note.* ! indicates measured used in classification criteria. !! indicates all language impaired children with at least one other disorder.
Table 3.7

Comparing AD and ADHD on mean classification and validation scores using clinical criteria

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control (n=13)</th>
<th>ADHD (n=13)</th>
<th>AD (n=6)</th>
<th>ADHD + Dyslexia (n=9)</th>
<th>Dyslexia (n=9)</th>
<th>AD + Dyslexia (n=12)</th>
<th>ANOVA</th>
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</thead>
<tbody>
<tr>
<td></td>
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<td>95% CI’s</td>
<td>M</td>
<td>95% CI’s</td>
<td>M</td>
<td>95% CI’s</td>
<td></td>
</tr>
<tr>
<td>Reading Fluency</td>
<td>96.1</td>
<td>90.6 - 101.5</td>
<td>100.3</td>
<td>94.6 - 106.0</td>
<td>89.5</td>
<td>85.5 - 93.5</td>
<td></td>
</tr>
<tr>
<td></td>
<td>80.4</td>
<td>77.0 - 83.8</td>
<td>81.1</td>
<td>77.9 - 84.3</td>
<td>80.1</td>
<td>78.1 - 82.1</td>
<td></td>
</tr>
<tr>
<td>ADHD Measures</td>
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<td></td>
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<td></td>
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<tr>
<td>Mean Hyp (P/T)</td>
<td>54.6</td>
<td>50.6 - 58.6</td>
<td>74.4</td>
<td>69.6 - 79.2</td>
<td>75.5</td>
<td>71.9 - 79.2</td>
<td>F(5,59) = 13.17, p &lt; .001</td>
</tr>
<tr>
<td></td>
<td>55.2</td>
<td>46.1 - 64.2</td>
<td>72.8</td>
<td>67.3 - 78.2</td>
<td>51.0</td>
<td>46.7 - 57.2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>56.6</td>
<td>53.6 - 59.5</td>
<td>56.6</td>
<td>53.6 - 59.5</td>
<td>56.6</td>
<td>53.6 - 59.5</td>
<td></td>
</tr>
<tr>
<td>Mean AD (P/T)</td>
<td>55.1</td>
<td>50.8 - 59.5</td>
<td>75.5</td>
<td>71.9 - 79.2</td>
<td>71.2</td>
<td>64.9 - 77.5</td>
<td>F(5,59) = 19.87, p &lt; .001</td>
</tr>
<tr>
<td></td>
<td>76.4</td>
<td>73.6 - 79.3</td>
<td>56.5</td>
<td>51.9 - 61.1</td>
<td>57.5</td>
<td>51.2 - 67.3</td>
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<td></td>
<td>71.5</td>
<td>72.2 - 78.1</td>
<td>75.1</td>
<td>72.2 - 78.1</td>
<td>75.1</td>
<td>72.2 - 78.1</td>
<td></td>
</tr>
<tr>
<td>Mean EF (P/T)</td>
<td>56.4</td>
<td>50.6 - 62.3</td>
<td>66.6</td>
<td>62.2 - 71.1</td>
<td>67.2</td>
<td>59.4 - 74.9</td>
<td>F(5,59) = 31.37, p &lt; .001</td>
</tr>
<tr>
<td></td>
<td>73.7</td>
<td>70.0 - 77.4</td>
<td>54.7</td>
<td>52.5 - 56.9</td>
<td>57.1</td>
<td>68.0 - 74.2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>80.8</td>
<td>89.5 - 112.4</td>
<td>100.5</td>
<td>93.4 - 107.5</td>
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<td></td>
</tr>
<tr>
<td>PRI</td>
<td>105.4</td>
<td>98.4 - 112.4</td>
<td>103.3</td>
<td>97.9 - 108.7</td>
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<td>94.7 - 110.8</td>
<td>F(5,59) = 0.63, p = .678</td>
</tr>
<tr>
<td></td>
<td>89.8</td>
<td>82.4 - 100.6</td>
<td>100.5</td>
<td>93.4 - 107.5</td>
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</tbody>
</table>

The three groups with dyslexia did not differ significantly on reading fluency. The AD only group had significantly poorer reading fluency than the ADHD group, who did not differ from the control group.

The two groups with ADHD did not differ significantly on hyperactivity symptoms. Both groups reported significantly greater symptoms than all other groups who did not differ.

The group with dyslexia and the control group did not differ significantly and had significantly lower scores on the AD and executive functioning index than all other groups who did not differ significantly from one another.

The group with dyslexia performed more poorly than the control group. There were no other group differences.
3.16 Language and other disorders

On each of the reading and attention measures the multiple deficit group did not differ from the groups with dyslexia on reading fluency, or the groups with AD on reports of attentional difficulties. The language impaired group performed significantly more poorly than all other groups on the measures of language and non-verbal ability.

3.17 Comparing AD and ADHD using clinical criteria

Inspection of Table 3.7 shows that the AD only group had significantly poorer reading fluency than the ADHD group, who did not differ from the control group. The ADHD groups (single or multiple deficit) did not differ in severity on the hyperactivity measure. Both groups had significantly more symptoms than all the other groups who did not differ. On the measures of AD and executive function, the group with dyslexia only and the control group did not differ significantly and had significantly lower scores on these measures than the other groups who did not differ from one another. No other significant group differences were found.

3.18 Discussion

The purpose of this study was to investigate the prevalence of single and multiple developmental disorders in a sample of primary school children. The participants were recruited based on a clinical diagnosis of dyslexia and/or ADHD, or they were perceived by their parents to have reading and/or attention difficulties. We also evaluated whether children with more than one deficit (each at a clinical and subclinical level) had more severe symptoms of each disorder than children with a single disorder only. The impact of single and multiple disorders on reading and attention, and the implications of these findings for the assessment and remediation of developmental disorders are discussed.
Prevalence of single and multiple disorders

Overall, the prevalence rates for single and multiple disorders were similar when clinical classification criteria were adopted, with AD being more prevalent than dyslexia. However, when subclinical symptoms were included, most children had more than one disorder, indicating that many children diagnosed with a single disorder will have subclinical symptoms of at least one other disorder. These findings indicate that the assessment of a child with suspected reading difficulties should also include an assessment for ADHD, particularly an attention deficit, while a child suspected of having AD or ADHD should also be assessed for reading difficulties. The finding that there were children with a single diagnosis of dyslexia, ADHD or AD, as well as children with a diagnosis of both disorders suggests that these are separate but co-occurring disorders, with independent underlying aetiologies (Sexton et al., 2012). This is consistent with genetic studies that have found shared (Gayán et al., 2005; Willcutt, Pennington, et al., 2007; Willcutt et al., 2002), as well as specific genetic risk factors (e.g., Loo et al., 2004) to be associated with dyslexia and ADHD.

Prevalence of dyslexia and other disorders

Multiple disorders, specifically dyslexia + AD, were more prevalent than a single diagnosis of dyslexia, and this was evident even when the more stringent clinical criteria were adopted. The prevalence estimate for a single disorder was considerably higher in the current study than those reported in unselected samples. This difference was expected as the sample was biased, with approximately 39% of the participants having a previous diagnosis of dyslexia, and 6% with a previous diagnosis of co-occurring dyslexia + ADHD. In addition, many children even without a diagnosis of dyslexia were undergoing remediation for these difficulties. The finding that more than two-thirds of children with dyslexia had an additional attention deficit is consistent with studies that have found a strong genetic association between reading and inattention (e.g., Ebejer et al., 2010; Greven et al., 2012;
Willcutt, Pennington, et al., 2007), and indicates that children with clinical symptoms of dyslexia also frequently have significant attention difficulties.

**Prevalence of ADHD or AD and other disorders**

Using clinical criteria, the prevalence estimates for the group with AD alone, and the group with co-occurring AD + dyslexia were similar (45.7%). However, when subclinical symptoms were included in the classification of dyslexia, more than half the children displayed symptoms of AD + dyslexia (63%), with a much smaller proportion of children showing evidence of AD alone (26.1%). These findings indicate that a high proportion of children with clinical symptoms of AD also have reading difficulties at clinical and subclinical levels. When single and multiple disorders were considered for ADHD, the results were consistent with those found for the AD subtype. A single diagnosis of ADHD was more prevalent using clinical criteria, however when subclinical symptoms were considered more than half the children with ADHD had additional reading difficulties. Within the ADHD prevalence literature, the rates of co-occurring dyslexia using clinical criteria are reported to be between 15% to 45% (Carroll et al., 2005; Sexton et al., 2012; Willcutt et al., 2001). This is consistent with the overlap found in this study between both AD + dyslexia, and ADHD + dyslexia. However, the clinical estimate for ADHD alone was higher than estimates reported in a recent international meta-analysis (Thomas et al., 2015). This might have occurred because different populations were used. The review examined the prevalence of child and adolescent ADHD in 175 studies that used the diagnostic criteria from DSM-III, DSM-III-R, or DSM-IV, with samples from community, school populations, or national surveys (intervention or treatment studies were excluded). The review included 179 ADHD prevalence estimates and established a benchmark pooled prevalence estimate of 7.2% compared to 31.9 % in the current study. The authors suggested that population rates that
exceed this estimate, such as those reported in the current study, may reflect over-diagnosis of the disorder (Thomas et al., 2015).

However, other recent studies that have also applied DSM (DSM-IV-TR) diagnostic criteria estimate the prevalence of ADHD to be significantly higher than that reported by Thomas and colleagues, and an argument for the under-estimation of the disorder has been submitted (Rowland et al., 2015). For example, Rowland and colleagues (2015) used population-based samples, multiple informants as well as DSM-IV criteria to estimate the prevalence of ADHD in school-aged children. The study derived three groups from a sample of 1160 children: 475 children with ADHD, 442 children without ADHD, and 243 children identified with sub-threshold symptoms of ADHD. The study found that their estimate of 15.5% was significantly higher than those commonly reported for ADHD. The authors proposed a number of explanations for the underestimation of ADHD prevalence: 1) clinical diagnosis in community samples misses many children, particularly those with subclinical symptoms, 2) no systematic screening at school for all children with ADHD, and school referrals are influential in determining which children are identified, 3) many studies exclude children on medication because of a positive medication response, 4) the age-of-onset criterion may eliminate some children as inattention has been found to emerge later than hyperactive/impulsive symptoms (Barkley & Biederman, 1997), 5) many studies have failed to include younger children, and this is when prevalence of ADHD peaks (Skounti, Philalithis, & Galanakis, 2007), and 6) studies using only parents have missed teachers as a key source of data that likely limits prevalence estimates.

In the current study, the estimates for AD and ADHD were still considerably higher than the 15.5% reported by Rowland and colleagues (2015), however this again was expected, as the study was not a non-selected community sample. It was specifically targeted toward children with a diagnosis of dyslexia and ADHD, or those underperforming in school,
potentially because of reading and attention difficulties. On this basis alone, the prevalence rates for ADHD and AD would be higher. In addition, diagnosis was based on parent and teacher reports only, and as the full diagnostic classification was not used, this may have resulted in the over-identification of some cases. Further, many parents in the study noted their children had academic difficulties and were underperforming in school. Consequently, the inattentive symptoms noted by parents may be a result of these academic difficulties, rather than a true attention deficit disorder. In addition, inattention has been found to emerge later than hyperactivity/impulsivity (Barkley & Biederman, 1997), and this may provide another explanation for the higher estimation of AD in this sample, as many studies have not included children whose symptoms did not meet the age-of-onset criterion.

In the current sample, there were twice as many children with AD as there were with ADHD combined subtype. However, when the children with AD were disregarded there was no significant difference between the ADHD, ADHD + dyslexia and the dyslexia group alone (which contains many of the children with AD alone) on reports of severity of attentional difficulties. The group with dyslexia alone contained many children with an attention deficit (without hyperactivity) because they failed to meet the full ADHD diagnostic criteria. As most studies assess only for ADHD, many children who have an attention deficit without hyperactivity fail to be diagnosed but are subsequently included in dyslexia samples when they have a co-occurring reading disorder. This finding supports many studies reporting poorer attentional focus in children with dyslexia, but also indicates that children with dyslexia alone may not all have problems with attentional focus. These findings also support previous reports that AD is more closely associated with dyslexia than the combined ADHD subtype, as reading difficulties may result in a lack of academic engagement and focus, while inattention and poor focus may negatively impact the acquirement of reading skills. However, it should be noted that a diagnosis of AD based on rating scales alone may not be
sufficient to identify true cases of AD, and further clinical evaluation may be warranted as parents and teachers may have over-estimated the reports of inattention.

**Overlap between dyslexia and AD or ADHD**

The finding that the group with dyslexia showed subclinical evidence of AD, and the groups with AD and ADHD showed subclinical evidence of dyslexia supports the findings of Willcutt and colleagues (2007) who investigated children with dyslexia and ADHD longitudinally. The sample was initially assessed at approximately 10.57 years and was then followed up five years later. The results showed that although the individuals in the single deficit group did not meet criteria for another co-occurring diagnosis, they exhibited subclinical symptoms of the other disorder (Willcutt, Betjemann, Pennington, et al., 2007).

Similarly, Karmiloff-Smith and colleagues have proposed that general, low-level processing deficits may affect several ability domains rather than just one isolated deficit. Consequently, these researchers warn against a research strategy that ignores lower level (subclinical) deficits (Karmiloff-Smith, 1998, 2009). In addition, there were few individuals in the current study without clinical or subclinical deficits, supporting the reports of these parents that their children were not performing as well as expected academically.

**Dyslexia, AD, ADHD and their co-occurrence with SLI and DCD**

Despite some studies reporting high rates of co-occurrence between dyslexia + ADHD or AD with language impairment and DCD, few children in the current study were identified with these additional disorders. Of the 5 children in the current sample who were identified with a language impairment, one had SLI alone, with the remaining children showing evidence of dyslexia and SLI, or ADHD and SLI. No children in the sample were found to be clinically impaired on the motor tasks, and this may explain the lack of association between AD or ADHD with SLI. However, some studies have observed that it is
children with the combined ADHD subtype and co-occurring motor impairments who are most likely to also have language deficits (Tervo et al., 2002).

Another explanation for the lack of association between these disorders may be our use of objective language measures to identify SLI (e.g., CELF-IV) rather than reliance on rating scales (e.g., the Child Behaviour Checklist (CBCL) and the Teacher Report Form (TRF; Achenbach, 1991), as these have been found to incorporate indicators of attention difficulties (Redmond & Rice, 2002). Some studies have also found that the early language difficulties of many children resolve, and many are reported to have at least average language skills by 12 years of age (Hayiou-Thomas et al., 2014). The current sample was aged between 9 and 11 years, and this is the period when language difficulties have been observed to resolve and may provide an alternative explanation as to why few children with either dyslexia, AD or ADHD were identified with SLI. Further, the majority (65%) of children in the sample were receiving remediation for literacy at school and/or privately, and some researchers have suggested the language difficulties of children who receive effective treatment or educational interventions may be ameliorated (Hayiou-Thomas et al., 2014).

The finding that few children with dyslexia alone showed evidence of motor deficits is consistent with those of a recent longitudinal investigation by Gooch and colleagues (2014). The study examined whether family risk of dyslexia and early language impairment were associated with co-occurring weaknesses in motor skills and attention in a sample of 3 to 5-year old children. Using the same standardized measures as this study (CELF-IV and MABC-2), the motor performance of children at family risk of dyslexia only, and family risk of dyslexia + current language impairment was compared to the performance of children with a current language impairment only and typically developing controls. The study found that children at family risk of dyslexia without a language impairment performed better than those with a language impairment on the objective measures of motor skills, leading the authors to
question whether motor deficits are only apparent (or more apparent) in individuals with a history of language impairment (Gooch et al., 2014). As few children were found to have a language impairment, this may also explain the lack of motor deficits in the current sample. The lack of association between dyslexia and DCD is also consistent with an earlier study by Kaplan et al., (2001), who found no evidence of children with co-occurring dyslexia + DCD, as well as a meta-analysis by Rochelle and Talcott (Rochelle & Talcott, 2006) who observed a stronger association between DCD and ADHD than between DCD and dyslexia.

A more recent study by Marchand-Krynski, and colleagues (2017) assessed increasingly complex gross motor skills in children and adolescents. In line with the current study, the results indicated normal performance for children with AD, dyslexia, and AD + dyslexia on simple motor-speed tests such as tapping, however all three groups were found to be impaired on complex motor coordination (out of phase movements) tasks (Marchand-Krynski et al., 2017). The motor tasks used in the current study were designed to screen for gross and fine motor impairment and were likely more comparable to simple motor-speed tasks, than the complex tasks used by Marchand-Krynski and colleagues. In addition, participants in the current study spanned two age bands (7 to 10 and 11 to 16 years) on the MABC tasks (Henderson et al., 2007). The mean age of the participants was 9.83 years, placing most at the upper end of the younger age band. Consequently, there is a possibility that the motor tasks were too easy for many of the participants, creating a ceiling effect.

**Severity of single and multiple disorder groups**

No significant differences in severity were found between the three groups with dyslexia using clinical or subclinical criteria on the reading fluency measures, indicating the presence of more than one deficit did not result in poorer reading fluency. Similarly, regardless of the classification criteria used, no differences in severity were found for the three groups with AD, indicating that the presence of more than one deficit did not result in
greater inattention, hyperactivity or executive dysfunction for these children. Further, no differences in severity were found for the two groups with ADHD using clinical criteria, indicating that the presence of more than one deficit did not result in greater inattention, hyperactivity, or executive dysfunction.

Although no differences in symptom severity were observed between the single and multiple disorders groups in the current study, others (e.g., Dyck & Piek, 2010; 2014) have found children with diagnosed disorders were up to seven times more likely to have clinically significant deficits across language and motor domains. However, these studies compared children with one disorder to those with no disorders (control group), while this study compared children with one disorder to those with two disorders. There are also other explanations for the differing findings. For example, previous studies have not investigated the pervasiveness of difficulties in a sample of children with dyslexia, or the combination of dyslexia and AD. Instead, samples have predominantly included children aged 3 to 18 years with severe neurodevelopmental disorders such as Williams Syndrome (Karmiloff-Smith & Thomas, 2003), or severe language, motor and behavioural disorders (Dyck & Piek, 2010; 2014). The focus of the current study is dyslexia, while the samples used in these studies represented children with a severe developmental disorder (Karmiloff-Smith & Thomas, 2003), or who were recruited from specialist language units and occupational therapists. For example, the study by Dyck and Piek (2010) used children with language deficits who were recruited from language development clinics and had severe receptive and expressive language impairments. In addition, although the participants with motor difficulties did not have a formal diagnosis of DCD, they were referred by special education teachers and occupational therapists and were subsequently found to have scores at the 5th percentile or lower. The participants used in these prior studies were selected based on their severe language and motor impairments and are not representative of the individuals in the current
study who primarily have a reading impairment. Finally, most studies have adopted a broader age range. In the current study the narrow age range was designed to produce greater stability across the assessment measures (e.g., Dyck et al., 2009).

None of the groups, except for children with SLI, showed evidence of poor language skills. While we expected the groups with dyslexia + AD or dyslexia + ADHD to show poorer performance than the single disorder and control groups, it was the small group with SLI + dyslexia/AD who were observed to have significantly poorer reading, language, and non-verbal ability than the other groups. Despite the small group size, the addition of SLI with dyslexia + AD had an association with the language, literacy, and non-verbal skills of this group. This finding is consistent with the model of dyslexia and SLI proposed by Bishop and Snowling (2004). For example, the group with SLI did not differ from the other dyslexia groups on the reading fluency measure, supporting the proposal of common phonological processing deficits. However, the group with SLI performed significantly more poorly on the language measures than the groups with dyslexia (who did not differ from the control group), and this is in line with their hypothesis that there are additional cognitive deficits associated with SLI that result in language impairments. These findings highlight the importance of including language measures in a developmental assessment, as the additional language deficits in the current sample had a strong impact on the performance of these children.

3.19 Conclusion

This study found evidence to support the hypothesis of multiple disorders occurring commonly among primary school children who are showing evidence of reading and attention difficulties in school. The finding that the groups with co-occurring dyslexia + AD or dyslexia + ADHD were not more impaired than the single disorder groups may reflect the fact that this was a self-referring sample. The participants therefore may not have been as impaired as a clinical sample, which usually identifies more severe cases of a disorder.
Similarly, studies that have not controlled for other co-occurring disorders may have attributed more severe presentations to the combination of dyslexia + ADHD when other undiagnosed disorders such as language impairment may be present.

There was also extensive evidence of subclinical dyslexia in children with AD, and subclinical AD in children with dyslexia, justifying the use of continuous groupings. These findings accord with others that have found that even when individuals with dyslexia or ADHD do not meet full criteria for both disorders, they often exhibit subclinical elevations of the other disorder (Willcutt, Betjemann, Pennington, et al., 2007). These results support a comprehensive assessment of children with a known developmental disorder, as well as for children who are generally not performing as well as expected academically, as the results may provide important information for educators and clinicians in identifying clinical as well as subclinical difficulties, and for identifying appropriate remedial interventions. These finding should be interpreted with the consideration that there could be some level of overestimation of ADHD, and particularly AD in this sample. This could be the result of academic difficulties, age of on-set, or parents over reporting symptomatology.

Recent research has shown that dyslexia and ADHD are each highly heritable disorders that share some of the same genes, although the association has been primarily attributed to genetic influences shared between dyslexia and AD (Gayán et al., 2005; Greven et al., 2011; Greven et al., 2012; Nigg, Nikolas, & Burt, 2010; Paloyelis et al., 2010; Willcutt, Pennington, et al., 2007). Until recently, the cognitive processes underlying a behavioural outcome (like reading ability or attention) and the impact of familial risk (e.g., for dyslexia or ADHD) have been studied in isolation (van Bergen, Bishop, van Zuijen, & de Jong, 2015). However, the difficulties associated with dyslexia and ADHD are longstanding (Das, Cherbuin, Butterworth, Anstey, & Easteal, 2012; Larsson, Chang, D’Onofrio, & Lichtenstein, 2014; Snowling, Muter, & Carroll, 2007) and current research highlights the important role
of family risk in the manifestation of each disorder, as well as the implications of identifying these pre-cursors for preventative support, early intervention and clear academic recommendations (Gallagher et al., 2000; Snowling et al., 2003; Snowling et al., 2007; Velez-van-Meerbeke, Talero-Gutierrez, Zamora-Miramon, & Guzman-Ramirez, 2017). Moreover, the issue of subclinical symptomatology has been highlighted for children at family risk of dyslexia, as literacy outcomes have been noted to be distributed continuously, and children who do not meet criteria for a clinical diagnosis of dyslexia have been found to have subclinical literacy and language deficits (Boets et al., 2010; Gallagher et al., 2000; Pennington & Lefly, 2001; Snowling & Hulme, 2008; Snowling & Melby-Lervåg, 2016; van Bergen et al., 2012; van Bergen et al., 2011). These findings have important implications for identifying children at family risk, as subclinical literacy, language or attention deficits are more likely to be undetected and may place these children at risk of poorer academic attainment. Following in Chapter 4, the validation of a self-report protocol for reading and attention deficits in adults is presented. In addition, the prevalence of dyslexia, ADHD and AD in a parent sample is discussed in relation to determining whether there are associations between parent and child symptomatology.
Chapter 4: Family history and the prevalence of dyslexia and ADHD: the association between parent self-report and children’s performance on reading and attention measures.

4.1 Introduction

Developmental dyslexia and ADHD are the two most common and complex of the childhood neurodevelopmental disorders (Bental & Tirosh, 2007). Dyslexia is recognised as a highly heritable language based disorder that persists through development (Pennington & Gilger, 1996; Pennington & Olson, 2005). It is also well recognised that ADHD has a strong genetic component (Loo et al., 2004), with little evidence for the involvement of shared environmental influences on the individual differences in ADHD symptoms (Brikell, Kuja-Halkola, & Larsson, 2015; Larsson, Anckarsater, Råstam, Chang, & Lichtenstein, 2012; Thapar, Cooper, Eyre, & Langley, 2013). Consequently, having a family member with dyslexia and/or ADHD conveys a much greater risk for the development of the disorders, and recent studies have noted the advantages of incorporating screening protocols to estimate parental risk of reading and attention difficulties, to identify those children at heightened risk (Snowling et al., 2012). The focus of the current study was first to validate an adult reading and ADHD screening protocol, second to determine the prevalence of reading and attention deficits in the parent sample, and finally to determine whether these were associated with the prevalence of single or multiple disorders in the child sample.

Family risk of developmental disorders: genetics

While familial studies have demonstrated that the relative risk of dyslexia is 4 to 8 times higher for individuals with a first-degree relative with dyslexia, and 6 to 8 times higher for individuals with a biological family member with ADHD (Boada et al., 2012), they cannot determine whether the underlying aetiology of the disorder, or the source of comorbidity, is due to genetic or environmental factors. Consequently, twin studies have
been conducted to estimate the degree to which a disorder is due to genetic and/or environmental influences (Boada et al., 2012; Willcutt, Betjemann, et al., 2010). Indeed, behavioural genetic studies that have compared relative similarities of monozygotic and dizygotic twins have found that dyslexia and ADHD share some of the same genes, supporting a genetic overlap between these two conditions (Willcutt et al., 2000). The association between the disorders has been largely attributed to genetic influences shared between reading and the inattentive (AD) symptom domain of ADHD (Gayán et al., 2005; Greven et al., 2011; Greven et al., 2012; Nigg et al., 2010; Paloyelis et al., 2010; Willcutt, Pennington, et al., 2007), while other literacy variables such as print knowledge, vocabulary and grammar have been found to be primarily influenced by shared environmental factors (Ebejer et al., 2010).

Evidence from cross-sectional twin studies investigating genetic overlap between dyslexia and inattention indicate that the genes involved in their association are highly stable over time, and are consistent in the childhood (Ebejer et al., 2010) and adolescent stages of development (Greven et al., 2012). For example, Ebejer and colleagues (2010) examined the developmental trajectories of reading and inattention in 989 monozygotic and dizygotic twin pairs aged between 6 and 9 years from Australia, Norway, Sweden, and the United States. The results of the study supported previous findings of a genetic overlap between dyslexia and inattention, as well as indicating that this overlap is in place from kindergarten onwards, with no new genetic contribution to the relationship during this period of development (Ebejer et al., 2010). Ebejer and colleagues concluded that the best explanation of the association between dyslexia and inattention in children aged 6 to 8 years was a common genes account, however, from 8 to 9 years of age, inattentiveness was found to impact reading ability more directly, a result they attributed to the greater cognitive demands associated with reading and reading comprehension at that age. Other longitudinal twin
studies investigating reading, inattention, and hyperactivity/impulsivity at the preschool level found that reading variables such as phonological awareness, rapid naming and verbal memory were each subject to substantial genetic influence (Byrne et al., 2002; Samuelsson et al., 2005), as were parent-derived reports of inattention and hyperactivity/impulsivity (Ebejer et al., 2010; Willcutt, Betjemann, Wadsworth, et al., 2007). In addition, the reading variables were significantly correlated with inattention, but not the hyperactive/impulsive symptom domain (Willcutt, Betjemann, Wadsworth, et al., 2007).

**Family risk of dyslexia**

Heritability for dyslexia is multifactorial and continuous, and is thought to be modified by the complex interaction of genetic effects acting alongside environmental mechanisms that result in heterogeneity in literacy outcomes (Snowling et al., 2007). For example, the presence or absence of phenotypical markers such as phonological awareness or short-term memory deficits, or indicators of other developmental disorders such as inattention or language impairment lead to continuous variations in the dyslexia phenotype (Snowling et al., 2007). In addition, evidence indicates that the characteristics of dyslexia may change with age. For example, compensations motivated by prolonged exposure to written text, and education (Fink, 1998; Lefly & Pennington, 1991, 2000) mean that although compensated adults may no longer meet the childhood criteria of dyslexia, sub-clinical symptoms may be evident, and when complex reading requirements exceed compensatory mechanisms, they continue to have significant reading and writing difficulties (Griffiths & Frith, 2002; Lefly & Pennington, 1991). There is also research to suggest that boys may be more vulnerable than girls to dyslexia (Wolff & Melngailis, 1994). While this is controversial (e.g., Shaywitz et al., 1990), a review examining ascertainment bias in the selection of individuals with reading difficulties determined there is still a preponderance of boys with dyslexia (Liederman, Kantrowitz, & Flannery, 2005). Moreover, some studies
investigating the gender ratios of affected adult family members of individuals with dyslexia, have also observed a male vulnerability for the disorder (e.g., Wolff & Melngailis, 1994). In addition, it has been noted that those studies which failed to find a significant male bias lacked the statistical power necessary to detect a gender ratio of 2.0 (Liederman et al., 2005). Further, a more recent study investigating reading development and its precursors in children differing in family risk for dyslexia, found in both at-risk groups (at risk dyslexic and at risk non-dyslexic), almost 75% of the children had a father with dyslexia (van Bergen et al., 2011).

Depending on the definition of dyslexia (categorical disorder versus continuously distributed trait) and differences in the criteria used to define the clinical and subclinical range, between 18% and 72% of children at family risk (FR) go on to develop dyslexia, whereas the percentage of children without FR ranges from 6% to 16% (e.g., Blomert & Willems, 2010; Pennington & Lefly, 2001; Plomin & Kovas, 2005; Torppa, Lyytinen, Erskine, Eklund, & Lyytinen, 2010; van Bergen et al., 2011). The use of continuous measures includes those children with subclinical difficulties, increasing the prevalence estimates for the disorder. The importance of identifying children at FR of literacy difficulties and examining the intergenerational transfer of reading skills has recently been highlighted in the literature (Dilnot, Hamilton, Maughan, & Snowling, 2017; Hayiou-Thomas, Carroll, Leavett, Hulme, & Snowling, 2017; Snowling et al., 2012; van Bergen, de Jong, Maassen, & van der Leij, 2014). For example, Snowling and colleagues (Gallagher et al., 2000; Snowling et al., 2003; Snowling et al., 2007) conducted a longitudinal study of children with a family history of dyslexia starting from the age of 3 years, 9 months. Parents who had volunteered as ‘dyslexic’ by self-report completed a set of literacy tests to assess their skills, as well as participating in a semi-structured interview. The children were assessed on nonverbal, language, phonological and literacy measures at 3.9 years. At 6 years, the test battery
included general cognitive ability, language measures, basic reading, reading comprehension, spelling, and phonological measures. When children were 8 years of age, the assessments included cognitive abilities, reading comprehension, literacy skills and phonological awareness measures. When the children were aged 12 to 13 years, they were given a battery of tests of literacy and language skills and were classified into impaired and unimpaired groups. The children were also assessed for problems in sustained attention as well as attention switching, which required inhibiting a familiar response. The results indicated that the literacy difficulties of children at FR of dyslexia are longstanding, with no evidence of catch-up in reading skills between 8 and 13 years of age. Further, in adolescence ‘at risk’ unimpaired children showed weak orthographic skills and were significantly less fluent at reading than controls. They were also as slow as ‘at risk’ impaired readers on timed tasks, suggesting reading skills were not fully automatic in both at-risk impaired and non-impaired participants. These findings are consistent with other studies that have found FR children who do not fulfil criteria for dyslexia perform significantly more poorly than controls on certain literacy (e.g., grapheme knowledge, spelling, nonword reading accuracy) measures (Boets et al., 2010; Gallagher et al., 2000; Pennington & Lefly, 2001; Snowling, 2008; Snowling et al., 2003; van Bergen et al., 2012; van Bergen, van der Leij, et al., 2014), and supports the proposal that familial risk of dyslexia is continuously distributed (Pennington & Lefly, 2001). Snowling and colleagues (2007) also found that the at-risk dyslexia group, but not the at-risk no dyslexia group were impaired on an attention switching measure, and this finding was supported by parent-ratings of inattention, as well as by cognitive measures. In addition, although the study found that parent and child literacy levels were correlated in control families (indicating heritability of reading skills), there was a lack of association between parental and child literacy levels in children with a FR of dyslexia (i.e., it was not the case that more severely affected parents had more severely affected children), leading the
authors to suggest that these findings indicate an unusual route for literacy development in children with a family history of dyslexia. However, this conclusion was contradicted by the findings of van Bergen and colleagues (2011) who found that within the group of parents with dyslexia, those whose child also developed dyslexia at age 11 performed more poorly in both word and pseudoword decoding tasks. The authors suggested these findings agree with a continuous view of the family risk of dyslexia; parents with more severe dyslexia are genetically more affected, and will have offspring who are also at a higher genetic risk of the disorder (van Bergen et al., 2011). Indeed, studies investigating an equal-liability assumption for the two FR groups (FR dyslexia and FR no dyslexia) have found that the parents of affected children were more severely impaired than those of unaffected children (Torppa, Eklund, Bergen, & Lyytinen, 2011; van Bergen et al., 2012; van Bergen et al., 2011).

Another study by van Bergen and colleagues (2014) examined the intergenerational transfer of reading skills in a sample of 196 children at FR of dyslexia. When children were 3½ years old, parents were asked to complete a questionnaire about their own literacy, and the home literacy environment. At 6 years of age children were tested on preliteracy skills, and on school achievement at age 9. The results showed that within the FR sample, the non-dyslexic parent of children with dyslexia, reported on average more difficulties themselves, and that the self-reported literacy (reading and spelling) difficulties of the non-dyslexic parent differentiated family risk children with and without dyslexia. The authors concluded that these findings were in support of the view that parental skills are indicative of a child’s liability, and that children who go on to develop dyslexia have a higher genetic predisposition towards the disorder. In contrast, the study did not find that the self-reported literacy difficulties of the dyslexic parent made any additional contribution to the prediction of children’s reading beyond the fact that they had dyslexia (van Bergen, de Jong, et al., 2014). However, this was not the case in an earlier study that assessed the same sample using an
objective measure of parental word reading fluency (van Bergen et al., 2012). In the previous study the results indicated that children at FR, with and without dyslexia, could be differentiated using word reading fluency. The authors suggested the discrepancy reflected measurement differences, and that using objective measures to assess parental skills may be more reliable than using self-reported difficulties. However, despite this disparity, an intergenerational transfer of literacy skills was observed when the risk and no-risk samples were combined. A regression analysis showed that even after accounting for risk and differences in the educational level of each parent, parental literacy difficulties predicted children’s reading fluency. However, parental literacy was no longer predictive when children’s pre-literacy skills were considered (van Bergen, de Jong, et al., 2014), and indicates parental literacy is a distal risk factor for poor reading fluency. When skills such as poor phonological processing, letter knowledge, and slow rapid naming are added to the regression equation, this distal factor failed to continue to make a significant unique contribution to children’s reading fluency.

A recent meta-analysis by Snowling and Melby-Lervåg (2016) reviewed 95 FR studies in differing alphabetic languages. The studies included 420 children with dyslexia, and the results indicated a heightened prevalence of dyslexia in the offspring of affected parents, with an estimated 45% of “at risk” children developing dyslexia. The findings were consistent across languages; children from families with an impaired first-degree relative were four times more likely to have reading difficulties than children without family risk. The study found that infants and toddlers at FR of dyslexia experienced delayed language development, and had significant difficulties acquiring the foundations of word decoding (letter knowledge, phonological awareness and rapid automatic naming) in the preschool years. Their review of longitudinal studies indicated that preschool children at FR who later fulfilled criteria for dyslexia had more severe language impairments in preschool than FR
children without dyslexia. Furthermore, children who did not go on to develop dyslexia performed less well than controls. Similarly, at school age, FR of dyslexia was associated with significantly impaired phonological awareness and literacy skills. The study also conducted an analysis of moderator variables which indicated that the cut-off for diagnosing dyslexia had a significant impact on the mean prevalence. For example, when the criterion for diagnosing dyslexia was set above the 10th percentile, the mean prevalence was 53%, but fell to 33.7% when the criterion was set below the 10th percentile.

To estimate how much the risk of dyslexia is increased in families with a history of reading difficulty, the review also assessed the prevalence of dyslexia in 540 control children without a FR. The review found that the mean prevalence of dyslexia in samples without FR was 11.6%. In studies where the criterion for diagnosing dyslexia was above the 10th percentile, the mean prevalence was 16% while in studies with a criterion below the 10th percentile, the mean prevalence was 7.8%. Literacy outcomes were found to be distributed continuously, and many children who did not meet criteria for a clinical diagnosis of dyslexia were found to have subclinical reading impairments (Snowling & Melby-Lervåg, 2016). While the review found no significant evidence to indicate children at family risk may be brought up in an environment that differs significantly from controls, there was a tendency for the children’s parents to have lower educational attainment, and to read less frequently to them. For example, during the preschool years, children who were later identified as impaired readers were read to less often by their fathers, while for mothers there was less joint reading at 2.5 years than for controls. Similarly, several studies have found that the literacy outcomes of children in a family-risk group were unrelated to home, family or school environmental factors. The studies found that the home environment of children at family risk (with and without dyslexia) did not differ in terms of shared reading, parental reading and writing frequency, access to print (library membership or number of books in the home).
or cognitive stimulation (e.g., Elbro, Borstrøm, & Petersen, 1998; Torppa et al., 2007; van Bergen et al., 2011; van Bergen, van der Leij, et al., 2014) although van Bergen and colleagues (2014) found that control families tended to have more newspapers and books in the home. These findings are in accord with behavioural genetic studies that indicate low levels of shared environmental influence (van Bergen et al., 2012; van Bergen, van der Leij, et al., 2014) and confirm the elevated risk of dyslexia among the children of impaired parents, with up to 80% attributed to genetic influences (e.g., Byrne et al., 2009; Haworth et al., 2009).

Overall, the research literature provides evidence that children at family risk are at a significantly higher risk of developing dyslexia, and even when they are not diagnosed with dyslexia, they perform more poorly on many literacy and language measures.

**Family risk of ADHD**

The underlying causes of ADHD are also multifactorial and influenced by both genetic and environmental factors (Biederman, 2005; Faraone & Doyle, 2001). According to adoption studies of children with ADHD, shared genes rather than shared environment are thought to be primarily responsible for the transmission of the disorder, with reports of 18% ADHD in biological parents versus 6% in adoptive parents (Faraone, Biederman, & Milberger, 1994). However, identifying the individual genes contributing to ADHD has proven difficult (Franke, Neale, & Faraone, 2009), and results have been conflicting with few associations replicated across studies (e.g., Faraone et al., 2005). Despite this, twin studies of ADHD report high heritability estimates for the disorder, with most studies reporting in the range of 50% to 90% for child and adolescent samples, with a mean estimate of 76% (Faraone et al., 2005; Greven et al., 2011; Greven et al., 2012; Nikolas & Burt, 2010; Thapar et al., 2013), regardless of whether the disorder is measured as a dimensional trait or a categorical disorder (Neale et al., 2010). Family risk studies investigating ADHD in clinical
samples have also consistently shown that the disorder aggregates in families (Brikell et al., 2015), and findings indicate a large proportion of children with ADHD (25% to 55%) have at least one affected parent (Smalley et al., 2000; Starck et al., 2016; Takeda et al., 2010), with an even larger percentage of parents with subclinical ADHD symptomatology (Thissen et al., 2014). While ADHD is highly prevalent in children at family risk, research has failed to find any significant familial correlations between ADHD traits (Faraone, Biederman, & Friedman, 2000; Faraone, Biederman, Mick, et al., 2000; Faraone, Biederman, Spencer, et al., 2000) as parents and their offspring have not been found to show similar patterns of subtypes (Starck et al., 2016).

The disorder has also been noted to occur predominantly in males, thus it has been hypothesised that risk is transmitted mainly through the paternal line (Rhee, Waldman, Hay, & Levy, 1999). Although several studies have found support for a stronger paternal transmission of the disorder (Hawi et al., 2010; Quist et al., 2003; Smoller et al., 2006; Takeda et al., 2010), the results are inconsistent, and some studies have found no evidence for parent-of-origin effects (Anney et al., 2008; Kim et al., 2007; Laurin et al., 2007) while others have found larger effects for maternal than paternal history of ADHD (Goos, Ezzatian, & Schachar, 2007). More recently, Thissen and colleagues (2014) used self-reported ADHD symptoms in a sample of parents, as well as data on their children’s ADHD symptoms, to examine parent-of-origin effects in ADHD, and whether such effects may be gender specific. The findings were consistent with previous studies (Faraone & Doyle, 2000; Minde et al., 2003; Sprich, Biederman, Crawford, Mundy, & Faraone, 2000) that found the ADHD symptomatology of both parents increased the risk for ADHD in their children to a similar extent, with comparable effects for boys and girls. In addition, Takeda et al. (2010) found no additive effects on the severity of a child’s ADHD when both parents, in comparison to when only one parent, had ADHD.
The genetic influences in childhood and adolescent ADHD have been found to be relatively stable. For example, a meta-analysis of longitudinal twin studies showed no evidence for changes in the heritability of ADHD from childhood to early adulthood (Bergen, Gardner, & Kendler, 2007), and some twin studies have indicated that the stability of the disorder can be attributed to genetic factors (Kuntsi, Rijssdijk, Ronald, Asherson, & Plomin, 2005; Larsson, Larsson, & Lichtenstein, 2004). Despite the presence of similar underlying factors from childhood to adulthood, studies that have explored the heritability of self-rated ADHD symptoms in adults have found estimates to be lower than those reported in child and adolescent samples. For example, the heritability of ADHD in adults has been reported to be between 30% and 44% (Boomsma et al., 2010; Polderman et al., 2013; Reiersen, Constantino, Grimmer, Martin, & Todd, 2008; van den Berg, Willemsen, de Geus, & Boomsma, 2006), with comparable estimates across rating scales, subtypes and gender (Brikell et al., 2015). The decrease in heritability is thought to coincide with the switch from using maternal ratings in children under 12 years, to relying on self-ratings in adolescence and adulthood (Brikell et al., 2015). The research literature has found that self-ratings of ADHD are significantly lower when compared to same parent and same teacher ratings, and the effect has been found to be independent of variables such as age or whether self-ratings relate to current or retrospective symptoms (Brikell et al., 2015).

Twin studies that have assessed the heritability of ADHD using different informants compared parent, teacher, and self-ratings collected at the same age in adolescence (Kan, van Beijsterveldt, Bartels, & Boomsma, 2014; Merwood et al., 2013). Parent-ratings estimated the heritability of ADHD symptoms at 82%, while self-ratings were only 48%. Estimates also varied significantly when the same teacher rated both twins within a pair (76%) compared to different teacher ratings of each twin within a pair (49%) (Merwood et al., 2013). Kan and colleagues (2014) collected data from 8,312 twins aged 12 years and
included ratings from each parent, as well as teacher and self-ratings. The study found that regardless of whether parent, teacher, or self-ratings were used, heritability estimates derived from the same parent or same teacher were consistently high at around 75%, but estimates were only 54% when collected from different raters (Kan et al., 2014). Adult studies using cross-informant data (i.e., combined parent and self-ratings or clinical diagnoses) have found heritability estimates of adult ADHD to be between 70% - 80% (Chang, Lichtenstein, Asherson, & Larsson, 2013; Larsson et al., 2014). This is comparable with results from child and adolescent studies that have used the same parent/teacher ratings as well as cross-informant methods (McLoughlin, Rijsdijk, Asherson, & Kuntsi, 2011; Merwood et al., 2013; Nikolas & Burt, 2010). These findings indicate that the heritability of ADHD in childhood and adulthood are comparable when using the same informant across time, and when cross-informant approaches are used (Brikell et al., 2015).

However, current research findings have now challenged the notion of ADHD as a childhood neurodevelopmental disorder, and the assumption that adult ADHD is necessarily a continuation of childhood ADHD (Shaw & Polanczyk, 2017). Three independent longitudinal studies following prospective community samples from New Zealand, the UK, and Brazil, from childhood to early and middle adulthood, consistently revealed that most cases of adult ADHD were not a continuation of childhood ADHD (Agnew-Blais et al., 2016; Caye et al., 2016; Moffitt et al., 2015). For example, adult ADHD cases without a history of childhood ADHD were not fully explained by comorbidities, subclinical symptoms, and information bias, and it has now been proposed that subclinical cases in childhood emerge as clinical cases in adulthood when demands exceed capacities. In addition, there is growing evidence that the genes involved in the onset of ADHD may be largely distinct from those that determine the course of the disorder, and that childhood genetic risk factors may not contribute to the adult presentation. Consequently, it has been posited that child onset and
adult onset ADHD may be two distinct syndromes with distinct developmental trajectories (Caye et al., 2016; Shaw & Polanczyk, 2017).

4.2 Adult Reading and ADHD History

Familial clustering of dyslexia was observed over 100 years ago (Hinshelwood, 1907; Stephenson, 1907; Thomas, 1905), and genetic contributions to reading development have now been identified as a primary risk factor, as research findings confirm the heightened risk of dyslexia among the offspring of parents with dyslexia (van Bergen et al., 2012). However, only a few self-report instruments have been developed and validated to screen for adult dyslexia (e.g., Reading History Questionnaire; Finucci, Whitehouse, Isaacs, & Childs, 1984; Adult Reading History Questionnaire; Lefly & Pennington, 2000). While some comprehensive adult dyslexia screening measures exist (e.g., the Dyslexia Adult Screening Test; Fawcett & Nicholson, 1998), these generally involve a battery of time consuming tests that are costly and require individual administration (Bjornsdottir et al., 2014).

A growing body of research investigating familial risk for dyslexia has observed several advantages associated with self-report measures of adult learning difficulties. For example, despite age and instruction, the deficits that impair the acquisition of literacy skills are stable, and persist into adulthood (Bruck, 1992; Davis et al., 2001; Elbro, Nielsen, & Petersen, 1994; Felton, Naylor, & Wood, 1990; Hatcher, Snowling, & Griffiths, 2002; Shaywitz, Morris, & Shaywitz, 2008; Vukovic, Wilson, & Nash, 2004), making them readily reportable in self-report measures. In addition, there is evidence that parents of children diagnosed with dyslexia recognise in themselves the same disabilities displayed by their children (Lefly & Pennington, 2000; Pennala et al., 2010; Remschmidt, Hennighausen, Schulte-Körne, Deimel, & Warnke, 1999; Scerri & Schulte-Körne, 2010). Self-report measures are also cost and time effective, and are a convenient way to assess parents who may not otherwise be prepared to commit to an extensive evaluation (Gilger, Pennington, & Defries, 1991; Schulte-Körne,
Deimel, & Remschmidt, 1997). Importantly, there is evidence from several studies to support the reliability (Deacon, Cook, & Parrila, 2012; Lefly & Pennington, 2000; Remschmidt et al., 1999; Schulte-Körne et al., 1997) and validity of self-report procedures in FR studies of dyslexia (Davis, Knopik, Wadsworth, & DeFries, 2000; Elbro et al., 1998; Finucci et al., 1984; Gilger et al., 1991; Lefly & Pennington, 2000; Schulte-Körne et al., 1997; Wolff & Lundberg, 2003). For example, in an early study using the Adult Reading History Questionnaire (ARHQ), Lefly and Pennington (2000) confirmed that the information provided by adults was accurate and valid. The ARHQ demonstrated high consistency with other diagnostic reading measures, as well as high levels of both internal consistency and test-retest reliability, and the extended length of the test-retest interval indicated the measure had considerable longitudinal stability. Furthermore, Wolff and Lundberg (Wolff & Lundberg, 2003) found self-report to be a more powerful discriminator than phonological or orthographic tasks. Overall, results from FR studies support the notion that adults have an accurate perception of their abilities, and self-reports have not been found to differ from data obtained by reading measures. In addition, self-report questionnaires gather multiple and reliable information, are not time-consuming, and aid in the identification of individuals with (or at risk of) reading impairments (Gimenez et al., 2015).

More recently, the common co-occurrence between dyslexia and AD or ADHD has highlighted the need for FR studies to incorporate screening protocols designed to estimate the risk of parental attention and hyperactivity difficulties, as well as reading difficulties (Snowling et al., 2012). Aside from the importance of early identification of children at family risk of ADHD, parental ADHD is also reported to influence family functioning and parenting practices (Banks, Ninowski, Mash, & Semple, 2008; Harvey, Danforth, McKee, Ulaszek, & Friedman, 2003; Johnston, Mash, Miller, & Ninowski, 2012; Murray & Johnston, 2006; Weiss, Hechtman, Weiss, & Jellinek, 2000). For example, it has been suggested that
inattention and impulsivity impact parenting skills by leading to impaired organisational skills (Kendziora & O'Leary, 1993), as families with parents suffering from ADHD have been observed to be less structured in everyday life (Chronis-Tuscano & Stein, 2012; Mokrova, O'Brien, Calkins, & Keane, 2010). Further, parental ADHD characteristics such as difficulty organising their environments, developing routines, or difficulties applying behaviour modifications or responding appropriately to their children’s behaviours and emotions may exacerbate their child’s symptomatology (Chronis-Tuscano & Stein, 2012; Starck et al., 2016).

One protocol used in the current cognitive literature has been found to provide a good measure of adult reading and attention difficulties and was chosen on this basis for use in this study. Snowling and colleagues (2012) adapted the Adult Reading Questionnaire (ARQ), which was drawn from several sources including the Adult Dyslexia Checklist (Smythe & Everatt, 2001), and the short-form of the Adult ADHD Self-Report Scale (ASRS: Kessler et al., 2005). The protocol includes self-rated reading, writing and spelling proficiency, expressive language difficulties (word finding), as well as a six-item screening tool for problems of organization, attention and hyperactivity in adults. The ARQ has demonstrated strong concurrent relationships with measured literacy skills (e.g., decoding fluency and spelling), and adults with self-reported symptoms of dyslexia have been found to perform more poorly on reading measures than those without those symptoms. Further, severity ratings of reading difficulties have been found to correlate negatively with measures of reading and spelling (Gilger et al., 1991; Schulte-Körne et al., 1997; Wolff & Lundberg, 2003).

Familial risk for dyslexia and ADHD have been identified as a primary risk factor and highlights the importance of identifying children at risk of dyslexia and ADHD. An estimated 45% of children with dyslexia and up to 55% of children diagnosed with ADHD have an
affected parent, with an even larger percentage of parents with subclinical difficulties (Pennington et al., 2012). The early identification of dyslexia is paramount, as early intervention has important implications for remedial success. Many parents are unable to undertake an extensive evaluation, consequently self-report measures are important as they provide a convenient and simple way to identify children at family risk. There are few validated self-report measures available, and this is the first Australian study to replicate the factor structure of the ARQ/ASRS independently of the authors. This is also the first study to distinguish between ADHD and AD when investigating parent report of these disorders.

4.3 Aims of the present study

The mothers and fathers of participating children reported their own reading, attention and hyperactivity difficulties by responding to the Adult Reading Questionnaire (ARQ: Snowling et al., 2012) and the World Health Organisation (WHO) Adult ADHD Self-Report Scale (ASRS: Kessler et al., 2005; Kessler et al., 2007). In the current study, our primary aim was to replicate the factor structure of the ARQ and ASRS, and to validate the findings with the current sample of parents. The second aim was to replicate prior research using the parent data to determine the prevalence of symptoms of dyslexia and ADHD in the parent sample. The scores of parents on each of the validated scales were used to determine if parent-reports of symptoms of dyslexia, AD or ADHD were associated with the prevalence of single (dyslexia, AD, ADHD, Specific Language Impairment) or multiple disorders in the children assessed in the current study. Based on previous research indicating that a substantial number of children at family risk of dyslexia or ADHD develop the disorders, it was expected that children from families with parents reporting high symptomatology of dyslexia, ADHD or AD would be more likely to have these disorders than children without a family history of these disorders.
4.4 Method

Participants

This sample consisted of 64 children aged between 9 and 11 years (49 boys, and 15 girls) who were recruited as part of a larger investigation that has been described in detail in Chapter 2. There was a total of 117 parents (64 mothers and 53 fathers) who completed the self-report reading, attention and hyperactivity questionnaire. This study had University Human Ethics Committee approval. Caregivers, schools, and teachers provided written consent, and child participants gave written assent prior to the commencement of testing.

Classification Measures

Children were classified with dyslexia using the Sight Word Efficiency (SWE) and Phonemic Decoding Efficiency (PDE) subtests (Form A) from the TOWRE-2 (Torgesen, et al, 2012). Participants in the current study who scored at or below the 15th percentile on the average score on both the SWE and PDE were classified with dyslexia. For further details, see Chapter 1.

Children were classified as having ADHD and AD using the Conners-3 (Conners, 2008) parent and teacher reports. Children with average (parent and teacher) T-scores exceeding 64 on each the Attention and Hyperactivity scales, were classified with ADHD, and children with scores exceeding 64 on the Attention subscale were classified with AD. The clinical cut-off was a T-score ≥ 65; subclinical 60 – 64; and average range ≤ 59. These guidelines have been determined by Conners (2008). For further details, see Chapter 3.

Specific Language impairment (SLI) was assessed using four subtests from the CELF-IV (Semel et al., 2003). Receptive language was assessed with Concepts and Directions and Word Classes 2. Expressive language was assessed with Recalling Sentences and Formulated Sentences. The Core Language score was used to diagnose the presence or
absence of SLI and was derived by summing the scaled scores from the four subtests. The clinical cut-off was ≤ 85; subclinical 86 - 89; and average ≥ 90. These cut-offs are based on the authors’ guidelines.

**General Cognitive Ability**

The Picture Concepts and Matrix Reasoning subtests from the WISC-IV (Weschler, 2003) were used as measures of non-verbal intellectual ability and to estimate general cognitive ability. The scaled scores are combined to form a nonverbal fluid reasoning index (Flanagan & Kaufman, 2009). The clinical cut-off was a standard score ≤ 85, which is based on the authors’ guidelines.

**Family Reading History**

The Adult Reading Questionnaire (ARQ) and the short-form of the Adult ADHD Self-Report Scale (ASRS: Snowling et al., 2012) were used to assess reading and attention impairment, and hyperactivity in the parent sample. The ARQ consists of 15 items: 7 items require the respondent to rate symptoms of dyslexia on a scale of 0 - 4, such as difficulties with literacy skills, word finding and organization (e.g., “do you find it difficult to read words you haven’t seen before?” Never/Rarely/Sometimes/Frequently/Always). Another 2 items require a yes/no/maybe response (e.g., “Are you a good reader?”), and a further 2 items require the respondent to rate their reading and writing frequency (e.g., “How often do you write in everyday life?”). In addition, the questionnaire contains the following definition: Dyslexia is difficulty with reading and writing in people who a) do OK in other aspects of life (so their difficulty is mostly with reading and writing), and b) have had the chance to learn to read but have not been able to learn like others. Following this, the final 4 items specifically ask about a dyslexia diagnosis (e.g., “Based on this, do you think you are dyslexic?” yes/no/maybe).
Items on the ARQ are scored numerically, with higher scores associated with more severe difficulty or greater likelihood of impairment. Item scores ranged from 0 - 1 or 0 - 4, depending on the question, with a possible range of scores from 0 – 43 for the full scale (15 items). This study used the same 11 items as Snowling and colleagues (2012), which had a possible range of 0 – 37. In the original development of the scale, two reading factors were generated. These were difficulty with reading ($\alpha = 0.81$) and difficulty with word finding ($\alpha = 0.60$). While the internal consistency of the difficulty with reading factor was good, the internal consistency of the word finding factor was poor. The validity of the ARQ is good and shows strong concurrent relationships with measured literacy skills, particularly decoding fluency and spelling, and reports of the presence or absence of poor reading skills are well discriminated by responses to the reading and word finding factors on the scale (Snowling et al., 2012).

The complete ASRS scale was developed in conjunction with the revision of the World Health Organisation (WHO) Composite International Diagnostic Interview (CIDI) (Kessler & Ustun, 2004) for the WHO World Mental Health (WMH) Survey Initiative (Demyttenaere et al., 2004). The short form 6-item screener in its original conceptualisation has better reliability than the original 18-item scale (Kessler et al., 2005). In this study, ADHD symptoms were measured using the short-form screener of the ASRS, consisting of the six items that correspond to the presentation of ADHD symptoms (in accordance with the Diagnostic and Statistical Manual of Mental Disorders Fourth edition: DSM-IV) in adults (Adler et al., 2006; Kessler et al., 2005). In the analysis conducted, the attention difficulties scale included each of the 4 items that tap difficulties with sustained attention, organization and prospective memory from the ASRS, and an additional item from the ARQ regarding difficulties with organisation.
The reliability of the attention factor from the original scale is reported to be good (\( \alpha = 0.81 \)).

Finally, the 2-item hyperactivity scale was the same as that used in the ASRS. The reliability of the hyperactivity scale is reported to be modest (\( \alpha = 0.58 \)).

When using the ASRS alone, each of the items is rated on a five-point Likert scale with possible responses ranging from 0 (Never) to 4 (Very Often). A summary score with a theoretical range of 0 - 24 is obtained as an equally weighted sum of response scores for all questions, with higher scores indicating an increased risk of ADHD. The complete 6-item ASRS screener has good sensitivity (68.7%), specificity (99.5%), as well as total classification (97.9%) accuracy (Kessler et al., 2005). The sensitivity and specificity of the modified scale has not been evaluated.

In previous studies that have used the ARQ (e.g., Leavett et al., 2014; Snowling et al., 2012), a report of probable dyslexia was used to distinguish potential dyslexia from no dyslexia among the parents in the sample. Consistent with Snowling and colleagues (2012) a self-report of probable dyslexia was used as an indication of a positive history of reading disorders (ARQ15). To assess ADHD symptom dimensions, we calculated a total ADHD score using a cut-off of 13. Inattention (cut-off 8) and hyperactivity (cut-off 4) item scores were calculated separately.

4.5 Procedure

Parents were asked to complete an ARQ/ASRS questionnaire for themselves, and a Conners-3 parent-report for their child. Consenting teachers were asked to complete the Conners-3 Teacher report. The full details of this process were provided in Chapter 2. With regards to the current study, the order of administration of the relevant measures to the children were as follows: the WISC-IV subtests (matrix reasoning and picture concepts),
the CELF-IV subtests (Concepts and Following Directions, Recalling Sentences, Formulated Sentences, and Word Classes 2), and the TOWRE-2 (Sight Word Efficiency and Phonemic Decoding).

**Analysis strategy**

Confirmatory factor analysis (CFA) models were generated to evaluate the factor structure of the ARQ. Correlation, regression, logistic regression analyses and odds ratios were used to generate statistics of the influence of reports of reading and attention difficulties in parents on child reading and attention difficulties.

### 4.6 Results

**Parent-reports of symptoms of reading difficulty and ADHD**

The descriptive statistics for the items consistent with (Snowling et al., 2012) and used in the Confirmatory Factor Analysis (CFA) are shown in Table 4.1, which includes only the 11 items used to replicate the study by Snowling et al. (2012). While mean scores are slightly lower than those reported by Snowling et al. (2012), all items have a good range of scores, and most span the minimum to maximum possible responses. The correlations between the reading and ADHD items are shown in Table 4.2. Consistent with the findings of Snowling and colleagues, strong positive associations were found between each of the items used to assess reading (ARQ1, ARQ2, ARQ3, ARQ5, ARQ6), and word finding difficulties (ARQ7, ARQ8, ARQ9). The five items used to assess attentional difficulties (ARQ10, and ASRS 1-4) were also highly correlated, with a moderate positive correlation found between the two items (ASRS5, ASRS6) used to assess hyperactivity. Although a smaller sample was used in the current study, these associations are remarkably consistent with those reported by Snowling and colleagues (2012).
Table 4.1

Descriptive statistics for the items on the Adult Reading Questionnaire and ASRS (N = 117)

<table>
<thead>
<tr>
<th>Code</th>
<th>Item</th>
<th>Mean (SD)</th>
<th>Min, Max</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td><strong>Adult Reading Questionnaire</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ARQ1</td>
<td>Do you think you are a good reader?</td>
<td>0.15 (0.34)</td>
<td>0, 1</td>
</tr>
<tr>
<td>ARQ2</td>
<td>Can you read quickly and easily?</td>
<td>0.17 (0.37)</td>
<td>0, 1</td>
</tr>
<tr>
<td>ARQ3</td>
<td>How good is your spelling?</td>
<td>0.66 (0.79)</td>
<td>0, 3</td>
</tr>
<tr>
<td>ARQ4</td>
<td>In your job, how often do you read?</td>
<td>0.88 (0.88)</td>
<td>0, 4</td>
</tr>
<tr>
<td>ARQ5</td>
<td>Do you find it difficult to read words you have not seen before?</td>
<td>1.21 (0.94)</td>
<td>0, 4</td>
</tr>
<tr>
<td>ARQ6</td>
<td>Do you find it difficult to read aloud?</td>
<td>0.68 (0.95)</td>
<td>0, 4</td>
</tr>
<tr>
<td>ARQ7</td>
<td>Do you find it difficult to find the right word to say?</td>
<td>1.21 (0.84)</td>
<td>0, 4</td>
</tr>
<tr>
<td>ARQ8</td>
<td>Do you ever confuse the name of things?</td>
<td>1.10 (0.83)</td>
<td>0, 4</td>
</tr>
<tr>
<td>ARQ9</td>
<td>Do you confuse left and right?</td>
<td>0.73 (0.95)</td>
<td>0, 4</td>
</tr>
<tr>
<td>ARQ10</td>
<td>Do you have problems with organisation and time management?</td>
<td>1.03 (1.00)</td>
<td>0, 4</td>
</tr>
<tr>
<td>ARQ11</td>
<td>How often do you write in everyday life?</td>
<td>1.10 (1.10)</td>
<td>0, 4</td>
</tr>
<tr>
<td></td>
<td><strong>ADHD Self-Report Screener</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASRS1</td>
<td>How often do you have difficulties wrapping the fine details of a project, once the challenging parts have been done?</td>
<td>1.16 (1.09)</td>
<td>0, 4</td>
</tr>
<tr>
<td>ASRS2</td>
<td>How often do you have difficulty getting things in order when you have to do a task that requires organisation?</td>
<td>0.96 (0.86)</td>
<td>0, 4</td>
</tr>
<tr>
<td>ASRS3</td>
<td>How often do you have difficulties remembering appointments or obligations?</td>
<td>0.97 (0.89)</td>
<td>0, 4</td>
</tr>
<tr>
<td>ASRS4</td>
<td>How often do you fidget or squirm with your hands or feet when you have to sit down for a long period?</td>
<td>1.40 (1.11)</td>
<td>0, 4</td>
</tr>
<tr>
<td>ASRS5</td>
<td>How often do you feel overly active and compelled to do things, like you were driven by a motor?</td>
<td>1.43 (0.97)</td>
<td>0, 4</td>
</tr>
</tbody>
</table>
Table 4.2

*Correlations of test items from the Adult Reading Questionnaire and the ASRS (N = 117)*

<table>
<thead>
<tr>
<th>Variables ARQ1</th>
<th>ARQ2</th>
<th>ARQ3</th>
<th>ARQ5</th>
<th>ARQ6</th>
<th>ARQ7</th>
<th>ARQ8</th>
<th>ARQ9</th>
<th>ARQ10</th>
<th>ASRS1</th>
<th>ASRS2</th>
<th>ASRS3</th>
<th>ASRS4</th>
<th>ASRS5</th>
</tr>
</thead>
<tbody>
<tr>
<td>ARQ2</td>
<td>0.86</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ARQ3</td>
<td>0.56</td>
<td>0.62</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ARQ5</td>
<td>0.47</td>
<td>0.55</td>
<td>0.60</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ARQ6</td>
<td>0.68</td>
<td>0.73</td>
<td>0.58</td>
<td>0.57</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ARQ7</td>
<td>0.38</td>
<td>0.30</td>
<td>0.25</td>
<td>0.32</td>
<td>0.36</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ARQ8</td>
<td>0.38</td>
<td>0.29</td>
<td>0.28</td>
<td>0.34</td>
<td>0.38</td>
<td>0.56</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>ARQ9</td>
<td>0.26</td>
<td>0.19</td>
<td>0.24</td>
<td>0.22</td>
<td>0.20</td>
<td>0.47</td>
<td>0.54</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ARQ10</td>
<td>0.17</td>
<td>0.15</td>
<td>0.26</td>
<td>0.19</td>
<td>0.24</td>
<td>0.33</td>
<td>0.17</td>
<td>0.09</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASRS1</td>
<td>0.47</td>
<td>0.43</td>
<td>0.40</td>
<td>0.28</td>
<td>0.32</td>
<td>0.46</td>
<td>0.40</td>
<td>0.33</td>
<td>0.58</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASRS2</td>
<td>0.27</td>
<td>0.21</td>
<td>0.31</td>
<td>0.28</td>
<td>0.30</td>
<td>0.39</td>
<td>0.33</td>
<td>0.21</td>
<td>0.69</td>
<td>0.75</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASRS3</td>
<td>0.22</td>
<td>0.22</td>
<td>0.26</td>
<td>0.16</td>
<td>0.36</td>
<td>0.33</td>
<td>0.26</td>
<td>0.06</td>
<td>0.46</td>
<td>0.41</td>
<td>0.44</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>ASRS4</td>
<td>0.27</td>
<td>0.25</td>
<td>0.26</td>
<td>0.15</td>
<td>0.32</td>
<td>0.50</td>
<td>0.34</td>
<td>0.27</td>
<td>0.62</td>
<td>0.65</td>
<td>0.61</td>
<td>0.46</td>
<td>-</td>
</tr>
<tr>
<td>ASRS5</td>
<td>0.32</td>
<td>0.28</td>
<td>0.35</td>
<td>0.29</td>
<td>0.30</td>
<td>0.36</td>
<td>0.30</td>
<td>0.08</td>
<td>0.29</td>
<td>0.40</td>
<td>0.31</td>
<td>0.29</td>
<td>0.41</td>
</tr>
<tr>
<td>ASRS6</td>
<td>0.18</td>
<td>0.16</td>
<td><strong>0.18</strong></td>
<td>0.10</td>
<td>0.15</td>
<td>0.06</td>
<td>0.15</td>
<td>0.13</td>
<td>0.01</td>
<td><strong>0.27</strong></td>
<td>0.14</td>
<td>0.11</td>
<td>0.14</td>
</tr>
</tbody>
</table>

*Note.* Significant correlations are in bold.

ARQ questions 1, 4 & 11 were excluded as per the factor structure validated by Snowling et al., (2012)
Confirmatory factor analysis (CFA)

Using these data, a CFA was conducted to reproduce the same four factor solution (reading, word finding, attention and hyperactivity) produced by Snowling et al. (2012). The model was generated using a maximum likelihood estimation procedure. Model fit was assessed using the $\chi^2$ statistic to test the absolute fit of the model. In the current study a $\chi^2$ value two to three times greater than the degrees of freedom is acceptable (Carmines & McIver, 1981). The Comparative Fit Index (CFI) was used as a measure of incremental fit and contrasted the model generated with a model in which all parameters were independent. The $\chi^2$ and comparative fit indices compare the specified model to one with complete independence: a non-significant $\chi^2$ and CFI values greater than .9 to .95 reflect a good model fit. The Root Mean Square Error of Approximation (RMSEA; Byrne & Van de Vijver, 2010) estimates error due to the approximate fit of the model: values below .08 reflect a good model fit (Jackson & Gillespy, 2009). The fit of the model attempting to replicate the four-factor structure general by Snowling et al. (2012) was poor, $\chi^2 (83) = 148.86, p < .001, \chi^2/df = 1.79, GFI = .86, CFI = .93, RMSEA = .083, PCLOSE = .009$, so two separate models were produced; one for the two-factor reading measures and a second using the two-factor model of ADHD symptoms originally proposed by Kessler et al. (2005).

Parent self-reported symptoms of reading difficulties and ADHD

The fit of the two-factor model for the adult reading questionnaire using the items from the two-factor reading component of the model was satisfactory, $\chi^2(17) = 19.80, p = .258, \chi^2/df = 1.16, GFI = .96, CFI = .99, RMSEA = .038, PCLOSE = .575$. The model coefficients (see Figure 4.1) ranged between 0.64 and 0.85, with a moderate correlation found between the factors. The internal consistency of both factors was acceptable, Reading, $\alpha = .83$ and word finding, $\alpha = .76$. This model is consistent with the reading components of the original model (Snowling et al., 2012).
Figure 4.1  Factor structure from the CFA, for the reading and word finding factors ($N = 117$).

The second two-factor model that was generated using the original attention and hyperactivity items from the short form of the ASRS to assess ADHD was satisfactory $\chi^2(8) = 12.89, p = .115, \chi^2/df = 1.61, GFI = .96, CFI = .98, RMSEA = .073, PCLOSE = .264$. The model coefficients (see Figure 4.2) ranged between 0.50 to 0.88, and are consistent with those found by Snowling et al. (2012). The only difference between this model and the model initially produced by Snowling and colleagues was the exclusion of item ARQ10 (“Do you have problems with organisation and time management?”) from the ARQ. Although there are strong associations between this item and those for the attention factor, inclusion of the item produced a poor model fit. The internal consistency of the attention scale ($\alpha = .83$) was high but the internal consistency of the two-item hyperactivity scale, ($\alpha = .62$) was poor.
Figure 4.2  Factor structure from the CFA, for the attention and hyperactivity factors ($N = 117$).

**Associations between factors and indicator variables**

On each of the four scales (reading, word finding, attention and hyperactivity), the higher the score the more the reported difficulties (see Table 4.3). Moderate positive associations were found between the four factors, with higher scores associated with reports of increased reading or attention difficulties, and confirm previous reports of the association between reading and attention difficulties (Ebejer et al., 2010; Greven et al., 2011; Paloyelis et al., 2010; Plourde et al., 2015; Willcutt et al., 2000; Willcutt, Pennington, et al., 2007). There were no significant associations between reading frequency and the reading and attention factors. Less writing in everyday life was associated with reports of greater reading difficulties and higher scores on the reading factor. The associations found for the frequency of reading and writing in everyday life are similar to those reported previously (Snowling et al., 2012).
Table 4.3

Pearson Correlations showing associations between reading and attention measures, rated difficulty in reading, and frequency of reading and writing on an everyday basis.

<table>
<thead>
<tr>
<th></th>
<th>Mean (SD)</th>
<th>Read</th>
<th>Words</th>
<th>Attention</th>
<th>Hyper</th>
<th>Difficulty</th>
<th>F. Read</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reading Scale</td>
<td>2.88 (2.83)</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Word finding</td>
<td>3.03 (2.16)</td>
<td>.42**</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Attention</td>
<td>4.39 (310)</td>
<td>.42**</td>
<td>.48**</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hyperactivity</td>
<td>2.83 (1.77)</td>
<td>.33**</td>
<td>.26**</td>
<td>.38**</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rated difficulty Read</td>
<td>0.41 (0.67)</td>
<td>.64**</td>
<td>.41**</td>
<td>.58**</td>
<td>.26</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frequency read</td>
<td>0.88 (0.88)</td>
<td>.18</td>
<td>.11</td>
<td>.13</td>
<td>.12</td>
<td>.10</td>
<td>-</td>
</tr>
<tr>
<td>Frequency Write</td>
<td>1.10 (1.08)</td>
<td>.34**</td>
<td>.01</td>
<td>.29**</td>
<td>.11</td>
<td>.24**</td>
<td>.60**</td>
</tr>
</tbody>
</table>

Note: ** p < .01

To determine how well scores on the two reading factors discriminated parental self-report of potential reading difficulties, adults reporting “yes”, or “maybe” to the item “Do you think you are dyslexic” were grouped into a reading difficulty group (n = 31). Those who responded no to this item were classified as those with no reading difficulties (n = 86). Independent samples t-tests were conducted to determine whether those reporting that they either had, or potentially had dyslexia, were discriminated from those reporting no reading difficulties, on the four factors produced in the CFA’s (see Table 4.4). The group reporting potential reading difficulties had significantly higher scores than those without difficulties on the total reading measure, t(115) = 13.07, p < .001, Cohen’s d = 2.46, the word finding measure, t(115) = 4.98, p < .001, Cohen’s d = 0.96, and the attention t(115) = 6.30, p < .001, Cohen’s d = 1.23, and hyperactivity scales, t(115) = 4.51, p < .001, Cohen’s d = 0.97. These results are consistent with those reported by Snowling et al. (2012) for the same item on the
ARQ and indicate that not only reading but attention difficulties are associated with perceived poor reading performance.

Table 4.4

*Differences between groups on the four factors with and without potentially reported dyslexia*

<table>
<thead>
<tr>
<th></th>
<th>Potential Dyslexia (n = 31)</th>
<th>No dyslexia (n = 86)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
</tr>
<tr>
<td>Reading score (/13)</td>
<td>6.50 (2.4)</td>
<td>1.57 (1.5)</td>
</tr>
<tr>
<td>Word finding (/12)</td>
<td>4.55 (2.5)</td>
<td>2.49 (1.7)</td>
</tr>
<tr>
<td>Attention (/16)</td>
<td>7.00 (3.3)</td>
<td>3.45 (2.4)</td>
</tr>
<tr>
<td>Hyperactivity (/8)</td>
<td>3.97 (1.5)</td>
<td>2.42 (1.7)</td>
</tr>
</tbody>
</table>

To determine how well the single item reported the potential presence or absence of parental dyslexia, a logistic regression analysis was conducted with the two reading factors from the CFA, word reading and word finding difficulties entered simultaneously. The assumptions of the analysis were met. Consistent with previous scales that have obtained index scores of parental reading history (e.g., Bonifacci, Montuschi, Lami, & Snowling, 2014; Conlon et al., 2006; Pennington & Lefly, 2001), the probability of the presence of dyslexia was set at 0.30. Scores on the reading factor were positive predictors of reading status, with higher scores indicating a greater likelihood of the potential presence of dyslexia, $\chi^2(1) = 25.66, p < .001, \text{OR} = 2.68$ (95% CI’s 1.83 to 3.92). A significant unique contribution to the outcome was not made by word finding difficulties, $\chi^2(1) = 3.35, p = .067, \text{OR} = 1.38$ (95% CI’s: 0.98 to 1.95). Sensitivity (95.2%) and specificity (87.1%) were high (see Table 4.5). Misclassifications appeared to occur because participants either failed to report reading difficulties, despite reporting the potential presence of dyslexia, or participants reported substantial symptoms of dyslexia, yet reported no potential experience of dyslexia.
Table 4.5

Classification of participants using logistic regression analysis

<table>
<thead>
<tr>
<th>Classified</th>
<th>No dyslexia</th>
<th>Dyslexia</th>
<th>% Correct</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dyslexia</td>
<td>4</td>
<td>27</td>
<td>87.1%</td>
</tr>
<tr>
<td>No dyslexia</td>
<td>82</td>
<td>4</td>
<td>95.3%</td>
</tr>
</tbody>
</table>

Finally, we determined the extent to which parent gender influenced reports of potential dyslexia in the parent sample. The odds ratio generated indicated that fathers were 3.02 (95% CI’s: 1.25 to 7.26) times more likely than mothers to report the potential presence of dyslexia, $\chi^2(1) = 6.36, p = .012$.

**Attention Deficit and Attention Deficit Hyperactivity Disorder classification**

Based on the screening instrument, several different techniques have been introduced to determine a scoring criterion to establish whether adults potentially have difficulties with AD or ADHD. First, using the six items from the ASRS, a response of sometimes to very often on the first three items (ASRS1, ASRS2, ASRS3) is regarded as a positive indicator of the presence of the symptom. For the remaining three items (ASRS4, ASRS5, ASRS6) a response of often, or very often are considered positive indicators of the presence of the symptom. Using this criterion, there were 11 (9.4%) parents (5 mothers, 6 fathers) who reported positive indicators of ADHD. When a total score of 13 or greater (Kessler et al., 2007) was used for the ASRS, there were 14 (12%) parents (6 mothers, 8 fathers) who reported positive symptoms of ADHD. When symptoms of AD only were evaluated, using scores of 8 or greater for the four attention items from Factor 1 of the CFA, there were 15 (12.8%) parents (6 mothers, 9 fathers), with self-identified symptoms of inattention. The overlap between the AD and ADHD groups was 85%. Regardless of the criterion used, there
was no significant association between gender and classification for either combined ADHD, \(\chi^2(1, N = 117) = 0.90, p = .343\) or inattention symptoms only, \(\chi^2(1, N = 117) = 1.50, p = .221\).

T-tests were conducted to determine if the presence or absence of probable AD and ADHD showed differences on the reading and word finding measures of the ARQ (see Table 4.6). The group reporting potential ADHD had significantly higher scores on both the total reading, \(t(115) = 3.74, p < .001\), Cohen’s \(d = 0.98\), and word finding measures, \(t(115) = 4.47, p < .001\), Cohen’s \(d = 1.31\), than those without ADHD. The group with potential AD also reported significantly more problems with word reading, \(t(115) = 3.30, p = .001\), Cohen’s \(d = 0.81\), and word finding difficulties, \(t(115) = 4.3, p < .001\), Cohen’s \(d = 1.23\) than those not reporting problems with AD. These findings support the hypothesis of the association between attention difficulties and reading difficulties.

Table 4.6

Descriptive statistics of the groups with and without potential ADHD and AD on the reading and word finding factors from the Adult Reading Questionnaire.

<table>
<thead>
<tr>
<th></th>
<th>Potential ADHD (n = 14)</th>
<th>No ADHD (n = 103)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
</tr>
<tr>
<td>Reading score (/13)</td>
<td>5.39 (3.2)</td>
<td>2.53 (2.6)</td>
</tr>
<tr>
<td>Word finding (/12)</td>
<td>5.29 (1.9)</td>
<td>2.73 (2.0)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Potential AD (n = 15)</td>
<td>No AD (n = 102)</td>
</tr>
<tr>
<td>Reading score (/13)</td>
<td>5.03 (3.4)</td>
<td>2.56 (2.6)</td>
</tr>
<tr>
<td>Word finding (/12)</td>
<td>5.13 (1.9)</td>
<td>2.72 (2.0)</td>
</tr>
</tbody>
</table>

Finally, the association between reported symptoms of ADHD and AD, and potential reading difficulties were investigated. Parents reporting positive symptoms of ADHD were
15.22 times (95% CI’s 3.87 to 56.7) more likely to report the potential presence of dyslexia, 
$\chi^2(1, N = 117) = 22.14, p < .001$. Parents reporting positive symptoms of AD were 11.27
times (95% CI’s 3.25 to 39.13) more likely to report the potential presence of dyslexia, $\chi^2(1, N = 117) = 19.38, p < .001$. Of the 31 parents who reported potential dyslexia, there were 11
(35.5%) who also reported potential ADHD. Of the parents who reported no potential
reading difficulties (83), three reported potential problems with ADHD. The pattern was
similar if the AD symptoms only were used, however there was one additional parent without
symptoms of dyslexia who reported potential AD.

One-way ANOVA was conducted using the group with potential dyslexia + ADHD,
dyslexia alone, and a control group of parents who did not report either disorder. The three
parents with self-reported ADHD were excluded from the analysis because of the small
sample size. However, the means and 95% confidence intervals for this group were reported
for comparison. The four factor scores for the ARQ and the ASRS were each used as the DV
(see Table 4.7). Significant group effects were found for each variable. For the attention,
hyperactivity and word finding scales, the group with dyslexia + ADHD had significantly
higher scores than the group with dyslexia only, who had significantly higher scores than the
control group (all $p$’s < .05). For the word reading scale, the control group had significantly
lower scores than the two groups with dyslexia, who did not differ. Inspection of the scores
for the ADHD only group indicates similar scores to the control group for the reading scale,
but scores were similar to the other disorder groups for the other three measures. Overall,
these findings continue to provide support for the hypothesis of greater attentional difficulties
among individuals with reading difficulties.
Influence of parent-reports of symptoms of dyslexia and ADHD on child diagnosis of developmental disorders.

The prevalence of developmental disorders in the sample of 72 children included in the current study was reported in Chapter 3. Of these children, there were 64 for whom at least one parent completed the Adult Reading Questionnaire. These children were included in the following analyses.

Table 4.7

Descriptive statistics for parent groups: control, dyslexia, dyslexia + ADHD and ADHD for the ARQ and ASRS factors.

<table>
<thead>
<tr>
<th>Group</th>
<th>Control (n = 83)</th>
<th>Dyslexia + ADHD (n = 11)</th>
<th>Dyslexia (n = 20)</th>
<th>ADHD (n = 3)</th>
<th>ANOVA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Reading Mean, (95% CI)</td>
<td>Word Finding Mean, (95% CI)</td>
<td>Attention Mean, (95% CI)</td>
<td>Hyperactivity Mean, (95% CI)</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>1.78 (1.24 - 1.91)</td>
<td>2.46 (2.07 - 2.83)</td>
<td>3.21 (2.75 - 3.67)</td>
<td>2.34 (1.98 - 2.70)</td>
<td></td>
</tr>
<tr>
<td>Dyslexia + ADHD</td>
<td>6.50 (4.72 - 8.28)</td>
<td>5.82 (4.70 - 6.93)</td>
<td>10.73 (9.56 - 11.89)</td>
<td>4.91 (3.73 - 6.09)</td>
<td></td>
</tr>
<tr>
<td>Dyslexia</td>
<td>6.50 (5.40 - 7.60)</td>
<td>3.85 (2.62 - 5.08)</td>
<td>4.95 (4.10 - 5.80)</td>
<td>3.45 (2.95 - 3.94)</td>
<td></td>
</tr>
<tr>
<td>ADHD</td>
<td>1.33 (.10 - 2.77)</td>
<td>3.33 (.46 - 7.13)</td>
<td>10.0 (7.52 - 12.48)</td>
<td>4.67 (1.80 - 7.53)</td>
<td></td>
</tr>
<tr>
<td>ANOVA</td>
<td>F (2, 111) = 81.62, p &lt; .001</td>
<td>F (2, 111) = 16.96, p &lt; .001</td>
<td>F (2, 111) = 67.96, p &lt; .001</td>
<td>F (2, 111) = 15.80, p &lt; .001</td>
<td></td>
</tr>
</tbody>
</table>

Influence of parent-reports of dyslexia on the prevalence of dyslexia and other developmental disorders in the sample.

A parental report of potential dyslexia from the ARQ was the criterion used to determine the potential presence of family risk of dyslexia. Using this criterion there were 31 parents (26.5%) who reported positive symptoms of dyslexia. The association between
parent-report and gender was investigated within families, with no significant association found, $\chi^2(4, N = 53) = 0.649, p = .461$.

There were four families where both parents reported potential dyslexia; in 16 families the father reported potential dyslexia, and in 7 families the mother reported potential dyslexia. Based on these results, there were a total of 27 children from a family with one or both parents reporting reading difficulties. A single variable that included the positive family history in either parent was generated to investigate the associations between family report of dyslexia and diagnoses of dyslexia, ADHD, AD, and multiple disorders including language in these children. From the 27 children with family reported dyslexia, there were 19 (70.4%) children with scores in the clinical range for dyslexia. From the 37 children in the group with no family history of dyslexia, there were 16 children (43.2%) identified with clinical symptoms of dyslexia. Children from a family with parent-reported reading difficulties were 3.17 times (95% CI OR: 1.10 to 8.92) more likely to have clinical symptoms of dyslexia than children coming from a family without parental report of reading difficulties, $\chi^2(1, N = 64) = 4.73, p = .030$, providing support for the hypothesis that dyslexia would be most prevalent in children at family risk.

When the prevalence of dyslexia, ADHD, language disorders and the presence of multiple disorders was evaluated based on a family history of dyslexia (see Table 4.8), there were only a small proportion of children in the control group with a parental history of reading difficulties. There was little difference in the proportion of children in either the groups with dyslexia alone or ADHD alone with or without a parent with dyslexia. However, children with multiple deficits (dyslexia + ADHD or language impairment + dyslexia or ADHD) were more likely to have a parent with self-reported reading difficulties, although this association was not statistically significant, $\chi^2 (4, N = 64) = 7.82, p = .098$. 
Table 4.8

*Number and percentage of children in the control, dyslexia, ADHD, dyslexia + ADHD and language groups with a mother, father or both mother and father with symptoms of dyslexia.*

<table>
<thead>
<tr>
<th></th>
<th>Control (n = 17)</th>
<th>Dyslexia (n = 21)</th>
<th>Dyslexia + ADHD (n = 8)</th>
<th>ADHD (n = 12)</th>
<th>Language + (n = 6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No Parental Dyslexia</td>
<td>13 (76.5%)</td>
<td>12 (57.1%)</td>
<td>2 (25%)</td>
<td>8 (66.7%)</td>
<td>2 (33.3%)</td>
</tr>
<tr>
<td>Family History</td>
<td>4 (23.5%)</td>
<td>9 (42.9%)</td>
<td>6 (75%)</td>
<td>4 (33.3%)</td>
<td>4 (66.7%)</td>
</tr>
<tr>
<td>Father History</td>
<td>4</td>
<td>7</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Mother History*</td>
<td>2</td>
<td>2</td>
<td>3</td>
<td>2</td>
<td>2</td>
</tr>
</tbody>
</table>

*In the control group there were two cases in which both parents reported potential dyslexia. There was also one case in the ADHD and language groups where both parents reported potential dyslexia.

The same analysis was repeated using reports of AD, not ADHD (see Table 4.9). No significant association was found between reports of AD and a parent-report of reading difficulties, or other developmental disorders, $\chi^2 (4, N = 64) = 4.74, p = .315$.

Table 4.9

*Number and percentage of children in the control, dyslexia, AD, dyslexia + AD and language groups with a mother, father or both mother and father with symptoms of dyslexia*

<table>
<thead>
<tr>
<th></th>
<th>Control (n = 11)</th>
<th>Dyslexia (n = 8)</th>
<th>Dyslexia + AD (n = 21)</th>
<th>AD (n = 19)</th>
<th>Language + (n = 5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No Parental Dyslexia</td>
<td>8 (72.7%)</td>
<td>4 (50.0%)</td>
<td>10 (47.6%)</td>
<td>13 (68.4%)</td>
<td>2 (40%)</td>
</tr>
<tr>
<td>Family History</td>
<td>3 (27.3%)</td>
<td>4 (50.0%)</td>
<td>11 (52.4%)</td>
<td>6 (33.3%)</td>
<td>3 (60.0%)</td>
</tr>
<tr>
<td>Father History</td>
<td>3</td>
<td>4</td>
<td>6</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>Mother History</td>
<td>2</td>
<td>0</td>
<td>5</td>
<td>3</td>
<td>1</td>
</tr>
</tbody>
</table>

*In the control group there were two cases in which both parents reported potential dyslexia. There was also one case in the group with AD and language group where both parents reported potential dyslexia.
ADHD

There were 64 children in the sample with at least one parent responding to the ASRS. Using the criterion of a total score of 13 or more on the scale, there were 14 (21.9%) with a family history of ADHD. Of these children 6 children (42.9%) satisfied the criteria for a clinical diagnosis of ADHD, while 8 (57.1%) did not, $\chi^2(1, N = 64) = 1.08, p = .298$. Further, parent-report of ADHD symptoms was not associated with the prevalence of dyslexia, ADHD or multiple deficits in these children, $\chi^2(1, N = 64) = 1.08, p = .289$ (see Table 4.10). When sub-clinical levels of ADHD were evaluated, a significant association was found between parent-report of ADHD and child diagnoses, $\chi^2(1, N = 64) = 9.51, p = .050$. Those children whose parents reported symptoms of ADHD were more likely to have symptoms of ADHD at a clinical or subclinical level than children coming from a family without parental report of ADHD.

Table 4.10

*Separate findings for mothers and fathers were not reported because of the small numbers included.*
**AD only**

Using a cut-off ≥ 8 on the ASRS inattention items, there were 14 (21.9%) parents (6 mothers, 9 fathers) who identified with self-reported symptoms of inattention. Of the 42 children with clinical level symptoms of AD, there were 8 (19%) children from families reporting AD symptoms. The remaining 34 (81%) children had no family history of significant AD symptoms. No significant association was found between parent-report of AD and child diagnosis, $\chi^2(1, N = 64) = 0.56, p = .455$. The results do not support the prediction that AD would be most prevalent in children at family risk of the disorder.

The influence of parent AD was evaluated across dyslexia, AD, AD + Dyslexia and language impairments (see Table 4.11). Reports of high parental symptoms of AD, had no influence either on the diagnosis of clinical AD in children or for other developmental disorders, $\chi^2(4, N = 64) = 1.910, p = .752$.

Table 4.11

<table>
<thead>
<tr>
<th></th>
<th>Control $(n = 11)$</th>
<th>Dyslexia $(n = 8)$</th>
<th>Dyslexia + AD $(n = 21)$</th>
<th>AD $(n = 19)$</th>
<th>Language + $(n = 5)$</th>
</tr>
</thead>
<tbody>
<tr>
<td>No Parent AD</td>
<td>8 (72.7%)</td>
<td>6 (75%)</td>
<td>18 (85.7%)</td>
<td>15 (78.9%)</td>
<td>3 (60%)</td>
</tr>
<tr>
<td>Parent AD</td>
<td>3 (27.3%)</td>
<td>2 (25%)</td>
<td>3 (14.3%)</td>
<td>4 (21.4%)</td>
<td>2 (40%)</td>
</tr>
</tbody>
</table>

**Potential family dyslexia + ADHD**

When the distribution of parent-reported dyslexia, ADHD, and dyslexia + ADHD was examined for the children in the sample, there were 16 (25%) parents, reporting dyslexia, 11 (17.2%) reporting possible dyslexia + ADHD, with only 3 (4.7%) reporting ADHD alone. For the group with dyslexia there were 14/16 (87.5%) of children showing evidence of one or more disorders. The same result was found for parent-report of dyslexia + ADHD, with 9/11
(81.8%) of children being diagnosed with one or more disorder. There is no evidence to indicate that the presence of more than one parental disorder increased the likelihood of one or more disorders in their child.

4.7 Discussion

The main aim of the present study was to examine the factor structure of the ARQ/ASRS, and validate the measure using the current sample of parents. We were unable to produce the same single scale as Snowling et al. (2012), however the factor structure of the ARQ and ASRS (without the organisation item from the ARQ) were replicated in two separate scales. These were consistent with the two separate factors produced with the ARQ (Snowling et al., 2012), and the ASRS (Kessler et al., 2005). While the mean scores were slightly lower in the current study, all items displayed a good range of scores and most spanned the minimum to maximum possible responses. The only difference between the model in the current study and that of Snowling and colleagues was the exclusion of the item relating to problems with organisation and time management.

Using the ARQ/ASRS the mothers and fathers of participating children rated their own reading, attention and hyperactivity behaviours. The results for the ARQ indicated that parents reporting potential reading difficulties had significantly higher scores on the total reading measure than parents without difficulties, and these scores were positive predictors of reading status. This is also in accord with evidence that adults have an accurate perception of their abilities (Gimenez et al., 2015), and support the use of the ARQ self-report protocol as a valid continuous measure of literacy skills in adults.

Parents with potential dyslexia, AD or ADHD were also observed to have higher scores on the alternative measures to those on which they were classified. For parents with self-reported symptoms of dyslexia, the greater the reading difficulties reported, the higher the scores on both the reading and attention scales, and of this group 35.5% were more likely
to self-report potential AD or ADHD. Similarly, parents with self-reported AD or ADHD had poorer reading and word finding skills and were 11 to 15 times more likely to report the potential presence of dyslexia than the group without AD or ADHD. These findings support other genetic and behavioural research that has identified an association between reading difficulties and inattention (e.g., Ebejer et al., 2010; Gayán et al., 2005; Greven et al., 2011; Greven et al., 2012; Nigg et al., 2010; Paloyelis et al., 2010; Willcutt, Betjemann, Wadsworth, et al., 2007; Willcutt, Pennington, et al., 2007), and are in accord with Study 1 which identified that individuals with dyslexia, AD or ADHD commonly show clinical or subclinical evidence of the other disorder.

The second aim of the study was to determine the prevalence rates for dyslexia, AD and ADHD in the parent sample. The prevalence of dyslexia in the parent sample was estimated at 26.5%, with fathers being 3 times more likely to report the potential presence of dyslexia than mothers. This finding is in line with other studies that have observed a significant male bias in the affected family members of individuals with dyslexia (van Bergen et al., 2011; Wolff & Melngailis, 1994). Depending on the criteria used, the prevalence of ADHD in the parent sample was between 9.4% and 12%, while the prevalence of AD was 12.8%. Despite some studies finding parent-of-origin effects (e.g., Goos et al., 2007; Hawi et al., 2010; Quist et al., 2003; Smoller et al., 2006; Takeda et al., 2010), our finding is in line with those studies that found no evidence of gender specific effects in the transmission of risk for ADHD (Anney et al., 2008; Kim et al., 2007; Laurin et al., 2007).

Finally, the scores of parents on each of the validated scales were used to determine if parent-reported symptoms of dyslexia, AD or ADHD were associated with the prevalence of single (dyslexia, AD, ADHD, SLI) or multiple disorders in the children assessed in the current study. Based on prior research findings it was expected that children from families reporting high symptomatology of dyslexia, AD or ADHD would be more likely to have
these disorders than children whose parents did not report difficulties in these areas. Using parents’ self-report of dyslexia as a single proxy for FR status, we found that 43.2% of children without FR of dyslexia had scores in the clinical range on the reading measures, compared to 70.4% of children identified to be at FR of dyslexia. These estimates indicate that children from at-risk families were 3.17 times more likely to have clinical symptoms of dyslexia than children from a family without parental report of reading difficulties, and are consistent with recent findings by Snowling and Melby-Larvag (2016) who found that 53% of children at family risk were diagnosed with dyslexia when the cut-off for diagnosis was set above the 10th percentile. However, using a criterion of the 15th percentile in the current study, the prevalence of dyslexia in control samples without family risk was higher than expected, which could be explained by the characteristics of the sample used in the current study.

These findings support the hypothesis that dyslexia would be most prevalent in children at family risk. They are also consistent with Snowling et al. (2012) who found that children at FR were 4 times more likely to be diagnosed with dyslexia. In this study, the number of children without FR who were classified with dyslexia was significantly higher than estimates reported in other research (i.e., 11.6%; Snowling & Melby-Lervåg, 2016). However, this may reflect bias in the current sample, as children were recruited into the study based on prior diagnoses of dyslexia and/or ADHD, as well parental perceptions that their children had difficulties with reading and were not performing as well as could be expected academically. When the prevalence of dyslexia, ADHD, SLI, and the presence of multiple disorders was evaluated based on FR of dyslexia, there was a trend for children with multiple disorders (dyslexia + ADHD, or SLI + dyslexia/ADHD) to be more likely to have a parent with self-reported reading difficulties. This is in accord with research that has found common genetic influences, and high heritability to be involved in dyslexia, ADHD and SLI, with one
another, as well as separately (Greven et al., 2012; Nigg et al., 2010; Paloyelis et al., 2010), as well as genetic studies that have identified common genetic variants that influence language and reading abilities (Eicher et al., 2013; Gialluisi et al., 2014; Luciano et al., 2013; Nudel et al., 2014). Finally, the majority of children in the sample with dyslexia were male, supporting research that suggests that boys are more vulnerable to the disorder than girls (Wolff & Melngailis, 1994). However, there were no significant performance differences found between boys and girls on any task, so this issue is not further discussed.

When parent-reported symptoms of ADHD were evaluated, 42.9% of the children at FR met criteria for a clinical diagnosis, however, more than half (57.1%) of the children at FR showed no symptomatology of the disorder. When subclinical levels of ADHD were also considered, the children of parents with self-rated symptoms were more likely to have clinical or subclinical symptoms than children from a family with no risk of ADHD. These findings only partially support our prediction that ADHD would be more prevalent in children at FR, however, our estimate is in accord with other studies that have found the rates of ADHD to be in the range of 41% - 44% in children with at least one affected parent (Starck et al., 2016; Takeda et al., 2010).

No significant association was found between parent-reported AD symptoms and child diagnosis, as the majority (81%) of children with AD were from families with no reported history of AD symptoms. Moreover, there was no evidence that children with a family history of AD or ADHD were more likely to present with multiple disorders. There are several factors that may have contributed to these findings. For example, the ASRS is a screening tool with 4 items to identify potential inattention, and only 2 items to identify potential hyperactivity. When compared to the greater objectivity of the items used in the ARQ to determine symptoms of dyslexia, the items used to rate inattention and hyperactivity are very subjective and may not be sensitive enough to identify true AD or ADHD. Further,
multiple raters were used to identify children with AD and ADHD, while only self-ratings were used to identify symptoms in the parent sample, and the research literature has found self-ratings of ADHD to be significantly lower when compared to multiple ratings (Brikell et al., 2015). Another explanation comes from recent longitudinal studies that suggest childhood and adult ADHD may be distinct disorders. This proposal has also found support from genetic studies that have observed the risk of adult-onset ADHD emerging in a monozygotic twin is not increased when the co-twin has childhood-onset ADHD (Agnew-Blais et al., 2016). This lack of shared risk between monozygotic twins suggests distinct genetic contributions for child and adult-onset ADHD (Agnew-Blais et al., 2016). Finally, the AD identified in children and/or adults in the current sample, may be the result of normal attention lapses, based on a lack of interest due to reading problems, however this will be further discussed in study 4.

There are several limitations to this study that should be mentioned. This was a small self-selecting community sample, that predominantly focussed on children with dyslexia, and the age range of the sample was very narrow (9 – 11 years of age). Therefore, interpretation of the results should not be generalised to other populations. In addition, parent reading problems were assessed only by self-report, and the lack of objective assessment of parental reading ability may have increased the likelihood of a dyslexia diagnosis. However, it should also be noted that some cases of parental dyslexia might have been undiagnosed, as some parents may not think they have a reading problem when in fact they do. Finally, parents level of education was not considered in the sample, and this has been found to be an important variable in previous studies.

4.8 Conclusion

Our results suggest that familial risk of reading difficulties is an important indicator of children likely to develop reading difficulties. The current study presented evidence that
children aged 9 – 11 years whose parents reported reading difficulties were more likely to have poorer performance on reading fluency tasks, and to be rated by their parent and teachers as inattentive. These results highlight the importance of familial risk factors and the role of genetics in the development of dyslexia. At a cognitive level, the predominant view of dyslexia has focussed on a single cognitive cause, however, more recently the intergenerational multiple deficit model proposes more complex associations between cognitive variables and dyslexia. In the following chapter, the main aim is to determine whether a single deficit, multiple deficit, or the intergenerational extension to the multiple deficit model of dyslexia is the best predictor of reading fluency in the group of children who took part in the current study.
Chapter 5: Exploration of single, double, and an (intergenerational) multiple deficit model of dyslexia.

5.1 Developmental Dyslexia

Dyslexia is a developmental disorder that affects the efficient acquisition of literacy skills (Morken, Helland, Hugdahl, & Specht, 2017). A child’s risk of dyslexia can be predicted from two sources of information (van Bergen et al., 2015). The first is the observation of cognitive deficits in domains such as phonological awareness, rapid automatic naming (RAN), and oral language deficits. The second is a family history of dyslexia (van Bergen et al., 2015), as the disorder tends to run in families, and children of parents with dyslexia are more likely to develop dyslexia themselves than children without family risk (Snowling et al., 2003; Torppa et al., 2010; van Bergen, van der Leij, et al., 2014). This heightened risk has been observed in studies investigating the neuro-anatomical, neuro-functional, cognitive, and environmental precursors of dyslexia (Swagerman et al., 2015). For instance, some studies have noted differences in structural brain networks in language areas in children at familial risk (Hosseini et al., 2013), and twin and family studies indicate that genetic factors explain the majority (60% - 80%) of individual differences in children’s word-level reading ability (Byrne et al., 2009; Haworth et al., 2009). Moreover, some studies have found no correlation between the reading accuracy of parents and their non-biological children (Swagerman et al., 2017; Wadsworth, Corley, Hewitt, Plomin, & DeFries, 2002), suggesting environmental factors could have a minimal impact on the disorder.

In the current study, the aim was to replicate the findings of Pennington and colleagues (2012), who used single and multiple deficit models of dyslexia to examine several cognitive predictors of the disorder. A further aim was to extend these findings by examining the contribution of parents’ self-reported reading difficulties, using the intergenerational multiple deficit model.
5.2 Single Deficit Model

Historically, single causal models of dyslexia have dominated the literature (Pennington, 1991), and a large body of research has documented poor phonological skills as a reliable predictor of reading difficulties in both children and adults (Caravolas et al., 2012; Moll et al., 2014; Puolakanaho et al., 2007; Ramus, Rosen, et al., 2003; Snowling, 2000; Snowling & Melby-Lervåg, 2016; Vellutino et al., 2004; Ziegler, Perry, & Zorzi, 2014). Consequently, the central hypothesis has been the phonological processing deficit hypothesis (e.g., Vellutino, 1979; Vellutino et al., 2004), that has argued that the primary cause of reading problems in individuals with dyslexia is the result of a single core impairment in the phonological component of the language system (Melby-Lervag et al., 2012; Vellutino et al., 2004), particularly problems in learning to associate letters and letter combinations with the sounds of language (Catts, McIlraith, Bridges, & Nielsen, 2017).

Despite robust support for a phonological core deficit model, current evidence indicates that a phonological deficit alone is not sufficient to account for dyslexia (Catts et al., 2017), as numerous studies examining the cognitive precursors of reading achievement have generally found that factors beyond phonological awareness are needed to account for the full range of word reading abilities (Catts, Nielsen, Bridges, Liu, & Bontempo, 2013; McGrath et al., 2011; van Bergen et al., 2011). In addition, some individuals with a phonological deficit do not demonstrate the word reading problems that are the hallmark of dyslexia (Pennington et al., 2012; Snowling, 2008). For example, in two large samples of children, Pennington et al. (2012) found numerous children with significant phonological deficits without word reading impairments. Similarly, studies investigating children at family risk of dyslexia have also reported that some unaffected siblings with no word reading difficulties have a phonological deficit (Moll, Loff, & Snowling, 2013; Snowling et al., 2003; van Bergen, van der Leij, et al., 2014). Rather than negating the role of a phonological deficit in childhood...
dyslexia, these findings have contributed to the current view that phonological deficits range in severity across individuals continuously, increasing or decreasing the impact on reading outcome depending on additional co-occurring risk or protective factors (Bishop & Snowling, 2004; Catts et al., 2017; Peterson, Pennington, Shriberg, & Boada, 2009). Consequently, researchers have now proposed multifactorial models to explain the disorder (Catts & Adlof, 2011; Pennington, 2006; Snowling, 2008; Torppa et al., 2013; van Bergen, de Jong, et al., 2014).

5.3 Double Deficit Hypothesis (DDH)

Many individuals with dyslexia have also been observed to have difficulties with fluent naming of visual items (Vaessen, Gerretsen, & Blomert, 2009), and research has found evidence of associations between rapid automatized naming (RAN) and various aspects of literacy in both typical and poor readers (Allor, 2002). These include reading fluency (e.g., Bowers, 1995; Moll, Fussenegger, Willburger, & Landerl, 2009; Papadopoulos, Georgiou, & Kendeou, 2009; Torppa, Georgiou, Salmi, Eklund, & Lyytinen, 2012; Wimmer, Mayringer, & Landerl, 2000), reading accuracy (Compton, 2000; Cornwall, 1992; Cutting & Denckla, 2001; Kirby, Parrila, & Pfeiffer, 2003; Manis, Seidenberg, & Doi, 1999; Schatschneider, Fletcher, Francis, Carlson, & Foorman, 2004; Wolf, 1997, 1999), reading comprehension (Papadopoulos et al., 2009; Schatschneider et al., 2004), and spelling (Georgiou, Torppa, Manolitsis, Lyytinen, & Parrila, 2012; Savage & Frederickson, 2005; Savage, Pillay, & Melidona, 2008; Torppa et al., 2012; Wimmer et al., 2000).

Wolf and Bowers (1999; 2000) presented the DDH as an alternative to the single phonological deficit hypothesis and conceptualise phonological processing and RAN as two partially independent core deficits. In addition, RAN is thought to contribute independent variance to reading fluency (Bowers & Swanson, 1991; de Jong & van der Leij, 1999; Kirby et al., 2003). Combinations of these deficits may result in three sub-types of dyslexia: a
single phonological deficit, a single naming speed deficit, or both phonological and naming speed deficits. Wolf, Bowers and Biddle (2000) propose that RAN “represents a demanding array of attentional, perceptual, conceptual, memory, lexical, and articulatory processes” (p. 393), while others report that problems with rapid naming may reflect wider temporal processing, recognition, and perceptual speed difficulties than those found with a phonological deficit alone (Savage & Frederickson, 2005). The theory proposes that the phonological and naming speed deficits are additive, and that the reading deficit is more severe in individuals with both impairments. A further hypothesis is that the individual deficits are associated with different aspects of literacy; phonological awareness has been primarily connected with reading and spelling accuracy, while rapid naming is more closely associated with reading fluency and rate (Compton, Defries, & Olson, 2001; Furnes & Samuelsson, 2011; Georgiou, Parrila, Cui, & Papadopoulos, 2013; Georgiou, Parrila, & Papadopoulos, 2008; Kairaluoma, Torppa, Westerholm, Ahonen, & Aro, 2013; Moll et al., 2014; Papadopoulos et al., 2009; Pennington, Cardoso-Martins, Green, & Lefly, 2001; Sunseth & Greig Bowers, 2002; Torppa et al., 2012; Vaessen et al., 2009; Vukovic et al., 2004; Wimmer et al., 2000).

While several longitudinal and cross-sectional studies have found unique contributions of naming speed in reading ability (Bowers & Swanson, 1991; Manis, Doi, & Bhadha, 2000; Pennington et al., 2012; van den Bos, Zijlstra, & van den Broeck, 2003), not all studies support an independent role for RAN in explaining dyslexia. For example, some studies have found that compared with phonological awareness, the contribution of naming speed to reading ability is modest (Cardoso-Martins & Pennington, 2004; Pennington et al., 2001; Plaza & Cohen, 2004), and others have found no unique contribution at all (Patel, Snowling, & de Jong, 2004; Torgesen, Wagner, & Rashotte, 1994).
Over the last few decades research has highlighted the heterogeneous nature of dyslexia, and in addition to impairments in phonological processing skills and RAN, other cognitive variables have been observed to be associated with the disorder. Consequently, the current consensus is that multiple cognitive difficulties can each contribute to the presence of dyslexia (Moll et al., 2013; Pennington et al., 2012).

5.4 Multiple Deficit Model (MDM)

Pennington (2006) proposed the multiple deficit model (MDM) to account for the multiple cognitive impairments found in dyslexia (as well as the other complex developmental disorders). In this model, multiple risk and protective factors interact in a probabilistic fashion to increase or decrease liability to the disorder. A phonological deficit can still be a primary contributing factor but would be accompanied by other cognitive impairments (Catts et al., 2017). For example, some research has identified oral language deficits as an important additional causal factor in children with dyslexia (Catts et al., 2017; Snowling et al., 2003).

Language based difficulties in phonological processing have long been associated with the presence of dyslexia in children, and phonological deficit theories are also prominent in theories of language impairment (Ramus, Marshall, Rosen, & van der Lely, 2013). However, the language difficulties found in children with dyslexia are not necessarily specific to phonological language processes, and children at family risk who are later diagnosed with dyslexia have been observed with broader speech and language impairments, such as slower speech, poorer grammar, articulation, and expressive vocabulary (Carroll, Mundy, & Cunningham, 2014; Carroll & Snowling, 2004; Catts, Fey, Zhang, & Tomblin, 2001; de Jong & van der Leij, 2003; Pennington & Bishop, 2009; Snowling & Melby-Lervåg, 2016). Consequently, language variables have been included in some studies investigating the multiple cognitive underpinnings of dyslexia.
Pennington and colleagues (2012) tested single, multiple, as well as a hybrid model of dyslexia in two large samples of children using measures of phonology, RAN and language. The major findings of the study were: (i) based on the variables examined in the study, approximately equal proportions of cases met both tests of model fit for the multiple deficit models (30% to 36%) and single deficit models (24% to 28%), indicating that the hybrid model (dyslexia involves multiple pathways, some involving single, and some involving multiple deficits) provided the best explanation of dyslexia, and (ii) in terms of variance in reading skills, only phonology and RAN each explained unique variance in dyslexia, and accounted for 51.9% of the variance in reading skill. Language was not found to be a significant predictor. A phonological awareness deficit was found in only 43% of the children identified with dyslexia, leading the authors to conclude that using only phonological awareness as a diagnosis would miss approximately half the cases of dyslexia. They also proposed that children with dyslexia should not be expected to fit a specific deficit profile, or to even have deficits in any of the constructs examined in their study.

**Intergenerational Multiple Deficit Model (iMDM)**

Until recently the cognitive deficits underlying a behavioural disorder such as dyslexia, and the impact of familial risk for the disorder, has mainly been studied in isolation (van Bergen et al., 2015). However, family risk of dyslexia has been found to be a strong predictor of reading outcome, with both parents transmitting risk to their offspring (Snowling et al., 2003; van Bergen, van der Leij, et al., 2014). Subsequently, the notion of multiple risk factors, (some of which are transmitted to offspring with and without dyslexia) is now widely accepted (Bishop, 2008; Pennington, 2006; Pennington & Lefly, 2001; Pennington et al., 2012; Snowling, 2008; Snowling et al., 2003; Willcutt et al., 2000).
In those children with dyslexia, the affected parent tends to be more severely impaired than those children who do not go on to develop the disorder (Torppa et al., 2011; van Bergen et al., 2012). Furthermore, the literacy abilities of the spouse of the dyslexic parent also add to the offspring’s liability (van Bergen, de Jong, et al., 2014).

Van Bergen, van der Leij, and colleagues (2014) have argued that family risk factors contribute to testing and specifying the MDM, and proposed an intergenerational addition to the model to account for the genetic risk passed on by parents. The intergenerational extension predicts that children at family risk inherit genetic risk factors from their parent or parents with dyslexia, and that at-risk children without dyslexia also inherit at least some disadvantageous gene variants, giving them a higher liability than control children (although lower than at-risk dyslexic children). Using the extended framework, the authors presented findings from a previous longitudinal study with 212 children at family risk for dyslexia (Dutch Dyslexia Programme: van der Leij et al., 2013). The study examined the effects of parental literacy skills on children’s reading outcome using cognitive precursors and correlates of dyslexia. Children at family risk with and without dyslexia were also compared concurrently and retrospectively with each other and typically developing controls on measures that included phonological awareness and RAN. In contrast to Moll et al. (2013) who found only phonological awareness was associated with family risk, the results corresponded with Pennington et al. (2012) and showed that compared to the other two groups, the FR group with dyslexia was impaired on both phonological awareness and RAN, suggesting both skills are associated with reading and risk status. The study also found that the affected parents of children with dyslexia had poorer word-reading fluency than the affected parents of children without dyslexia, however, the two at-risk groups did not differ in parental phonological processing ability. For the non-affected parents, there was a
difference between the two at-risk groups: the parents of the affected children reported more literacy difficulties compared to those of the unaffected children. The authors concluded that the differences in parental reading skills between at-risk children with and without dyslexia support the proposal that children at FR for dyslexia differ in their liability to the disorder.

A further study by van Bergen and colleagues (2015) measured reading fluency in both children and parents, and investigated three cognitive constructs in children as reported mediators of familial effects on children’s reading outcome: phonological awareness, RAN, and visual attention span. The sample consisted of 373 children (mean age 10.57) and their parents. Using the iMDM, the study investigated whether the correlations between parents and their offspring differed for mothers and fathers. The study found that the model explained 60% of the variance in children’s reading ability, with variance in children’s cognitive skills largely unexplained by parental reading skills. Parental reading skills accounted for 21% of the variance in child reading, and no significant differences were found in the effects of fathers and mothers. However, this finding contrasted with an earlier study by van Bergen et al. (2012) who found that children’s reading fluency was more strongly correlated with maternal reading ability than fathers, and another study based on an unselected sample also found no differences in the effects of fathers and mothers (Wadsworth et al., 2002).

5.5 Aims and Hypotheses

The aim of the current study was to determine whether the single, DDH, MDM, or iMDM model is the best predictor of reading fluency using measures of phonological processing, language skills, and RAN in a group of children with and without a family risk of dyslexia. Consistent with Pennington et al. (2012), a composite score of reading fluency was used as the outcome variable.
5.5.1 Hypotheses

Single deficit model

If the single deficit model was to be supported, it was expected that all children with dyslexia would show evidence of impaired phonological processing. Phonological processing was also expected to explain more variance in reading fluency than either RAN or language skills, which were not expected to add significant variance to the explanation of reading fluency.

Double deficit model

If the double deficit model was to be supported, phonological processing and RAN were each expected to make significant independent contributions to the explanation of reading fluency. There will be evidence that children can be impaired on phonological awareness, RAN or both.

Multiple Deficit Model

If the multiple deficit model was to be supported, phonological processing, language skills and RAN were each expected to make unique independent contributions to the explanation of reading fluency. Children were expected to show different patterns of impairments that could explain their reading difficulties.

Intergenerational Multiple Deficit Model

If reports of reading difficulties in mothers and fathers influence children’s reading skills, significant associations were expected between parent-reports of difficulties and child reading fluency. After controlling for parent self-reported reading skills, phonological processing, RAN and language skills were each expected to make additional contributions to the explanation of children’s reading fluency.
5.6 Method

Participants

Participants were a community sample who were recruited as part of a larger investigation that has been described in detail in previous chapters. This sample consisted of 72 children aged between 9 and 11 years (57 boys, and 15 girls), and a total of 117 parents (64 mothers and 53 fathers) who completed the Adult Reading Questionnaire (ARQ). Ethical clearance for the study was granted by the University Human Ethics Committee. Caregivers, schools, and teachers provided written consent, and child participants gave written assent prior to the commencement of testing.

Child Measures

Reading fluency

A mean composite score from the Sight Word Efficiency (SWE) and Phonemic Decoding Efficiency (PDE) subtests (Form A) from the TOWRE-2 (Torgesen et al., 2012) was used as the measure of reading fluency. Consistent with Pennington et al. (2012) children with an average reading fluency score of 85 or below were classified with dyslexia.

General Cognitive Ability

The Picture Concepts and Matrix Reasoning subtests from the WISC-IV (Weschler, 2003) were used as measures of non-verbal intellectual ability and to estimate general cognitive ability. The full description of this index has been provided in previous chapters. The scaled scores are combined to form a nonverbal fluid reasoning index (Flanagan & Kaufman, 2009). The clinical cut-off was a standard score ≤ 85, which is based on the author’s guidelines.
**Phonological Awareness**

The ability to detect the phonological components of spoken words was assessed using the Elision subtest from the CTOPP (Wagner et al., 1999). The Elision task (mean = 100; $SD = 15; \alpha = .95$) taps a child’s ability to say a word and then say the remaining part of the word after a specified phoneme or word sound is deleted, resulting in a new word or non-word. For example, participants might be instructed to say the word “cart” and then say “cart” without saying /t/. The performance index was the scaled score for the total number of words correctly identified.

**Phonological Decoding**

The participant’s ability to use phonological information to decode words was assessed with the pseudoword decoding (phonetic word attack) subtest from the Wechsler Individual Achievement Test Second Edition (WIAT-II: Wechsler, 2007). The performance index was the standard score for the total number of pseudowords correctly identified.

**Rapid Automatic Naming and Rapid Alternating Stimulus (RAN)**

Rapid automatic naming (Denckla & Rudel, 1976) tasks typically require the child to name visual stimuli presented in a linear sequence as quickly as possible (Denckla & Rudel, 1976; Wolf & Bowers, 1999). The stimuli are familiar and each one randomly occurs on multiple occasions. Generally, the stimuli are letters, digits, colours or common objects, each presented in a separate test. This study used RAN letters and digits, as the importance of distinguishing between alphanumeric and non-alphanumeric RAN has been argued. Naming of alphanumeric stimuli is considered more automatized than non-alphanumeric stimuli (van den Bos, Zijlstra, & Lutje Spelberg, 2002), and has been found to be a stronger additional
associate of word reading than non-alphanumeric stimuli (Stringer, Toplak, & Stanovich, 2004; van den Bos et al., 2002). This applies to a broad range of typically developing children as well as those with dyslexia (de Groot, van den Bos, van der Meulen, & Minnaert, 2015). The RAN tasks required oral naming of rows of continuous digits (i.e., 2, 4, 6, 7, and 9), and alphabet letters (i.e., a, d, o, p, and s). There was one practice trial for each type of stimulus (each consisting of 10 x 5 five rows of stimuli). Performance was taken as the time to correctly name the entire 10 x 5 rows (response time: RT) measured with a digital stopwatch.

Receptive and Expressive Language

The four subtests of the CELF-IV (Semel et al., 2003) were used to measure core areas of Receptive and Expressive language ability. Receptive language was assessed with Concepts and Directions and Word Classes 2. Expressive language was assessed with Recalling Sentences and Formulated Sentences. A full description of the subtests has been provided in previous chapters. Children were classified with an expressive or receptive language impairment if they had a score ≤ 85. This cut-off for clinical diagnosis is based on the authors guidelines.

Parent Measure

Family Reading History

The five-item reading factor from the Adult Reading Questionnaire (ARQ1, ARQ2, ARQ3, ARQ5, ARQ6), together with reports of probable dyslexia (ARQ15) were combined (ARQ1, ARQ2, ARQ3, ARQ5, ARQ6, ARQ15) with a possible score ranging from 0 – 13. Consistent with Snowling and colleagues the scores were added together to get a total score and were used as the parent measure of reading difficulties (Snowling et al., 2012). Higher scores on this measure were associated with increased self-reported reading difficulties (see Chapter 4).
5.7 Procedure

The current study was part of a larger investigation for which additional data was collected. For this study parents were asked to complete the ARQ/ASRS questionnaire. The full details of this process were provided in previous chapters. The child participants were administered the assessments in the following order: the WISC-IV subtests (matrix reasoning and picture concepts), the WIAT-II subtest (pseudoword decoding), the CELF-4 subtests (Concepts and Following Directions, Recalling Sentences, Formulated Sentences, and Word Classes 2) the TOWRE-2 subtests (Sight Word Efficiency and Phonemic Decoding), the CTOPP (Elision), and finally RAN digits and letters.

Analysis strategy

A combination of correlation, standard multiple regression and hierarchical regression analyses were used to evaluate the hypotheses posed. In the regression analyses reading fluency was used as the dependent variable, while pseudoword decoding, RAN and parent reading status were used as the independent variables. Given that there was missing data for 8 mothers and 19 fathers on the family history variable, a maximum likelihood estimation procedure was used to estimate these missing data. Data were analysed with and without the estimation procedure and the same results were obtained, indicating that the technique used did not influence the outcome of the data analysis. Impairment was determined by identifying children with a standard score below the 10th percentile of the control group (children without dyslexia) on the variables (phonological processing, RAN, and language skill) with the strongest association with reading fluency (Pennington et al., 2012; Ramus, Rosen, et al., 2003).
5.8 Results

Scores for reading fluency, phonological processing, language skills, RAN, and parent-reported reading difficulty variables are shown in Table 5.1.

Table 5.1

*Differences between the groups with (n = 36) and without (n = 34) dyslexia on key variables.*

<table>
<thead>
<tr>
<th></th>
<th>Dyslexia</th>
<th>Control</th>
<th>Group differences (t-test)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reading fluency</td>
<td>79.05 (5.23)</td>
<td>96.78 (9.62)</td>
<td>(t(68) = 9.65, p &lt; .001)</td>
</tr>
<tr>
<td>Non-verbal ability</td>
<td>99.00 (12.67)</td>
<td>102.79 (9.70)</td>
<td>(t(68) = 1.40, p = .166)</td>
</tr>
<tr>
<td>Mother reading difficulty</td>
<td>3.02 (2.84)</td>
<td>2.96 (3.78)</td>
<td>(t(68) = 0.76, p = .939)</td>
</tr>
<tr>
<td>Father reading difficulty</td>
<td>5.28 (4.12)</td>
<td>3.00 (2.59)</td>
<td>(t(68) = 2.75, p = .008)</td>
</tr>
<tr>
<td><strong>Rapid Naming</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Letters</td>
<td>88.83 (7.56)</td>
<td>99.18 (8.56)</td>
<td>(t(68) = 5.36, p &lt; .001)</td>
</tr>
<tr>
<td>Numbers</td>
<td>92.28 (8.70)</td>
<td>102.27 (7.86)</td>
<td>(t(68) = 5.08, p &lt; .001)</td>
</tr>
<tr>
<td>Letters/numbers</td>
<td>93.36 (11.12)</td>
<td>103.76 (9.01)</td>
<td>(t(68) = 4.35, p &lt; .001)</td>
</tr>
<tr>
<td><strong>Phonological Decoding</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pseudoword</td>
<td>88.42 (8.53)</td>
<td>101.85 (8.68)</td>
<td>(t(68) = 6.53, p &lt; .001)</td>
</tr>
<tr>
<td><strong>Phonological Awareness</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Elision</td>
<td>9.28 (2.13)</td>
<td>11.24 (2.00)</td>
<td>(t(68) = 9.65, p &lt; .001)</td>
</tr>
<tr>
<td><strong>Language</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Expressive</td>
<td>103.94 (9.01)</td>
<td>110.65 (10.82)</td>
<td>(t(68) = 2.83, p = .006)</td>
</tr>
<tr>
<td>Receptive</td>
<td>92.97 (10.60)</td>
<td>100.38 (8.89)</td>
<td>(t(68) = 3.16, p = .002)</td>
</tr>
</tbody>
</table>

\(^!^Two children with clinical language impairment were excluded from the control group for these analyses.

Apart from non-verbal ability and reports of mothers’ reading difficulty, for which no significant group difference was found, the group with dyslexia performed more poorly on all reading and language measures. The correlations between these variables are shown in Table 5.2. Better reading fluency was associated with better phonological processing, RAN, and
expressive language ability. Reading fluency had no significant linear association with non-verbal, or receptive language ability. Children with better pseudoword decoding also showed better performance on each of the RAN and expressive language tasks. Although significant, the associations between the Elision task and these measures was substantially lower. Except for a positive association between RAN letter naming and expressive language, there were no significant associations between RAN and the language measures. For children, reports of greater reading difficulties by their fathers, but not mothers, was associated with poorer reading fluency, pseudoword decoding, and slower performance on RAN letters and numbers.

**Single Deficit Models**

The initial analysis of the different measures used three standard multiple regression analyses to determine the extent to which phonological processing, RAN and language measures could separately explain variation in reading fluency (see Table 5.3). When either the measures of phonological processing or RAN were used as the IVs, at least 50% of the variance in reading fluency was explained. For the language measures, this was substantially less at 13.4%. Consistent with Pennington et al. (2012), for each of the analyses the variable with the strongest association with reading fluency was selected to use in the double (DDH) and multiple deficit (MDM, iMDM) model analyses. These were the pseudoword decoding task, RAN letter naming, and expressive language variables.

**Double Deficit Models**

In the DDM, phonological decoding and RAN letters should each contribute unique variance to children’s reading fluency. To determine the extent to which RAN letters, and pseudoword decoding, each contributed incremental variance to reading fluency, two hierarchical regression analyses were conducted (see Table 5.4).
Table 5.2

Pearson Correlations reading fluency, phonological measures, rapid naming, language and parent-report of reading difficulties (N = 72).

<table>
<thead>
<tr>
<th></th>
<th>Mean (SD)</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reading fluency (1)</td>
<td>87.94 (11.8)</td>
<td>.082</td>
<td>.368**</td>
<td>.697***</td>
<td>.692***</td>
<td>.610***</td>
<td>.610***</td>
<td>.356**</td>
<td>.309**</td>
<td>.233</td>
<td>-.366**</td>
</tr>
<tr>
<td>Non-verbal ability (2)</td>
<td>100.65 (11.5)</td>
<td>-</td>
<td>.202</td>
<td>.108</td>
<td>.020</td>
<td>.266</td>
<td>.136</td>
<td>.130</td>
<td>.387***</td>
<td>-.084</td>
<td>.082</td>
</tr>
<tr>
<td><strong>Phonological</strong></td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>Elision (3)</td>
<td>10.33 (2.3)</td>
<td>-</td>
<td>.361**</td>
<td>.290**</td>
<td>.306**</td>
<td>.361***</td>
<td>.122</td>
<td>.201</td>
<td>-.134</td>
<td>-.208</td>
<td></td>
</tr>
<tr>
<td>Pseudoword (4)</td>
<td>95.17 (10.8)</td>
<td>-</td>
<td>.503***</td>
<td>.496***</td>
<td>.457***</td>
<td>.364**</td>
<td>.219</td>
<td>.090</td>
<td>-.287**</td>
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<td></td>
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<tr>
<td><strong>Rapid Naming</strong></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Letters (5)</td>
<td>93.93 (9.4)</td>
<td>-</td>
<td>.709***</td>
<td>.706***</td>
<td>.276**</td>
<td>.246</td>
<td>.052</td>
<td>-.283**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Numbers (6)</td>
<td>97.22 (9.5)</td>
<td>-</td>
<td>.670***</td>
<td>.198</td>
<td>.191</td>
<td>.038</td>
<td>-.235**</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Letters/Numbers (7)</td>
<td>98.57 (11.1)</td>
<td>-</td>
<td>.230</td>
<td>.207</td>
<td>.169</td>
<td>-.095</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Language</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Expressive (8)</td>
<td>106.57 (10.9)</td>
<td>-</td>
<td>.700***</td>
<td>.002</td>
<td>-.193</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Receptive (9)</td>
<td>95.92 (11.0)</td>
<td>-</td>
<td>-.049</td>
<td>-.174</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Parent difficulty reading</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mother reading difficulty (10)</td>
<td>3.04 (3.3)</td>
<td>-</td>
<td>-.015</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Father reading difficulty (11)</td>
<td>4.20 (3.6)</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**p < .01, *** p < .05. Note. Correlations > .01 were not considered statistically significant**
Table 5.3

*Single variable explanations of reading fluency (N = 72)*

<table>
<thead>
<tr>
<th>Variables</th>
<th>B</th>
<th>SE (B)</th>
<th>β</th>
<th>sr²</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Phonological Skills, R² = 50.1% F(2, 69) = 34.62, p &lt; .001</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pseudoword decoding</td>
<td>.704</td>
<td>.099</td>
<td>.648***</td>
<td>36.6%</td>
</tr>
<tr>
<td>Elision</td>
<td>.673</td>
<td>.459</td>
<td>.134</td>
<td>1.5%</td>
</tr>
<tr>
<td><strong>Rapid Naming, R² = 52.2% F(3, 68) = 24.78, p &lt; .001</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Letters</td>
<td>.546</td>
<td>.165</td>
<td>.438**</td>
<td>7.7%</td>
</tr>
<tr>
<td>Numbers</td>
<td>.220</td>
<td>.155</td>
<td>.178</td>
<td>1.4%</td>
</tr>
<tr>
<td>Letters and numbers</td>
<td>.191</td>
<td>.133</td>
<td>.181</td>
<td>1.4%</td>
</tr>
<tr>
<td><strong>Language processing R² = 13.4%, F(2, 69) = 5.33, p = .007</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Expressive</td>
<td>.293</td>
<td>.17</td>
<td>.274</td>
<td>3.8%</td>
</tr>
<tr>
<td>Receptive</td>
<td>.125</td>
<td>.16</td>
<td>.118</td>
<td>0.7%</td>
</tr>
</tbody>
</table>

*Note.* ***p < .001, **p < .01, *p < .05*

In the first analysis, pseudoword decoding was entered first, followed by RAN letter naming, and in the second the reverse occurred. When entered at Step 1, both pseudoword decoding, and RAN letter naming accounted for 48% of the variance in reading fluency. Independently each accounted for approximately 16% of the variance, indicating that both variables contributed to reading fluency in the sample tested.

Using the scores based at the 10th percentile for the control group on the measures of pseudoword decoding and RAN letter naming, the number of children in the group with dyslexia with an impairment in phonological processing, RAN, or both were obtained.
Table 5.4

*Reading fluency explained by Pseudoword Decoding and RAN (N = 72).*

<table>
<thead>
<tr>
<th>Variables</th>
<th>B</th>
<th>SE (B)</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td>*Step 1 Pseudoword decoding, ( R^2_{\text{chg}} = 48.5% ) F(1, 70) = 66.00, ( p &lt; .001 )</td>
<td>.757</td>
<td>.093</td>
<td>.697***</td>
</tr>
<tr>
<td>Pseudoword task</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Step 2 Rapid Letter Naming, ( R^2_{\text{chg}} = 15.6% ) F(1, 69) = 30.15, ( p &lt; .001 )</td>
<td>.863</td>
<td>.108</td>
<td>.692</td>
</tr>
<tr>
<td>Rapid letter naming</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Step 1 Rapid Letter naming ( R^2_{\text{chg}} = 47.9% ) F(1, 70) = 64.4, ( p &lt; .001 )</td>
<td>.507</td>
<td>.091</td>
<td>.467***</td>
</tr>
<tr>
<td>*Step 2 Pseudoword decoding ( R^2_{\text{chg}} = 16.3% ) F(1, 69) = 31.33, ( p &lt; .001 )</td>
<td>.571</td>
<td>.104</td>
<td>.458***</td>
</tr>
</tbody>
</table>

\( R^2 = 64.2, \text{adj } R^2 = 63.1 \)

Of the 36 (50%) of the children with dyslexia, there were 10 (27.8%) children with an impairment in both phonological decoding and RAN, 10 (27.8%) with a phonological impairment only, and 7 (19.4%) with a RAN impairment only. Together with the hierarchical regression analyses, these results support the DDH, and indicate that either phonological decoding, RAN, or both can contribute to the presence of dyslexia in an individual child. However, there were 9 (25%) children in the sample with dyslexia without an impairment in phonological processing or RAN, a finding consistent with Pennington et al. (2012).

*Multiple Deficit Model*

Multiple deficit models suggest there are multiple cognitive deficits associated with dyslexia. The MDM was tested by adding the expressive language-based variable at the final step of the hierarchical regression analyses (shown in Table 5.4), after pseudoword decoding and RAN letters. Expressive language was used in the analysis, as this was the language
variable with the strongest association with reading fluency (see Table 5.2). Adding the language variable failed to add significant additional variance to the explanation of reading fluency, over that explained by pseudoword decoding and RAN letters, $F(1, 68) = 0.81, p = .372$. When expressive language was included, the unique contributions of the phonological and RAN variables changed little (see Table 5.5). These findings are consistent with Pennington et al. (2012), who also found that language skills were a weak predictor of reading fluency in their younger participants.

Table 5.5

*Multiple deficit model*

<table>
<thead>
<tr>
<th>Variables</th>
<th>B</th>
<th>SE (B)</th>
<th>$\beta$</th>
<th>$sr^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pseudoword task</td>
<td>.484</td>
<td>.094</td>
<td>.445***</td>
<td>13.8%</td>
</tr>
<tr>
<td>Rapid letter naming</td>
<td>.560</td>
<td>.105</td>
<td>.449***</td>
<td>14.8%</td>
</tr>
<tr>
<td>Expressive language</td>
<td>.075</td>
<td>.084</td>
<td>.070</td>
<td>0.04%</td>
</tr>
</tbody>
</table>

***p < .001 $R^2 = 64.6\%$ $F(3, 68) = 41.37, p < .001$

The number of children with dyslexia and an expressive language impairment was estimated by selecting children with a score on this variable below the 10th percentile score of the control group (see Table 5.6). These scores were combined with those children previously found to have impairments on the measures of phonological decoding and RAN letter naming. Consistent with the results of Pennington et al. (2012), children with dyslexia showed evidence of a variety of cognitive impairments. Children were most likely to have impaired phonological decoding only, or a combined deficit in phonological decoding and RAN letter naming. Consistent with the regression analysis, few children showed evidence of a language-based impairment. There were also a proportion of children with dyslexia who showed no evidence of impairments in phonological processing, RAN or language skills. These findings support the hypothesis that children would show evidence of multiple deficits
and replicate those of Pennington et al. (2012) who also found that language was a much weaker predictor of reading fluency in their younger sample.

Table 5.6

*Numbers of children with dyslexia with single and multiple deficits (N = 36)*

<table>
<thead>
<tr>
<th>Impairment</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phonology only</td>
<td>9</td>
<td>25%</td>
</tr>
<tr>
<td>Rapid Naming only</td>
<td>7</td>
<td>19.4%</td>
</tr>
<tr>
<td>Language only</td>
<td>1</td>
<td>2.8%</td>
</tr>
<tr>
<td>Phonology and Rapid Naming</td>
<td>9</td>
<td>25%</td>
</tr>
<tr>
<td>Phonology and Language</td>
<td>1</td>
<td>2.8%</td>
</tr>
<tr>
<td>Rapid Naming and Language</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Phonology, Rapid naming and Language</td>
<td>1</td>
<td>2.8%</td>
</tr>
<tr>
<td>No Deficit</td>
<td>8</td>
<td>22.2%</td>
</tr>
</tbody>
</table>

*Intergenerational multiple deficit model (iMDM)*

To test the iMDM, parent-reports of reading difficulties were entered at Step 1 of a hierarchical regression analysis, with language, RAN and phonological measures entered at Step 2 (see Table 5.7). Parent-reports of reading difficulties accounted for 18.6% of the variance in children reading fluency scores, with father’s reports of reading difficulties making the only unique contribution. Consistent with the previous analyses, a further 46.3% of the variance was accounted for with the addition of proximal cognitive variables to the model. Father’s reports of reading difficulties continued to make a significant contribution to their children’s reading fluency. Contrasting the analysis with the MDM that used only cognitive variables, there was a drop in the unique contribution by RAN letters only with the inclusion of parent reading difficulties. These results support the hypothesis that reports of
parent reading difficulties would contribute to the explanation of poor reading fluency in children.

Table 5.7

*Intergenerational multiple deficit model. Does parent-report of reading difficulty make a difference?*

<table>
<thead>
<tr>
<th>Variables</th>
<th>B</th>
<th>SE (B)</th>
<th>β</th>
<th>Sr²</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Step 1 Reading fluency R² = 18.6%</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Father reading difficulty</td>
<td>-1.18</td>
<td>.352</td>
<td>-.363**</td>
<td>13.1%</td>
</tr>
<tr>
<td>Mother reading difficulty</td>
<td>.803</td>
<td>.383</td>
<td>.228*</td>
<td>5.2%</td>
</tr>
<tr>
<td><strong>Step 2 R²chg= 46.3%, F(2, 69) = 7.87, p = .001, F(3, 66) = 26.96, p &lt; .001</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Father reading difficulty</td>
<td>-.613</td>
<td>.250</td>
<td>-.188**</td>
<td>3.2%</td>
</tr>
<tr>
<td>Mother reading difficulty</td>
<td>.463</td>
<td>.262</td>
<td>.131</td>
<td>1.7%</td>
</tr>
<tr>
<td>Expressive language</td>
<td>.084</td>
<td>.085</td>
<td>.078</td>
<td>0.2%</td>
</tr>
<tr>
<td>RAN letters</td>
<td>.369</td>
<td>.088</td>
<td>.349***</td>
<td>9.4%</td>
</tr>
<tr>
<td>Pseudoword decoding</td>
<td>.481</td>
<td>.096</td>
<td>.443***</td>
<td>13.3%</td>
</tr>
</tbody>
</table>

R² total = 64.8%, R²adj = 62.2%

One item on the Adult Reading Questionnaire (Snowling et al., 2012), asks respondents to determine whether they believe that they have dyslexia. Using this item, for children with dyslexia, 21 (58.3%) had parents who self-reported potential problems with dyslexia. Using the multiple deficit classification (see Table 5.8), of the 8 children with no cognitive impairment 3 had fathers reporting potential problems with dyslexia. Both children had impaired language processing, and half of those with a RAN impairment had fathers with potential dyslexia. Unexpectedly, few children with a phonological processing impairment had fathers reporting potential problems with dyslexia.
## Table 5.8

**Numbers of children with dyslexia with single and multiple deficits (N = 36)**

<table>
<thead>
<tr>
<th>Impairment</th>
<th>Number</th>
<th>Percentage</th>
<th>Father potential Dyslexia (15, 41.7%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phonological only</td>
<td>9</td>
<td>25%</td>
<td>2 (22.2%)</td>
</tr>
<tr>
<td>Rapid Naming only</td>
<td>7</td>
<td>19.4%</td>
<td>4 (57.1%)</td>
</tr>
<tr>
<td>Language only</td>
<td>1</td>
<td>2.8%</td>
<td>1 (100%)</td>
</tr>
<tr>
<td>Phonological and Rapid Naming</td>
<td>9</td>
<td>25%</td>
<td>4 (44.4%)</td>
</tr>
<tr>
<td>Phonological and Language</td>
<td>1</td>
<td>2.8%</td>
<td>1 (100%)</td>
</tr>
<tr>
<td>Phonological, Rapid naming and Language</td>
<td>1</td>
<td>2.8%</td>
<td>0</td>
</tr>
<tr>
<td>No Deficit</td>
<td>8</td>
<td>22.2%</td>
<td>3 (37.5%)</td>
</tr>
</tbody>
</table>

### 5.9 Discussion

Despite decades of research into dyslexia, there is no consensus on the causes or main mechanisms responsible for the problem. While research has indicated that many children with dyslexia perform poorly on tasks that rely on phonological functioning (Goswami, 2003), the current evidence shows a single underlying deficit cannot explain the multiple risk factors that have been found to be associated with the disorder (Pennington et al., 2012). The aim of this study was to examine whether the best predictor of reading fluency in a group of children was a traditional single, double, or the more recent multiple deficit model. As familial transmission is now recognised as an important risk factor for dyslexia and has been found to be a strong predictor of reading for children at family risk of the disorder, the intergenerational extension to the MDM was also included for testing. The results of the current study indicated that the group of children with dyslexia performed more poorly on all reading and language measures. Children with better reading fluency also had better...
phonological and language skills and were faster on the measures of RAN. In addition, better performance on pseudoword decoding was associated with better expressive language skills and faster performance on the RAN tasks.

When examining single deficit models of dyslexia, we found that phonological processing and RAN each explained similar significant amounts of variance in children’s reading fluency. This finding is in accord with Pennington and colleagues (2012) who also found that these variables each explained significant unique variance in reading fluency. However, when using the same outcome reading variable, the Pennington study found that phonological processing was a stronger predictor of children’s reading fluency than RAN. This difference may be explained by differences in age between the samples. The children assessed by Pennington et al. were measured first in kindergarten, and again in first grade, whereas the children in the current study were older, and were assessed in grades 3 to 6, suggesting RAN may be a better predictor of reading fluency in older than younger children.

When the double deficit model of dyslexia was examined, the equal contribution of phonological processing and RAN was again observed. Together they accounted for approximately half of the variance (whether phonological processing or RAN was entered first or independently), each accounting for around 16% of the variance. The results supported the DDH and indicated that three quarters of the current sample had an impairment in phonology, RAN, or both. However, consistent with Pennington et al. (2012) 25% of children with dyslexia had no impairment in phonological processing or RAN, suggesting that other cognitive deficits are involved in dyslexia.

When the MDM was examined, expressive language was included in the analysis along with pseudoword decoding and RAN letters, however, consistent with Pennington et al. (2012), the language based variable made no significant unique contribution to the explanation of reading fluency over that already explained by phonology and RAN. When
the number of children with impairments in phonological processing, RAN and language were estimated, the results were similar to the estimation for the DDH. Children with dyslexia were most likely to have impaired phonological processing only, RAN deficits only, or a combination of phonological processing and RAN deficits. An expressive language impairment failed to account for many of the children who showed no evidence of a phonological or RAN deficit, leaving almost a quarter of the sample without evidence of an impairment on any of the variables measured in this study. Finally, parent-reports of reading difficulties were entered to test the intergenerational MDM. The results were consistent with the hypothesis, and parent-reports of reading difficulties accounted for a substantial proportion (18.6%) of the variance in children’s reading fluency scores, a result that was also consistent with the findings of van Bergen and colleagues (2015). However, the current study found that only father reports of reading difficulties made a significant contribution to children’s reading fluency. Children whose fathers reported greater reading difficulties also had poorer reading fluency, pseudoword decoding and slower naming of letters and digits. In contrast, there was no significant association found between mother’s reports of reading difficulties and children’s reading fluency scores, and this might be explained by the small proportion of mothers’ reporting significant reading difficulties in the current sample.

Van Bergen and colleagues found that paternal reading ability was a better predictor of children’s reading ability for fathers with higher-level education (van Bergen et al., 2015), however, another study that used the same standardised measures of reading fluency for children and their parents, found no differences in the contributions between mothers and fathers (Wadsworth et al., 2002). It is a limitation of the current study that there was no objective measure of parent reading fluency, as the use of only a self-report measure of reading difficulties may not have provided as accurate a measure of parental ability as those studies that have used standardised reading measures to assess parent reading skills.
However, although there is not yet extensive documentation on the validity and reliability of adult self-reported reading measures, there is a growing body of family risk research that suggests self-reports are both a valid and reliable measure of adult reading difficulties (Leavett et al., 2014; Lefly & Pennington, 2000; Snowling et al., 2012).

5.10 Conclusion

The current study investigated single, double, and multiple deficit models of dyslexia using the same cognitive variables as Pennington and colleagues (2012). Consistent with the Pennington study, the results indicated there are various pathways to dyslexia, and based on the cognitive variables that were assessed, the hybrid model provided the best framework to describe the single and multiple deficits observed in the child sample. However, the range of cognitive variables examined was not extensive, and almost a quarter of the current sample did not have a phonological, RAN or language impairment. The findings support a multifactorial explanation of dyslexia but suggest there are other variables associated with dyslexia that were not included in the current study. A limitation of the current study is that it is not known whether the children with a single deficit have other deficits that remain unidentified, and future studies examining the MDM should assess for a broader range of cognitive deficits such as inattention, which has been shown to have a close association with reading difficulties.

The inclusion of the parental risk variable provided a significant contribution to the explanation of children’s reading fluency and supports the potential usefulness of the intergenerational model as a framework to conjointly investigate the cognitive deficits underlying dyslexia, as well as the transmission of familial risk factors for the disorder. However, another limitation of the present study is that it did not examine the transmission of any environmental influences, as it has also been proposed that in addition to the transmission of familial genetic risk factors, parental skills could be transferred to offspring via
environmental (i.e., parental reading habits, education level, socio-economic factors) pathways (van Bergen, van der Leij, et al., 2014), and these variables could be included in future studies.

In the current chapter we investigated whether a single or multiple deficit model was the best predictor of reading fluency in children with and without family risk of dyslexia. Following in Chapter 6 we investigate co-occurring dyslexia and ADHD using a multiple deficit framework to determine (a) whether there are neurocognitive deficits that are common to both disorders, and that may explain the co-occurrence between them, and (b) if there are neurocognitive deficits that are unique to each disorder.
Chapter 6: A multiple deficit model of dyslexia and attention deficit with hyperactivity (ADHD) and without hyperactivity (AD).

6.1 Theories of the co-occurrence between Developmental Dyslexia and Attention Deficit Hyperactivity Disorder (ADHD)

Several competing theoretical accounts have been proposed in the past to explain the association between developmental dyslexia and ADHD, each with a differing neuropsychological profile of the co-occurring condition (Germanò et al., 2010). For example, early single deficit theories proposed a double dissociation model. This model hypothesised that dyslexia and ADHD were linked by two opposing patterns of impairment in two different cognitive domains (e.g., individuals with dyslexia exhibited phonological processing deficits without executive function deficits, and individuals with ADHD exhibited executive function deficits in the absence of phonological deficits). Three independent phenotypes were considered: a phonological deficit for dyslexia only, an executive function deficit for ADHD only, and an additive combination of both disorders for the dyslexia + ADHD group (Pennington et al., 1993). While early results supported the double dissociation (e.g., Felton, Wood, Brown, Campbell, & Harter, 1987; Raberger & Wimmer, 2003; Semrud-Clikeman, Guy, Griffin, & Hynd, 2000), and other evidence indicated that the co-occurring condition showed the additive combination of the deficit associated with each disorder separately (Pisecco, Baker, Silva, & Brooke, 2001; Swanson, Mink, & Bocian, 1999; Willcutt et al., 2001), subsequent studies failed to replicate these findings (Nigg, Carte, Hinshaw, & Treuting, 1998; Reader, Harris, Schuerholz, & Denckla, 1994; Willcutt et al., 2001).

Another early theory (phenocopy hypothesis) proposed a bidirectional influence between the two disorders; problems with reading make children appear inattentive, whereas
problems associated with ADHD disrupt learning (Pennington et al., 1993). According to this theory the comorbid group exhibit the neuropsychological deficits of dyslexia only, and the behavioural characteristics of both dyslexia and ADHD. However, later studies found that the comorbid group was impaired on all measures (e.g., inhibition and working memory, as well as phonological awareness) failing to find support for this hypothesis (e.g., Willcutt et al., 2001; Willcutt, Pennington, et al., 2005). While others reported that co-occurring dyslexia + ADHD was associated with a combination of the cognitive impairments found in each disorder alone (Adams & Snowling, 2001; Nigg, Hinshaw, Carte, & Treuting, 1998; Raberger & Wimmer, 2003; Rucklidge & Tannock, 2002; Willcutt, Pennington, et al., 2005).

Alternative explanations for the association between dyslexia and ADHD include the cognitive subtype hypothesis (de Jong, Oosterlaan, & Sergeant, 2006). This hypothesis argues that co-occurring dyslexia + ADHD may reflect different causal mechanisms from those found for either disorder alone (de Jong et al., 2006). Support for this hypothesis comes from findings that children with dyslexia + ADHD show more severe impairments in phonological skills, rapid automatic naming (Rucklidge & Tannock, 2002), working memory (Katz, Brown, Roth, & Beers, 2011), and inhibition processes (Willcutt et al., 2001), than children with either disorder alone. For example, Willcutt and colleagues (2001) found that children with ADHD were impaired on EF tasks (e.g., inhibition, vigilance, and set shifting), while children with dyslexia showed impairment on phonological and verbal working memory measures. However, children with co-occurring conditions were more severely impaired on inhibition and phonological measures than the group with ADHD alone, dyslexia alone, and controls. Rucklidge and Tannock (2002) found that the group with ADHD + dyslexia were slower at naming digits than groups with ADHD or dyslexia alone. Another study that investigated ADHD and dyslexia found the co-occurring group had a unique deficit in naming digits (e.g., the groups with dyslexia alone and ADHD alone did not have a deficit
in naming digits), as well as more severe working memory difficulties than the single disorder groups (Bental & Tirosh, 2007). It was proposed that the co-occurring group may represent a distinct disorder (specific subtype hypothesis; Rucklidge & Tannock, 2002) with a separate etiological trajectory that differs from the simple additive combination of deficits from either disorder alone (DuPaul, Gormley, & Laracy, 2013; Germanò et al., 2010). However, Willcutt, Pennington and colleagues (2005) failed to replicate the findings of Rucklidge and Tannock (2002) that the co-occurring group was most impaired on letter and colour naming. The cross-assortment hypothesis explanation proposes dyslexia and ADHD are transmitted independently in families, and that their association may be the result of non-random mating (Faraone et al., 1993). According to this theory, the partners of individuals with ADHD had significantly higher rates of dyslexia than the partners of individuals without ADHD. It was proposed that the cognitive profile of the comorbid group would be consistent with the additive combination of the dyslexia only deficits, and ADHD only deficits. However, further studies indicated that the cross-assortment hypothesis was not able to provide a sufficient explanation for most comorbid cases (Doyle, Faraone, DuPre, & Biederman, 2001; Friedman et al., 2003). Another view (the ‘shared aetiology’ hypothesis) proposes that the co-occurring condition stems from shared genetic risk factors that contribute to different patterns of cognitive impairment for each disorder. In this hypothesis, the shared genetic risk factor(s) coincide with other genetic and environmental risk factors that result in the development of separate underlying cognitive impairments, which in turn produce co-occurring dyslexia + ADHD (de Jong et al., 2006).

Examining the phenotypic associations between dyslexia and ADHD has been a rigorous and active area of research in recent years (Peterson et al., 2017). There is now evidence of shared cognitive, as well as genetic risk factors (McGrath et al., 2011; Willcutt, Pennington, et al., 2010), and while the genetic association between dyslexia and ADHD
does not rule out the possibility of a distinct, or unique cognitive deficit for each disorder (van Bergen, van der Leij, et al., 2014), accumulating evidence indicates there are numerous shared etiological risk factors involved in the association between the disorders (Kere, 2014; Li, Chang, Zhang, Gao, & Wang, 2014; Mascheretti et al., 2017; McGrath et al., 2011; Moura et al., 2017; Rucklidge & Tannock, 2002). Some of these include executive function (EF) processes such as verbal working memory (Moura et al., 2017; Moura et al., 2015; Rucklidge & Tannock, 2002; Swanson et al., 2009; Willcutt, Pennington, et al., 2005), tasks that rely on speed of processing such as rapid automatic naming (Catts et al., 2002; Landerl et al., 2013; Shanahan et al., 2006; Willcutt, Pennington, et al., 2005; Wolf, 1997) and reading comprehension (e.g., Ghelani et al., 2004; Miller et al., 2013).

Support for the overlap of cognitive deficits among the developmental disorders also comes from neurological studies that suggest that any neurological deviation will cause diffuse and non-specific deficits across multiple neural systems (Karmiloff-Smith, 2009; Thelen & Bates, 2003). Consequently, the interactive nature of neural architecture, and the reported overlap in cognitive deficits, has led some researchers to question whether a clear division can be made between the core deficits specific to either dyslexia or ADHD (e.g., Pennington, 2006). It is increasingly recognised in developmental research that dyslexia and ADHD are best conceptualised as dimensional disorders (Peterson et al., 2017; Plomin & Kovas, 2005), that arise from the additive and interactive effects of multiple genetic and environmental risk factors, and that genuine comorbidity is the result of shared genetic risk factors that increase susceptibility for both disorders (Peterson et al., 2017).

The current study aimed to examine the extent to which impairments in phonological processing and rapid automatic naming are unique to dyslexia, and the extent that interference control is unique to ADHD and AD. Secondly, two cognitive variables that are potentially shared (working memory and reading comprehension) between the disorders are
examined using a multiple deficit framework. As research indicates each of the ADHD subtypes may differ in their cognitive profile (Nigg et al., 2002), as well as in their genetic association with dyslexia (Willcutt, Pennington, et al., 2007), we also investigate the unique and/or shared effects that may apply for attention deficit (AD), as it is the inattentive subtype of ADHD which has been found to be strongly related to reading difficulties (e.g., Willcutt, Pennington, et al., 2005).

**Multiple deficit model (MDM)**

The MDM was proposed by Pennington (2006) to explain the cause of co-occurrence, and the presence of the considerable overlap of neurocognitive deficits found between the developmental disorders. The model proposes that developmental disorders arise from multiple genetic and environmental risk (and protective) factors, and result in deficits in multiple neurocognitive domains (McGrath et al., 2011; Moura et al., 2017; van Bergen, van der Leij, et al., 2014). The model hypothesises that there may be etiological factors that are associated with each disorder alone (although the multiple deficit framework does not support the idea of a complete dissociation between disorders: Pennington, 2006), as well as some that will be shared with other developmental disorders. As some of these underlying cognitive skills are shared, the model predicts that co-occurrence among the developmental disorders would be common (McGrath et al., 2011; Peterson et al., 2017; van Bergen, van der Leij, et al., 2014). Accordingly, estimates of co-occurrence between dyslexia and ADHD are substantial, and range from approximately 20% to 50% (Bental & Tirosh, 2007; Del’Homme et al., 2007; Semrud-Clikeman et al., 1992; Willcutt, Pennington, et al., 2005), depending on the criteria for inclusion (August & Garfinkel, 1990; Dykman & Ackerman, 1991; Shaywitz et al., 1990; Willcutt & Pennington, 2000). Moreover, the threshold between impaired and unimpaired is proposed to be arbitrary, as the distribution of liability is continuous and quantitative, rather than discrete and categorical (Pennington, 2006). A recent study by
Petersen and colleagues (2017) examined the overlap among individual differences in word reading, attention, and math ability using the MDM framework. Participants included a total of 636 children and adolescents aged 8 to 16 years who were part of the Colorado Learning Disability Research Centre (CLDRC) twin study. The study compared two multiple predictor models. The first model specified relationships based on previous results for their sample, and the second model emphasized the contribution of executive function processes to symptom overlap. Model 1 predicted that phonological processing and naming speed would uniquely predict reading, while processing speed (a composite score of processing speed and naming speed) was hypothesised to be a significant predictor of both dyslexia and inattention. Model 2 predicted that working memory and inhibition would also account for comorbidity among the symptom dimensions. The findings were consistent with the predictions of the multiple deficit framework. Word reading and attention each had at least one unique predictor that was not shared. Phonological processing and naming speed were found to be unique predictors for reading, while response inhibition was a unique predictor for attention. Processing speed was found to explain significant variance between reading and attention. However, the authors noted significant age variance in their older and younger subgroups, and suggested that results may differ in larger samples, across narrower age bands, or in individuals younger or older than those included in the study. Research findings investigating the unique and shared cognitive deficits associated with dyslexia and ADHD or AD have been mixed, and a number of these variables will be discussed below.

**Deficits that differentiate dyslexia and ADHD or AD**

**Phonological Processing**

There is substantial support in the literature for a deficit in phonological processing for children and adults with dyslexia (Boada & Pennington, 2006; Olson, Forsberg, Wise, & Rack, 1994; Puolakanaho et al., 2007; Ramus, Rosen, et al., 2003; Snowling, 2000; van de
Voorde, Roeyers, Verté, & Wiersema, 2010; Vellutino et al., 2004). However, despite the large body of literature supporting word reading problems as a hallmark of dyslexia, there is research to indicate than a phonological deficit alone is insufficient to account for dyslexia (Pennington et al., 2012; see Chapter 5). For instance, studies examining the cognitive precursors of reading achievement have generally found that factors beyond phonological awareness are needed to account for the full range of word reading ability (Catts et al., 2013; McGrath et al., 2011; van Bergen et al., 2011). Moreover, as the results of our previous study showed, individuals have been observed with word reading impairments without the expected phonological impairments (Pennington et al., 2012; Snowling, 2008).

There have also been consistent reports of impaired reading proficiency in children with ADHD, and some investigations have revealed that children with ADHD, even without co-occurring reading disorders, show impaired lexical (e.g., orthographic decision task) and sub-lexical (e.g., phonological decision task) processing skills (de Jong, Licht, Sergeant, & Oosterlaan, 2012; de Jong et al., 2009; Willcutt, Pennington, et al., 2005). Furthermore, some children with ADHD have been observed to perform significantly less efficiently on measures of oral contextual and non-contextual reading, as well as on silent non-contextual reading fluency (Ghelani et al., 2004; Jacobson et al., 2011), and this provides some support for the proposal that there is no clear dissociation of core deficits specific to dyslexia and ADHD (e.g., Pennington, 2006).

Rapid Automatic Naming

There is substantial evidence of rapid automatic naming (RAN) deficits in individuals with dyslexia (Bowers, 1995; Moll et al., 2009; Papadopoulos et al., 2009; Torppa et al., 2012; Wimmer et al., 2000), and slow performance on RAN tasks is considered a core difficulty among children with the disorder (Semrud-Clikeman et al., 2000; Wolf & Bowers, 1999). Rapid naming tasks have been shown to be a significant predictor of reading
performance both concurrently (Plaza & Cohen, 2003; Wolf & Bowers, 1999) and longitudinally (Kirby et al., 2003; Schatschneider et al., 2004), and a close association has been proposed between RAN and reading fluency (Schatschneider, Carlson, Francis, Foorman, & Fletcher, 2002) based on timing demands and the necessary integration of phonological and lexical processes associated with the task (Bowers, 1995).

While RAN deficits have been primarily observed in dyslexia, some investigations have identified slowed naming speed among children with ADHD, without basic reading difficulties (Rucklidge & Tannock, 2002; Wodka, Simmonds, Mahone, & Mostofsky, 2009). One explanation proposes that the slow performance on RAN may reflect inefficiency in working memory (Brooks, Berninger, & Abbott, 2011). Others suggest weaknesses in executive control processes, or a failure to maintain levels of automaticity may underlie slowed naming speed among children with ADHD (Jacobson et al., 2011). Rapid naming tasks have been found to be among the best predictors of the inattentive symptoms associated with ADHD (Chhabildas et al., 2001; Rucklidge & Tannock, 2002; Weiler, Bernstein, Bellinger, & Waber, 2000). RAN relies on continuous responding and sustained attention to visual stimuli (Rucklidge, 2006), and it has been suggested that individuals with sustained attention deficits process visual information more slowly, particularly in the context of increased cognitive load and integration of multiple operations (Tannock, Martinussen, & Frijters, 2000).

A study by Pham and colleagues (2011) examined the relationship between inattention and RAN on oral reading fluency in a sample of 104 typically developing children aged 8 to 11 years. The study found all four RAN stimuli (letters, digits, objects and letters) contributed to reading fluency, however, RAN letters explained the most variance (24%), and was more strongly associated with reading fluency than the other RAN stimuli. Parent and teacher ratings of inattention predicted RAN speed, in addition to oral reading fluency.
Further, the combined parent and teacher ratings of inattention were negatively correlated with RAN speed, suggesting higher levels of attention are needed to perform well on RAN tasks, even among normally developing children. In addition, the study found that RAN performance had a mediating effect in the relationship between inattention and reading fluency (Pham, Fine, & Semrud-Clikeman, 2011).

Based on repeated reports that children with ADHD perform more slowly on RAN tasks than controls (Tannock et al., 2000; Waber, Wolff, Forbes, & Weiler, 2000), it has recently been proposed that RAN performance involves inhibition, particularly interference control processes (Bexkens, van den Wildenberg, & Tijms, 2015). Bexkens and colleagues (2015) examined whether interference control explained RAN performance of children with dyslexia, and whether those processes contributed to the association between RAN performance and reading skills in dyslexia. The results indicated that in addition to phonological processing and processing speed, interference control predicted rapid naming in dyslexia, however, interference control was not significantly associated with reading and spelling skills. After variance in reading and spelling associated with processing speed, interference control and phonological processing was partialled out, naming speed was no longer consistently associated with the reading and spelling skills of children with dyslexia. However, this finding is to be expected. Rapid naming tasks are thought to include a variety of speeded processes involving verbal, visual and motor systems (Arnett et al., 2012), and although measures of nonverbal processing speed (e.g., WISC-R Coding) and measures of verbal naming speed (e.g., RAN) load on separate factors in some samples, these constructs are highly correlated (e.g., r = .77; McGrath et al., 2011). Finally, dyslexic children were found to differ from normal readers on naming speed, literacy skills, phonological processing and processing speed, but not on inhibition processes.
Evidence that RAN deficits are shared between children with dyslexia and ADHD has also been obtained. Shanahan and colleagues (2006) examined a broad range of speeded tasks in groups with dyslexia, ADHD and co-occurring dyslexia and ADHD. The study found a significant interaction between dyslexia and ADHD that was under additive. The deficits observed in the co-occurring group were less severe than the sum of the deficits in each single deficit group, leading to the conclusion that the processing speed deficits in dyslexia and ADHD were partially shared. In addition, a recent study that examined the associations between the inattentive symptom domain of ADHD and RAN found a reciprocal predictive relationship between the individual differences in RAN (letters, digits, objects & colour) and AD in early childhood (Arnett et al., 2012). The authors concluded that inattentive behaviour leads to slower performance on RAN tasks, with slower RAN speed predictive of worse inattention. However, when testing the MDM of dyslexia and ADHD, McGrath and colleagues (2011) found that RAN was a unique predictor of dyslexia. In addition, no evidence of an association has been found between naming speed and ADHD in those studies that have controlled for reading skills (Raberger & Wimmer, 2003; Semrud-Clikeman et al., 2000), suggesting that slow naming speed could be unique to dyslexia.

**Interference Control**

The research findings are more variable for ADHD than dyslexia, and there is less agreement regarding the core neuropsychological deficits underlying the disorder (Andreou et al., 2007). A number of cognitive theories attribute impairments in executive functions (EF) as the core deficit in ADHD, and interference control (IC) is thought to play a fundamental role in these higher order processes (Barkley, 1997; Pennington & Ozonoff, 1996; van Mourik, Oosterlaan, & Sergeant, 2005). The construct has received consideration in the literature from theorists attempting to explain the behavioural symptoms of ADHD within a model of attention (e.g., Berger & Posner, 2000; Swanson et al., 1998). Posner and
Petersen (1990) describe three neural networks of attention, including the alerting network (i.e., the achievement and maintenance of an optimally alert attentional state), the orienting network (i.e., the movement of visual attention in space), and the executive network (Posner & Petersen, 1990). The executive network is associated with the control of goal-directed behaviour, inhibition, and conflict resolution (Berger & Posner, 2000). Interference control is a key function of the executive network (Posner & DiGirolamo, 1998), and an impairment in this domain is thought to result in (a) an inability to filter conflicting, irrelevant, or distracting information (Lansbergen, Kenemans, & Van Engeland, 2007; van Mourik et al., 2005), and (b) an inability to inhibit automatic response tendencies (van Mourik et al., 2009).

Stroop style tasks are often used as a measure of processing speed and IC and are thought to elicit conflict between an automatic response and a more controlled action. Numerous studies using variations of the Stroop Colour-Word task (Stroop, 1935), have found response times to be slower in individuals with ADHD, compared to controls (Derefinko et al., 2008; Fallgatter et al., 2004; Homack & Riccio, 2004), suggesting children with ADHD are more distracted by irrelevant and/or conflicting information than control children. However, findings are conflicting, and a number of meta-analyses report discrepant effect sizes on the IC score, when comparing groups with ADHD and controls, leading to general disagreement as to whether IC is compromised in individuals with the disorder (Frazier, Demaree, & Youngstrom, 2004; Homack & Riccio, 2004; Lansbergen et al., 2007; van Mourik et al., 2005). Some researchers have proposed that these discrepancies reflect methodological differences, as well as measurement limitations associated with the Stroop task (Del’Homme et al., 2007; Lansbergen et al., 2007). Consequently, the use of other methodologies other than the Stroop design has been suggested (van Mourik et al., 2009). An alternative experimental task used to quantify IC in children with ADHD in the current cognitive literature is the Eriksen Flanker task (Eriksen & Eriksen, 1974).
The Flanker task is a nonverbal computerized task offering a number of advantages for the study of interference control in children with reading and attention deficits (Mullane, Corkum, Klein, & McLaughlin, 2009). The computerized task is theoretically driven and quantifies interference effects in terms of response time and accuracy, allowing the control and manipulation of task and measurement variables. There are many variants of the original Flanker paradigm, however the basic effect involves the need to respond to stimuli "flanked" by irrelevant stimuli, and it is the irrelevant stimuli which affect the response (see p.162 for a detailed description of the Flanker Task used in this study). Various studies have observed that children with ADHD make more errors, or are significantly slower on the incongruent condition (e.g., flanker items require the opposite response of the target and are represented by different symbols) compared with the congruent condition (e.g., flankers call for the same response as the target, and may be identical) on the Flanker task (Crone, Richard Jennings, & Van Der Molen, 2003; Jonkman et al., 1999; Scheres et al., 2004; van Meel, Heslenfeld, Oosterlaan, & Sergeant, 2007). A review of studies that used reaction-time based interference control paradigms (e.g., the Eriksen Flanker task and the Simon task) in children with and without ADHD, found that children with ADHD were significantly more vulnerable to interference and additional processing demands during incongruent trials (Mullane et al., 2009).

There has also been some evidence to link interference control deficits to children with dyslexia (Berninger et al., 2006; Brosnan et al., 2002; Helland & Asbjørnsen, 2000; Kelly, Best, & Kirk, 1989; Purvis & Tannock, 2000; Reiter, Tucha, & Lange, 2005), and the Stroop Colour-Word paradigm has been commonly used to investigate IC in children with dyslexia, as well as ADHD. However, it has been suggested that the poor IC reported in children with dyslexia (Everatt, 1997; Helland & Asbjørnsen, 2000; Kapoula et al., 2010; Protopapas, Archonti, & Skaloumbakis, 2007) may be the consequence of poor reading skill.
For example, Wang and Gathercole (2015) found that the IC deficits of dyslexic readers on an incongruent Stroop colour-word task did not generalise to a corresponding nonverbal task that required matching of pictures with locations. The authors found that word reading ability was associated with Stroop interference in the poor readers, suggesting deficits on the Stroop task may be associated with task-specific aspects, rather than generalised interference impairment in children with reading difficulties (Wang & Gathercole, 2015).

**Potential shared cognitive deficits between dyslexia and ADHD**

**Working Memory**

Working memory is a multi-component cognitive system of limited-capacity that allows us to hold and manipulate information “on-line” for a few seconds, (Baddeley, 1996; Miyake & Shah, 1999). Working memory is thought to underlie a wide range of EF processes (Roberts, Hager, & Heron, 1994; Roberts & Pennington, 1996), and allows us to focus attention and resist distractors. It also assists during complex cognitive activities such as reasoning and reading comprehension (Baddeley, 2003; Daneman & Merikle, 1996; de Fockert, Rees, Frith, & Lavie, 2004; Gathercole & Pickering, 2000; Swanson, 1999).

An extensive body of research has examined working memory deficits in individuals with dyslexia (e.g., Gathercole, Alloway, et al., 2006; Gathercole, Lamont, & Alloway, 2006; Siegel & Ryan, 1989), and results have shown that working memory plays an important role in the development of reading skills (Moura et al., 2015). For example, it has been shown that in addition to phonological processing skills, working memory contributes to reading ability (Kibby, Lee, & Dyer, 2014), and many studies have demonstrated that better reading skills (word reading and comprehension) are associated with better working memory (Christopher et al., 2012; de Jong, 1998; Gathercole, Lamont, et al., 2006; Kibby & Cohen, 2008; Martinussen & Tannock, 2006; Marzocchi et al., 2008; Sesma, Mahone, Levine, Eason, & Cutting, 2009; Swanson et al., 2009; Swanson, Volkow, et al., 2006; Vellutino et
Further, the reading comprehension skills of some children with dyslexia has been found to deteriorate when the processing demands of a task exceed their limited working memory capacity (Berninger et al., 2006; Swanson, Howard, & Saez, 2006; Vellutino et al., 2004). However, other studies examining children with dyslexia have found no working memory difficulties, while others have noted verbal only, or both verbal and visual-spatial working memory impairments (Kibby, Marks, Morgan, & Long, 2004; Savage, Lavers, & Pillay, 2007; Swanson, Ashbaker, & Lee, 1996).

Attentional control and working memory rely on a common set of neural structures, (Burgess et al., 2010) and it has been suggested that working memory impairments may account for the attention deficits seen in ADHD (Burgess et al., 2010; Kofler, Rapport, Bolden, Sarver, & Raiker, 2010). For example, studies that have separated the symptom domains of ADHD have found that working memory impairments are more strongly associated with the inattentive symptom dimension than with the hyperactive/impulsive dimension (de Jong, 1998; Gathercole, Alloway, et al., 2006; Martinussen & Tannock, 2006; Swanson, Howard, et al., 2006). While a substantial amount of research supports verbal as well as visual working memory deficits in ADHD (see Martinussen et al., 2005), research findings have been mixed. Some studies have reported significant differences between children with ADHD and typically developing controls, and others have failed to replicate these results (for a review, see Pennington & Ozonoff, 1996). For example, when examining the relationship between dyslexia, ADHD, and working memory, no evidence was found of working memory deficits in children with ADHD alone (van de Voorde et al., 2010). Instead, the study found that working memory deficits were associated with the groups with dyslexia, with or without ADHD, suggesting that working memory problems are associated with reading difficulties and not ADHD. Other researchers have found an additive effect, where greater working memory deficits were associated with co-occurring ADHD and
dyslexia than for ADHD or dyslexia alone (Katz et al., 2011). Methodological confounds have been reported to contribute to these incongruent findings (Rapport et al., 2008), and these were addressed in a number of meta-analytic reviews that appear to support working memory deficits in children with ADHD relative to typically developing controls (Martinussen et al., 2005; Willcutt, Doyle, et al., 2005) (Willcutt et al. 2005).

**Reading Comprehension**

Reading comprehension (RC) is a complex function that relies on different cognitive processes, and reading abilities across the life span (Ghelani et al., 2004), and has been described as the joint product of oral language comprehension and word decoding. According to this view, children with dyslexia and those with SLI should both exhibit deficits in reading comprehension, but their profiles or causes may be different (Wong et al., 2017). Each of these variables (word decoding and oral language comprehension) have been found to explain unique variance in RC (e.g., Chen & Vellutino, 1997; Gough, Hoover, & Peterson, 1996), however, some studies have shown that a considerable amount of variance remains unexplained (e.g., Cutting & Scarborough, 2006; Keenan, Betjemann, & Olson, 2008), and additional skills such as reading fluency (Adlof, Catts, & Little, 2006; Cain et al., 2015) and verbal working memory (Cain, 2006) have been proposed. Working memory (WM) significantly predicts RC in typically developing children (Oakhill, Cain, & Bryant, 2003; Swanson & Howell, 2001), and is essential in the processing and integration of meanings, and for the ability to construct mental representations during reading comprehension (Rawson & Kintsch, 2005). Moreover, WM explains variance in RC even after controlling for general cognitive ability, word decoding and vocabulary (Cain, Oakhill, & Bryant, 2004). However, there are differing views on the role of WM in RC impairments (Ricketts, 2011). For example, Cain (2006) proposed that WM deficits subvert higher-level language skills such as inference and comprehension monitoring, resulting in poor RC, whereas other
researchers have proposed that oral language deficits cause problems in both WM and RC (Hulme & Snowling, 2011; Nation, Adams, Bowyer-Crane, & Snowling, 1999).

Dyslexia and ADHD have each been associated with poor reading comprehension. Most studies investigating the reading comprehension of individuals with dyslexia have focussed on underlying phonological and word recognition deficits (Schiff, Schwartz-Nahshon, & Nagar, 2011). Phonological processing deficits are believed to result in difficulties in phonemic decoding (Blau, van Atteveldt, Ekkebus, Goebel, & Blomert, 2009), which in turn, impact word identification performance and subsequent reading comprehension (Blachman, 2000; Snowling, 2000; Stanovich, 1991; Vellutino, Scanlon, Small, & Tanzman, 1991; Vellutino, Scanlon, & Tanzman, 1994). Generally, comprehensions skills develop alongside decoding skills in typically developing readers (Nation & Snowling, 1998), so when comprehension difficulties occur in dyslexia they are frequently attributed to a ‘bottleneck’ at the word level, due to a resource limitation for comprehending text (LaBerge & Samuels, 1974; Perfetti, 1985).

Attentional mechanisms have also been hypothesized to play an important role in reading comprehension abilities (Shaywitz & Shaywitz, 2008), and some research has found a significant association between measures of sustained attention and reading comprehension, suggesting that attention deficits in ADHD may have a direct impact on reading comprehension (Stern & Shalev, 2013). For example, an early study investigating reading comprehension difficulties in children with ADHD, without co-occurring language impairments, found that comprehension declined as the length of the passage increased (Cherkes-Julkowski, 1995). Similarly, children with ADHD were more impaired when reporting main ideas from passages than typical controls, despite average word reading accuracy and word reading rate (Brock & Knapp, 1996), suggesting longer passages placed
greater demands on attentional capacity for the group with ADHD, and may be the result of working memory difficulties (Miller et al., 2013).

Given the significant co-occurrence of dyslexia in children diagnosed with ADHD (Dykman & Ackerman, 1991; Gilger, Pennington, & DeFries, 1992; Shaywitz et al., 1995; Willcutt & Pennington, 2000), it has also been suggested that deficits in EF functions may contribute to difficulties in reading comprehension found in individuals with ADHD (Locascio, Mahone, Eason, & Cutting, 2010). Reading comprehension inherently requires more complex processes than basic phonological decoding and word identification. For example, identifying words and converting them into meaningful information involves multiple, interacting working memory processes, and deficiencies in working memory are commonly associated with reading comprehension difficulties in children with and without ADHD (Savage et al., 2007; Swanson & Alloway, 2012). Further, other EF processes such as organisation, planning (Chiarenza, 1990; Sesma et al., 2009), and inhibition (Savage, Cornish, Manly, & Hollis, 2006) have been associated with deficits in reading comprehension. It has been proposed that interference control within working memory is integral to reading and reading comprehension, particularly in the context of interference from distractors (Brosnan et al., 2002), and it has been suggested that the reading comprehension deficits in children with ADHD (without word reading difficulties) are the result of these ADHD related EF deficits (Brock & Knapp, 1996; McInnes, Humphries, Hogg-Johnson, & Tannock, 2003).

Early research findings that supported single deficit explanations of dyslexia and ADHD alone influenced the theoretical explanations of the co-occurring condition. However, these early comorbid explanations were either unsupported, or results have generally been inconclusive. Current research findings now emphasise the complex and multifactorial nature of neurodevelopmental disorders, and highlight the substantial overlap
between disorders, as well as the numerous shared neurocognitive deficits found to be associated with each (Moura et al., 2017). This has resulted in the proposal of a multifactorial framework (McGrath et al., 2011; Pennington, 2006; Pennington et al., 2012) designed to identify cognitive deficits that may be unique to a disorder, as well those that are shared, and which together may contribute to the explanation of the co-occurrence between them.

The research finding for the association between dyslexia and ADHD are variable. In order to establish which variables are unique or common to the disorders the current study was designed to replicate previous research that has found (a) poor phonological decoding is associated with dyslexia only, (b) poor interference control is associated with ADHD only, and (c) that poor working memory, slow rapid naming and poor reading comprehension are deficits common to both dyslexia and ADHD, and which explain the co-occurrence between the disorders. This study also examined the AD symptom dimension separately, and the unique impact of AD has not previously been investigated within this framework. Further, the current research literature argues that dyslexia and ADHD should be looked at as different points on a normal distribution. This study was unique using continuous variables rather than categorical groups with an arbitrary cut-off that ignores children with subclinical symptoms. Most studies have used categorical groupings which assumes the mean is an accurate representation of a group’s performance, regardless of any large variations between each individual’s actual performance. The use of continuous variables, instead of categorical groups is more precise, and provides an accurate representation of individual performances, as it uses each child’s individual score.

6.2 Aims and Hypotheses

Based on the findings of prior research the present study had two aims. First, to examine the extent that difficulties with phonological processing and rapid automatic naming
are associated with dyslexia only, and to examine whether difficulties with interference control are associated with ADHD and AD only. Second, using the framework of the Multiple Deficit Model, we aimed to determine the extent to which difficulties with working memory and reading comprehension are shared by the dyslexia, ADHD and AD groups.

Three hypotheses were proposed. First, that there would be a significant association between reading fluency, phonological processing and RAN. Children with poorer reading fluency would perform more poorly on phonological processing and RAN measures regardless of symptoms of ADHD or AD. Second, it was expected that children with ADHD or AD (with or without dyslexia) would make more errors and have slower response times on the incongruent interference control condition than the other groups. Third, that children with more symptoms of dyslexia, ADHD or AD would be more impaired on the working memory and reading comprehension tasks than those with fewer symptoms. Children with very severe symptoms would not differ from one another because of the shared nature of the deficit.

6.3 Method

**Participants and Group Classification**

The same 72 children who participated in previous studies constituted the sample for the current study. A description of the participants (chapter 2), and the classification measures (chapter 3), has been described in detail in these previous chapters.

**Measures**

**Phonological Decoding**

The participant’s ability to use phonological information to decode words was assessed with the pseudoword decoding (phonetic word attack) subtest from the WIAT-II (Wechsler, 2007). The performance index for this test was the standard score for the total number of non-words correctly identified.
Rapid Automatic Naming (RAN)

The current study used RAN (Denckla & Rudel, 1976) letters only. The task required oral naming of rows of continuous high frequency lowercase alphabet letters (i.e., a, d, o, p, and s). At the beginning of the test participants familiarised themselves with an untimed practice trial using stimuli identical to the test stimuli. Participants were asked to name the letters as quickly as possible following presentation without making mistakes. There was one practice and one trial for the letter stimulus (consisting of 10 x 5 five rows of stimuli). Performance was taken as the time to correctly name the entire 10 x 5 rows (response time: RT) measured with a digital stopwatch. Test retest reliability is 0.90 for letters.

Interference control

The Eriksen Flanker task (Eriksen & Eriksen, 1974) was used to measure interference control. The Flanker task is a nonverbal computerized task that has been found to offer several advantages for the study of interference control in children with reading and attention deficits (Mullane et al., 2009). Similar to the measure used by Shalev and Tsal (2003), this study used a version of the Flanker task in which participants must identify as quickly as possible a single target stimulus (a square or a triangle). The target appeared alone, or was flanked by two distractors, which required participants to ignore the peripheral stimuli (distractors) appearing in the same display. The distractors were either congruent (i.e., identical to the target), incongruent (i.e., belonged to the opposite response category, such as a central square flanked by two triangles), or neutral (i.e., two plus signs). Manipulating the identity of the distracters provided a sensitive measure of the participants ability to selectively focus attention while ignoring irrelevant information.

There were 200 trials in total, with 4 x blocks of 50 trials presented in counterbalanced order (ABBA). In each block, there was an initial 10 trial practice, followed by the 40-trial test. The test was presented on a Sony VAIO laptop computer, with
a 34cm x 19cm screen. The viewing distance was 50 cm, so that each centimetre represented 1.15° of visual angle. The target stimulus was a square or a triangle subtending 0.6° × 0.6° of visual angle. All stimuli were white and appeared on a dark background. Procedure: First, a fixation sign (#) was presented for 1 second. Then, following a 300-ms interval, the target stimulus, with or without flankers, was presented and remained on the screen until a response was made. In Experiment 1A (Blocks 1 and 4), the flankers appeared in close proximity (0.95°) to the target item. There were 25 baseline, 25 congruent, 25 incongruent and 25 neutral conditions that appeared in random order. In Experiment 1B (Blocks 2 and 3), the flankers appeared far (5.7°) from the target. Again, there were 25 baseline, 25 congruent, 25 incongruent and 25 neutral conditions that appeared in random order. At the beginning of each block of trials the participants received the following instructions on the computer screen: “When the trial begins you will see a “#” in the middle of the screen. Then you will see either a triangle or a square in the middle of the screen. When you see a triangle, you must press the Δ on the keyboard, and when you see a square you must press the □ on the keyboard. (The triangle corresponded to the letter F on the keyboard and the square corresponded to the letter J on the keyboard). You must do this as quickly as possible without making mistakes”. The participants pressed the space bar to initiate the start of each trial. The dependent variables were mean correct response time (RT: in milliseconds) and accuracy rates (% correct). Only close proximity conditions were used in the current analyses.

Potential Shared Variables

Reading Comprehension

The reading comprehension subtest from the WIAT-II (Wechsler, 2007) was used to assess participant’s ability to comprehend written text and provides a measure of textual comprehension and samples a wide range of comprehension skills. Written comprehension is
an important component to investigate in children with dyslexia and ADHD. Comprehension is the functional outcome of reading, and in this case, is a measure of what children can do with the skills that they have. A number of years ago reading comprehension measures were not included in many studies. However, recent research has highlighted reading comprehension as an important construct, as it is viewed as the joint product of oral language comprehension and word decoding. Dyslexia and ADHD have each been associated with poor reading comprehension and for these reasons the WIAT-II reading comprehension subtest was included in this study. The WIAT-II provides Australian norms and has strong psychometric properties. To increase ecological validity, children are able (without prompt) to look back at the passage for the answers to the comprehension questions. The performance index was an overall standard score for the reading comprehension and target words raw scores for correct answers ($r = > 0.95$ for test-retest reliability).

*Working Memory*

The Digit Span Forward and Backward, and Letter-Number Sequencing subtests from the WISC-IV were used as a measure of working memory. In the Digit Span subtest children are asked to repeat verbally presented digit strings, both forward and backward. Letter-Number Sequencing requires the child to read a sequence of numbers and letters and then to recall the numbers in ascending order and the letters in alphabetical order. The standard scores for each subtest were combined to form a working memory index (WMI). In addition, the Language Memory Index (LMI) from the CELF–4 was used as an additional measure of working memory and is designed to assess memory dependent language tasks. The LMI is a cumulative measure of a child’s performance on three subtests: Concepts and Following Directions which measures the ability to follow oral directions of increased procedural and linguistic complexity, Recalling Sentences which measures the ability to repeat sentences increasing in syntactic complexity, and Formulated Sentences, which requires the use of
certain words and phrases in full sentences. The WMI and LMI have a mean of 100 and a standard deviation of 15. A score of 100 on these scales represents the performance of the typical student of a given age. A child with a standard score of 85 or less was considered clinically impaired in that memory domain.

6.4 Procedure

The procedure was described in detail in Chapter 2. For the current study the test order was as follows: the WISC-IV subtests (digit span forwards and backwards, letter-number sequencing), the WIAT-II subtests (written comprehension, pseudoword decoding), the CELF-4 subtests (concepts and following directions, recalling sentences, formulated sentences, word classes 2) the TOWRE-2 (SWE and PWE), RAN (letters), and finally the Flanker task.

Analysis strategy

Two mixed factorial ANOVAs were conducted to assess the performance of the groups on the interference control task. In the first analysis a 2 (dyslexia, no dyslexia) × 2 (ADHD, no ADHD) × 2 (task: congruent or incongruent) analysis was conducted for each the mean correct response time and percentage correct for the interference control task. In the second analyses, the AD group was used, instead of the ADHD group. Due to the nature of the DVs, group means were considered more appropriate for the analysis, rather than the continuous variable approach used in the other analyses. Different metrics were used to measure the constructs, making them incompatible. All measures satisfied the assumptions of ANOVA.

Based on the continuous nature of each of the reading and attention variables, moderated regression analyses were conducted using reading fluency scores and either ADHD or AD scores, to assess the contribution of phonological decoding, as well as all variables potentially shared between reading and attention difficulties. All assumptions of
the regression analyses were met. When moderated regression was conducted, the continuous reading, AD and ADHD variables were mean centred.

6.5 Results

Interference Control

There were 6 children in the sample who failed to complete the interference control task. For the mean correct response time variable, any individual trial in which the response time exceeded three seconds were excluded from the calculation of the mean score.

ADHD and Dyslexia

For trials on the Interference Control task, there were 21 children in the group with ADHD, and 45 without ADHD. The group with dyslexia consisted of 32 children, and there were 34 in the group without dyslexia.

Analysis of the percentage correct responses produced two significant main effects, those of condition, $F(1, 62) = 21.21, p < .001, \eta^2_p = .25$ and ADHD group, $F(1, 62) = 4.81, p = .032, \eta^2_p = .07$. Significantly more errors were made in the incongruent ($M = 89.76\%, SD = 8.7\%$) than in the congruent condition ($M = 95.40\%, SD = 6.11\%$). The group with ADHD made significantly more errors ($M = 90.92\%, SD = 7.74\%$) than children without ADHD ($M = 96.33\%, SD = 5.16\%$). These findings are partially consistent with the hypothesis. Although it was expected that the ADHD group would be less accurate across the incongruent tasks only, more errors were made by this group across both the congruent and incongruent conditions. As expected, there were no significant accuracy effects concerning the group with dyslexia.

When the mean correct response time was used as the DV, there was a significant effect of condition, $F(1, 62) = 23.84, p < .001, \eta^2_p = .28$, and a significant interaction between the dyslexia group and condition, $F(1, 62) = 4.07, p = .048, \eta^2_p = .06$ (see Figure 6.1). The mean response time for both groups (dyslexia, no dyslexia) was significantly
longer for the incongruent than for the congruent condition and is consistent with the significant main effect of condition. The mean response time for the group with dyslexia was significantly longer than that for the group without dyslexia in the incongruent condition, $F(1, 62) = 4.22, p = .044, \eta_p^2 = .06$, and the response time for the groups with and without dyslexia was not different in the congruent condition, $F(1, 62) = 2.03, p = .159, \eta_p^2 = .03$. These results fail to support the hypothesis that the group with ADHD would have significantly slower response times in the incongruent condition than the other groups. In contrast to our prediction, it was the group with dyslexia who was significantly slower on this task condition.

![Figure 6.1](image.png)  

*Figure 6.1* The interaction between the groups with ($n = 32$) and without ($n = 34$) dyslexia and the congruent and incongruent conditions. Error bars represent ± 1 standard error.

**AD and Dyslexia**

For the Interference Control task, there were 42 children in the group with AD, and 24 children without AD. Consistent with the analysis for ADHD, there were 32 children with dyslexia, and 34 without dyslexia. Analysis of the percentage correct responses produced a
significant main effect of condition, $F(1, 62) = 21.21, p < .001, \eta^2_p = .25$ and a significant interaction between AD group and condition, $F(1, 62) = 4.67, p = .035, \eta^2_p = .07$ (see Figure 6.2). The group with AD made significantly more errors in the incongruent than in the congruent condition, $F(1, 62) = 23.92, p < .001, \eta^2_p = .27$, supporting the hypothesis. The group without AD did not differ on the proportion of errors made in the congruent and incongruent conditions, $F(1, 62) = 0.91, p = .345, \eta^2_p = .01$. As predicted there were no significant effects concerning the group with dyslexia.

![Figure 6.2](image_url) The interaction between the groups with ($n = 42$) and without ($n = 24$) AD and the congruent and incongruent conditions. Error bars represent ± 1 standard error.

When mean correct response time was used as the DV, there was a significant main effect found for AD Group, $F(1, 62) = 4.06, p = .048, \eta^2_p = .06$. The mean correct response time for the group with AD ($M = 1166.3$ ms, $SD = 472.9$ ms) was significantly longer than that found for the group without AD ($M = 965.8$ ms, $SD = 383.8$ ms). There was a significant main effect found for condition, $F(1, 62) = 25.50, p < .001, \eta^2_p = .29$, and consistent with the
analysis of mean correct response time for the ADHD and dyslexia groups, there was a significant interaction between the dyslexia group and condition, $F(1, 62) = 4.51, p = .038, \eta_p^2 = .07$ (see Figure 6.3). While the groups with and without dyslexia took significantly longer to respond to the incongruent than the congruent condition, the group with dyslexia took significantly longer than the group without dyslexia in the incongruent condition, $F(1, 62) = 4.34, p = .041, \eta_p^2 = .06$, with no significant differences found in the congruent condition, $F(1, 62) = 1.85, p = .179, \eta_p^2 = .03$. The finding that the AD group performed significantly slower on the incongruent condition than the congruent condition is consistent with the hypothesis, however contrary to the prediction the group with dyslexia was also found to have a slower response time on this task.

**Figure 6.3** The interaction between the groups with ($n = 32$) and without ($n = 34$) dyslexia and the congruent and incongruent conditions. Error bars represent $\pm 1$ standard error.

*Note.* Figures 6.1 and 6.3 are the same and are presented to emphasise the independence of AD and ADHD from dyslexia. There were two different analyses conducted. In the first, the presence or absence of ADHD and the presence or absence of dyslexia were evaluated.
In the second analysis the presence or absence of AD and the presence or absence of dyslexia were evaluated. In each case the DV was interference control. For both sets of analyses the same sample of children with dyslexia were used. What was different was the ADHD and AD groupings. The results of each analysis showed that there was no significant interaction between the groups with ADHD and dyslexia, or those with AD and dyslexia. In the analysis using the ADHD group, there was no association between interference control and the presence or absence of ADHD. When AD alone was examined, an association was found. However, in both cases this was independent of the presence or absence of dyslexia. The figure was presented twice to emphasise these findings, and dyslexia in terms of the incongruency effect.

Due to the large number of children in the sample with sub-clinical symptoms of dyslexia, ADHD or AD, multiple regression and moderated regression analyses were used to examine the extent to which phonological decoding, working memory, rapid automatic naming and reading comprehension were cognitive abilities that could occur in children with dyslexia, ADHD, AD, or a combination of both. Consistent with Pennington et al. (2012) reading fluency was measured using the combined sight word efficiency, and phonemic decoding efficiency scores from the TOWRE-2. The means for both the attention and hyperactivity subscales of the Connors-3 were used for each child for the analyses using ADHD. The AD measure used the score for the attention component of the Connors-3.

**Associations between Variables**

Based on the Pearson correlations (see Table 6.1), there were no significant linear associations found between the measures of ADHD, AD, phonological decoding, rapid naming, or the measures of working memory used. Better reading fluency was associated with better working memory, rapid naming, reading comprehension and pseudoword decoding. Better working memory, particularly the language memory index, was associated with better reading comprehension and pseudoword decoding.
These correlations are consistent with the results expected for reading fluency, but not for either measure of attention deficit disorder. This could have occurred because the interaction between ADHD + dyslexia and AD + dyslexia on the RAN, pseudoword decoding, reading comprehension and working memory measures was only present for children with better reading skills. If children had poor reading skills, there was no significant association between symptoms of ADHD or AD and these measures. However, for more skilled readers for RAN and working memory measures, the association between reading skills and RAN, and reading skills and working memory was not significant for children with high symptoms of either AD or ADHD.

Phonological decoding in dyslexia, AD and ADHD

Moderated regression analyses were conducted to determine whether children with ADHD or AD, as well as dyslexia, had increased difficulties with phonological decoding. There was no significant impact of the moderator AD and reading fluency, $F(1, 68) = 0.92, p = .763$, or ADHD and reading fluency, $F(1, 68) = 0.05, p = .860$. Subsequent linear regression analyses showed that 48.7% of the variance in phonological decoding was explained by symptoms of ADHD and reading fluency, $F(2, 69) = 32.82, p < .001$, and symptoms of AD and reading fluency, $F(2, 69) = 32.69, p < .001$. In both analyses, reading fluency made a significant unique contribution to the explanation of phonological decoding skills ($\beta_{\text{ADHD analysis}} = .703, p < .001$; $\beta_{\text{AD analysis}} = .697, p < .001$). Neither symptoms of ADHD, $\beta = -.035, p = .693$, or AD, $\beta = -.014, p = .869$ made a significant unique contribution to the explanation. These findings support the hypothesis that impairments in phonological decoding would be associated with dyslexia only.
Table 6.1

Descriptive statistics and correlations for AD, ADHD, reading fluency and variables expected to share variance (N = 72)

<table>
<thead>
<tr>
<th></th>
<th>Mean (SD)</th>
<th>ADHD</th>
<th>AD</th>
<th>WMI</th>
<th>LMI</th>
<th>RAN</th>
<th>Comp</th>
<th>Pseudo word</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reading fluency</td>
<td>87.70 (11.25)</td>
<td>.168</td>
<td>-0.11</td>
<td>.436***</td>
<td>.374***</td>
<td>.654***</td>
<td>.344**</td>
<td>.697***</td>
</tr>
<tr>
<td>ADHD T-score</td>
<td>64.86 (10.02)</td>
<td>-</td>
<td>0.892***</td>
<td>-0.074</td>
<td>-0.031</td>
<td>0.042</td>
<td>-0.048</td>
<td>-0.084</td>
</tr>
<tr>
<td>AD T-Score</td>
<td>68.26 (10.90)</td>
<td>-</td>
<td>-0.133</td>
<td>-0.099</td>
<td>-0.122</td>
<td>-0.123</td>
<td>-0.022</td>
<td></td>
</tr>
<tr>
<td>Working Memory Index (WMI)</td>
<td>96.63 (9.6)</td>
<td>-</td>
<td>.572***</td>
<td>.437***</td>
<td>.295**</td>
<td>.452***</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Language Memory Index (LMI)</td>
<td>102.3 (12.2)</td>
<td>-</td>
<td>0.342**</td>
<td>.700***</td>
<td>.407***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RAN</td>
<td>93.93 (9.4)</td>
<td>-</td>
<td>0.334***</td>
<td>.503***</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Comprehension (COM)</td>
<td>96.31 (12.1)</td>
<td>-</td>
<td>-</td>
<td>0.512***</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pseudoword</td>
<td>95.17 (10.83)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

p < .01**; p < .001***. With the exception of AD and ADHD scores, all are presented as standard scores

Potential variables with shared variance between dyslexia, ADHD and AD

Dyslexia and ADHD

Working Memory Index, ADHD and Reading Fluency

To determine how symptoms of ADHD influence the association between reading fluency and working memory, the working memory index was used as the DV, reading fluency as the IV, and ADHD score as the moderator. The interaction between ADHD and dyslexia was significant, $F(1, 68) = 8.23, p = .005$, accounting for 8.5% of the unique variance (see Table 6.2).
Table 6.2

*Influence of Reading Fluency and ADHD symptoms on Working Memory Index (N = 72).*

<table>
<thead>
<tr>
<th>Variables</th>
<th>B</th>
<th>SE (B)</th>
<th>β</th>
<th>Sr</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Working Memory</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$R^2 = 29.8%$, $F(3, 68) = 9.61, p &lt; .001$</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reading Fluency</td>
<td>-.359</td>
<td>.278</td>
<td>-.419</td>
<td>-.131</td>
</tr>
<tr>
<td>ADHD T-Score</td>
<td>-.080</td>
<td>.087</td>
<td>-.084</td>
<td>-.080</td>
</tr>
<tr>
<td>Reading Fluency*ADHD</td>
<td>-.026</td>
<td>.009</td>
<td>-.919</td>
<td>-.291**</td>
</tr>
</tbody>
</table>

| **Language Memory Index**  |     |        |      |     |
| $R^2 =20.4\%$,$F(3, 68) = 5.81, p = .001$ |     |        |      |     |
| Reading Fluency            | -.346 | .376   | -.318 | -.100 |
| ADHD T-score               | -.051 | .138   | -.042 | -.040 |
| Reading fluency*ADHD       | -.027 | .012   | -.739 | -.234* |

Note. *** $p < .001$, ** $p < .01$, * $p < .05$

The simple slopes analysis showed that there was a significant association between reading fluency scores and the working memory index when symptoms of ADHD were in the normal range, $t(68) = 4.95, p < .001$. As reading scores improved the working memory index improved. When clinical symptoms of ADHD were very high, the association between reading skills and working memory was much weaker, $t(68) = 2.00, p = .049$. These results suggest that when symptoms of ADHD are very high, better reading scores are not associated with improved working memory (see Figure 6.4). As predicted difficulties with both dyslexia and ADHD influence working memory performance.
When the language memory index was used as the DV, reading fluency as the IV and ADHD score as the moderator, the interaction between ADHD and dyslexia was significant, $F(1, 68) = 4.69$, $p = .034$, accounting for 5.5% of unique variance (see Table 6.2). The simple slopes analysis showed that there was a significant association between reading fluency score and the language memory index when symptoms of ADHD were in the normal range $t(68) = 3.84$, $p < .001$. As reading scores improved the language memory index improved. When clinical symptoms of ADHD were very high, there was no significant association between reading skills and the language memory index, $t(68) = 1.65$, $p = .103$. These results indicate that when symptoms of ADHD are very high, better reading fluency is not associated with an increase in language memory index scores (see Figure 6.5). As predicted, difficulties with both disorders impact this memory domain.

*Figure 6.4* Interaction between working memory index, ADHD and reading fluency ($N = 72$).

*Language Memory Index, ADHD and Reading Fluency*

When the language memory index was used as the DV, reading fluency as the IV and ADHD score as the moderator, the interaction between ADHD and dyslexia was significant, $F(1, 68) = 4.69$, $p = .034$, accounting for 5.5% of unique variance (see Table 6.2). The simple slopes analysis showed that there was a significant association between reading fluency score and the language memory index when symptoms of ADHD were in the normal range $t(68) = 3.84$, $p < .001$. As reading scores improved the language memory index improved. When clinical symptoms of ADHD were very high, there was no significant association between reading skills and the language memory index, $t(68) = 1.65$, $p = .103$. These results indicate that when symptoms of ADHD are very high, better reading fluency is not associated with an increase in language memory index scores (see Figure 6.5). As predicted, difficulties with both disorders impact this memory domain.
Figure 6.5 Interaction between language memory index, ADHD and reading fluency ($N = 72$).

Rapid Naming, ADHD and Reading Fluency

When rapid letter naming (RAN) was used as the DV with the same reading and attention variables, the interaction between ADHD and dyslexia was not significant, $F(1, 68) = 3.12, p = .082$ (see Table 6.3). On this basis, a linear regression analysis was conducted using reading fluency and ADHD scores as IVs. There was 43.2% of the variance in RAN explained by reading fluency and ADHD scores, $F(2, 69) = 26.26, p < .001$. Better reading fluency was associated with higher RAN scores. There was no significant unique association between RAN scores and ADHD scores (see Table 6.3). These results support the hypothesis that RAN is not associated with symptoms of the combined ADHD subtype.

Reading Comprehension, ADHD and Reading Fluency

When Reading Comprehension was used as the DV, with the same reading and attention variables, the interaction between ADHD and dyslexia was not significant, $F(1, 68) = 1.77, p = .188$. Linear regression analysis was conducted excluding the interaction term and accounted for 13% of the variance in reading fluency and ADHD, $F(2, 69) = 5.15, p =$
.008 (see Table 6.3). When controlling for ADHD, as reading fluency increased so did reading comprehension. There was no significant effect of ADHD on reading comprehension scores. These results fail to support the hypothesis that problems with reading comprehension would be associated with both dyslexia and ADHD.

Table 6.3

Influence of Reading Fluency and ADHD symptoms on RAN and reading comprehension (N = 72).

<table>
<thead>
<tr>
<th>Variables</th>
<th>B</th>
<th>SE (B)</th>
<th>β</th>
<th>Sr</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Rapid Letter Naming</strong> R² = 43.2%, F(2, 69) = 26.26, p &lt; .001</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reading Fluency</td>
<td>.558</td>
<td>.077</td>
<td>.666</td>
<td>.656***</td>
</tr>
<tr>
<td>ADHD T-Score</td>
<td>-.065</td>
<td>.087</td>
<td>-.069</td>
<td>-.068</td>
</tr>
<tr>
<td><strong>Reading Comprehension</strong> R² = 13% F(3, 68) = 5.15, p = .008</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reading Fluency</td>
<td>.390</td>
<td>.123</td>
<td>.362</td>
<td>.357**</td>
</tr>
<tr>
<td>ADHD T-score</td>
<td>-.132</td>
<td>.138</td>
<td>-.109</td>
<td>-.108</td>
</tr>
</tbody>
</table>

Note. *** p < .001, ** p < .01, * p < .05

Dyslexia and AD

Working Memory Index, AD and Reading Fluency

When the working memory index was used as the DV, reading fluency as the IV and AD score as the moderator, the interaction between AD and dyslexia was significant, F (1, 68) = 10.83, p = .003, and accounted for 10.9% of unique variance (see Table 6.4). The simple slopes analysis showed that there was a significant association between reading fluency score and the working memory index when symptoms of AD were in the normal range, t(68) = 5.30, p < .001. As reading score improved working memory index improved. When clinical symptoms of AD were very high, there was no significant association found between reading skills and working memory, t(68) = 1.08, p = .284, suggesting that when
symptoms of AD are very high improvements in reading skills are not associated with better working memory (see Figure 6.6). As predicted, difficulties with both disorders impact working memory capacity.

Table 6.4

Influence of Reading Fluency and AD symptoms on Working Memory Index (N = 72)

<table>
<thead>
<tr>
<th>Variables</th>
<th>B</th>
<th>SE (B)</th>
<th>β</th>
<th>Sr</th>
</tr>
</thead>
<tbody>
<tr>
<td>Working Memory Index $R^2 = 31.6%$, $F(3, 68) = 10.46$, $p &lt; .001$</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reading Fluency</td>
<td>.420</td>
<td>.090</td>
<td>.489</td>
<td>.48**</td>
</tr>
<tr>
<td>AD T-Score</td>
<td>-.074</td>
<td>.087</td>
<td>-.084</td>
<td>-.08</td>
</tr>
<tr>
<td>Reading Fluency*AD</td>
<td>-.027*</td>
<td>.008</td>
<td>-.337</td>
<td>-.33**</td>
</tr>
</tbody>
</table>

| Language Memory Index $R^2 = 22.4\%$, $F(3, 68) = 6.56$, $p = .001$ |       |        |      |     |
| Reading Fluency            | .456  | .118   | .418  | .41***|
| AD T-score                 | -.065 | .121   | -.058 | -.06 |
| Reading fluency*AD         | -.029 | .011   | -.281 | -.275*|

Note. *** $p < .001$, ** $p < .01$, * $p < .05$

Figure 6.6 Interaction between working memory index, AD and reading fluency ($N = 72$).
Language Memory Index, AD and Reading Fluency

When the language memory index was used as the DV, and the same reading and attention variables, the interaction between AD and dyslexia was significant, $F(1, 68) = 6.63$, $p = .012$, accounting for 7.6% unique variance (see Table 6.4). The simple slopes analysis showed that there was a significant association between reading fluency scores and the language memory index when symptoms of AD were in the normal range $t(68) = 4.21, p < .001$. As reading scores improved, language memory index improved. When clinical symptoms of AD were very high, there was no significant association found between reading skills and the language memory index, $t(68) = 0.92, p = .362$. These results suggest that when symptoms of AD are very high improvements in reading skills do not result in better working memory capacity (see Figure 6.7). As predicted, difficulties with both disorders impact working memory domains.

Figure 6.7 Interaction between language memory index, AD and reading fluency ($N = 72$).
Rapid Naming, AD and Reading Fluency

When rapid letter naming (RAN) was used as the DV, with the same reading and attention variables, the interaction between AD and dyslexia was significant, $F(1, 68) = 4.14$, $p = .046$, accounting for 3.2% unique variance (see Table 6.5). The simple slopes analysis showed that there was a significant association between reading fluency score and RAN when symptoms of AD were in the normal range, $t(68) = 6.29$, $p < .001$. As reading scores improved, rapid naming improved. When clinical symptoms of AD were very high there was also a significant association found between reading skills and RAN, $t(68) = 4.22$, $p < .001$. Inspection of Figure 6.8 shows that as reading fluency improves, RAN improves, but the slope is shallower than that found when AD symptoms are low. These results indicate a significant association between AD, reading fluency, and RAN.

Table 6.5
Influence of Reading Fluency and AD symptoms on RAN and Reading Comprehension ($N = 72$).

<table>
<thead>
<tr>
<th>Variables</th>
<th>B</th>
<th>SE (B)</th>
<th>β</th>
<th>Sr</th>
</tr>
</thead>
<tbody>
<tr>
<td>RAN $R^2 = 47.3%$, $F(3, 68) = 20.31$, $p &lt; .001$</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reading Fluency</td>
<td>.572</td>
<td>.075</td>
<td>.682</td>
<td>.673***</td>
</tr>
<tr>
<td>AD T-Score</td>
<td>-.078</td>
<td>.077</td>
<td>-.090</td>
<td>-.089</td>
</tr>
<tr>
<td>Reading Fluency*AD</td>
<td>-.014*</td>
<td>.007</td>
<td>-.183</td>
<td>-.179*</td>
</tr>
<tr>
<td>Reading Comprehension $R^2 = 22.4%$ $F(3, 68) = 6.56$, $p = .001$</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reading Fluency</td>
<td>.369</td>
<td>.121</td>
<td>.343</td>
<td>.343**</td>
</tr>
<tr>
<td>AD T-score</td>
<td>-.132</td>
<td>.125</td>
<td>-.119</td>
<td>-.119</td>
</tr>
<tr>
<td>Including Moderator</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reading fluency*AD</td>
<td>-.019</td>
<td>.011</td>
<td>-.193</td>
<td>-.188*</td>
</tr>
</tbody>
</table>
Figure 6.8 Interaction between RAN, AD and reading fluency (N = 72).

*Reading Comprehension, AD and Reading Fluency*

When Reading Comprehension was used as the DV, using the same reading and attention variables, the interaction between AD and dyslexia was not significant, $F(1, 68) = 2.90, p = .093$. Linear regression analysis was conducted excluding the interaction term and accounted for 13.2% of the variance in reading fluency and AD, $F(1, 69) = 5.27, p = .007$. When controlling for AD, as reading fluency increased so did reading comprehension. There was no significant effect of AD on reading comprehension scores. These results fail to support the hypothesis that problems with reading comprehension would be associated with both dyslexia and AD.
6.6 Summary of Results

**Phonological Decoding**

As predicted only the group with dyslexia had difficulties with phonological decoding and supports previous research that has found that ADHD or AD is not uniquely associated with poor phonological processing skills.

**Rapid Naming**

The hypothesis that slow naming speed would be uniquely associated with dyslexia was only partially met. The results indicated that symptoms of inattention influenced the association between reading fluency and rapid naming, although there was no additional effect over that of dyslexia. The symptoms of the combined ADHD subtype were not found to have a significant effect on the association between reading fluency and rapid naming.

**Interference Control**

It was hypothesised that the groups with ADHD and AD only would be slower and less accurate on the incongruent task condition of the interference control measure. The hypothesis was only partly supported. As predicted the group with AD was slower and made significantly more errors in the incongruent than in the congruent condition. However, the group with combined ADHD made more errors on both the congruent and incongruent conditions but were as quick to respond as the group without ADHD. In addition, contrary to our expectation, the group with dyslexia was significantly slower than the group without dyslexia on the incongruent condition, although there were no differences in accuracy between the groups with and without dyslexia.

**Working Memory**

As predicted, difficulties with working memory (WMI and LMI), were associated with dyslexia, ADHD and AD. The results indicated that severe symptoms of ADHD or AD
are associated with poorer working memory capacity in average readers, however, there is no additive effect when comorbid dyslexia and ADHD or AD is considered.

**Reading Comprehension**

The results indicated that reading fluency accounted for significant variance in reading comprehension and supports the hypothesis that reading comprehension would be impaired in the group with dyslexia. However, contrary to the hypothesis that impairments in reading comprehension would also be associated with ADHD and AD, no significant effect of ADHD or AD was found on reading comprehension scores.

**6.7 Discussion**

This study used a multiple deficit framework to compare groups with dyslexia and ADHD or AD on a battery of cognitive measures. The aim was to determine if the groups could be differentiated on measures of phonological decoding, rapid naming and interference control, and whether neurocognitive impairments in working memory and reading comprehension could explain the co-occurrence between dyslexia and ADHD or dyslexia and AD.

On the measure of phonological decoding, the group with dyslexia performed more poorly than the groups without dyslexia. This finding supported our hypothesis that a deficit in phonological decoding would be a unique predictor of dyslexia, and replicates other studies that have also found impairments in phonological decoding to be specific to dyslexia (Kroese, Hynd, Knight, Hiemenz, & Hall, 2000; McGrath et al., 2011; Pennington et al., 2012).

The hypothesis that the groups with ADHD and AD would be impaired on the measure of interference control was partially met. It had been predicted that both the ADHD and AD groups would make more errors and respond more slowly on the incongruent close proximity condition, than the other groups. This prediction was met for the group with AD,
however, the group with ADHD made more errors on both the congruent and incongruent condition but were as fast to respond on the incongruent condition as the group without ADHD. These findings contrast with studies that have found groups with combined ADHD make more errors on the incongruent task only, and have greater response latency compared to children without ADHD (e.g., Jonkman et al., 1999; Mullane et al., 2009), and may reflect the fact that previous studies have not separated the symptom domains of ADHD. In contrast to our hypothesis, the group with dyslexia was also slower in the incongruent Flanker condition, however, this result is consistent with a number of other studies that have found the involvement of cognitive interference in reading processes (Altemeier, Abbott, & Berninger, 2008; Savage et al., 2006). For example, a recent study found interference control predicts naming speed in children with dyslexia, although it was not found to be significantly associated with their reading and spelling skills (Bexkens et al., 2015). Other studies investigating interference control processes have found an association between RAN and the Stroop interference task, both in typical readers (Stringer et al., 2004) as well as poor readers (Amtmann, Abbott, & Berninger, 2007).

Contrary to the hypothesis that slow naming speed would be associated with dyslexia only, this study found that slow naming of letters was associated with the groups with dyslexia and AD, but not with the combined ADHD subtype. The finding of an association between dyslexia and RAN is consistent with many studies that have found RAN to be a significant predictor of reading performance, including reading fluency (McGrath et al., 2011; Pham et al., 2011; Savage & Frederickson, 2005), both concurrently (Plaza & Cohen, 2003; Wolf & Bowers, 1999) and longitudinally (Kirby et al., 2003; Schatschneider et al., 2004). Rapid naming deficits have also been reported in the literature for children with dyslexia + ADHD (e.g., Bental & Tirosh, 2007), and some researchers have proposed the association is at least in part attributable to a common cognitive processing speed deficit,
which, they hypothesize, is strongly linked to RAN. This study separately examined the combined ADHD subtype, as well as the AD only subtype, and found a significant association between RAN and the AD subtype only. This finding suggests that it is the inattentive symptoms that contribute to slower performance on RAN tasks and is in accord with those studies that found RAN tasks are among the best predictors of the inattentive symptoms associated with ADHD (Rucklidge & Tannock, 2002; Weiler et al., 2000). RAN relies on continuous responding and sustained attention to visual stimuli (Rucklidge, 2006), and it has been suggested that individuals with sustained attention deficits process visual information more slowly, particularly in the context of increased cognitive load and integration of multiple operations (Tannock et al., 2000). It may be that inattention and distractibility result in a failure to maintain adequate levels of automaticity (Jacobson et al., 2011), which slows performance on rapid naming tasks. Pham and colleagues (2011) found that combined (parent and teacher) ratings of inattention were negatively associated with RAN, suggesting higher levels of attention are needed to perform well on RAN tasks, even among unimpaired controls. Although previous studies have demonstrated that children with the combined ADHD subtype perform more poorly on RAN measures relative to control groups (e.g., Carte, Nigg, & Hinshaw, 1996; Semrud-Clikeman et al., 2000; van Mourik et al., 2005), these studies have not separated the ADHD symptom dimensions, and it is the inattentive subtype shown to be most strongly related to reading difficulties (e.g., Willcutt, Pennington, et al., 2005). It is possible that in previous studies children with ADHD could have had predominantly inattentive symptoms, as well as symptoms of hyperactivity.

Working memory underlies the capacity to perform complex tasks such as learning to read, reading comprehension, reasoning, and planning (Baddeley, 2003, 2007). The current results indicated a direct association between better reading fluency and better working memory capacity, and is consistent with a substantial body of research that has found
working memory plays an important and direct role in the development of reading and reading comprehension skills (Jacobson et al., 2011; Kibby et al., 2014; Moura et al., 2017; Weng, Li, & Li, 2016). It is also in accord with research that has found developmental increases in verbal memory span are associated with increased articulation rates, which are also determined by developmental improvements in processing speed (e.g., Chuah & Maybery, 1999; Hulme, Thomson, Muir, & Lawrence, 1984; Kail & Park, 1994). However, in this study we found no direct association between ADHD or AD and working memory. Instead, we observed that a severe presentation of ADHD or AD symptoms influenced the association between reading fluency and working memory. When symptoms of ADHD or AD were in the normal range, working memory improved as reading fluency improved, however, when clinical symptoms of ADHD or AD were very high, the association between reading skills and working memory variables was much weaker. This suggests when symptoms of ADHD or AD are severe, better reading fluency is less closely associated with improved working memory than when ADHD or AD is not present. Further, symptoms of ADHD or AD had no additional impact on poor working memory capacity over that of poor reading fluency, and does not support the view of working memory deficits as a central core component of ADHD or AD (Rapport et al., 2009). Rather, the findings fit with models of ADHD that view working memory deficits as one of several executive functions impacted by dysregulated inhibition processes (Barkley, 2014), or as one of multiple executive function weaknesses that comprise the ADHD profile (Willcutt, Pennington, et al., 2005).

The finding that poor reading comprehension was associated with poor reading fluency, phonological decoding, working memory, as well as slow naming speed is consistent with research that has found working memory to be involved in successful reading and reading comprehension (de Jong, 1998; Friedman & Miyake, 2004; Gathercole, Alloway, et al., 2006; Kibby et al., 2014; Martinussen & Tannock, 2006; Swanson, Howard, et al., 2006;
Swanson et al., 2009). For example, a recent study investigating the effects of working memory on RAN and reading comprehension, in a sample of Chinese college students, found that verbal working memory underlies the relationship between RAN and reading comprehension (Weng et al., 2016).

The finding in this study that slow RAN was also associated with AD is consistent with current research findings that indicate it is the inattentive symptom domain of ADHD that is strongly associated with reading difficulties, rather than hyperactivity or impulsivity (Wyllcutt et al., 2000). There was no significant effect of ADHD or AD on reading comprehension scores, so the hypothesis that problems with reading comprehension would be associated with dyslexia and ADHD and AD was not supported. However, some research has found poorer working memory explains poorer comprehension in children with ADHD (e.g., Miller et al., 2013). One explanation for the differing results may be the use of the WIAT-II reading comprehension in the current study, as this subtest allows participants to review each comprehension passage, and was the only measure used to assess reading comprehension. The ability to review the passages before responding to the questions may have mitigated any problem with working memory, so that children with more symptoms of AD or ADHD did not show evidence of poorer reading comprehension. However, the research on reading comprehension and ADHD has been mixed: some studies have observed reading comprehension difficulties (Brock & Knapp, 1996; Gregg et al., 2002; Samuelsson, Lundberg, & Herkner, 2004), while other findings are inconclusive (Ghelani et al., 2004). Some studies investigating comprehension difficulties in children with ADHD have used listening rather than reading comprehension tasks (Flake, Lorch, & Milich, 2007; Flory et al., 2006; Lorch et al., 2004; McInnes et al., 2003), and differences in attentional demands, such as text complexity and length (Keenan et al., 2008) may also contribute to differences in research findings.
Processing speed is a core cognitive ability that scaffolds other complex abilities such as working memory and reading comprehension (Gorman et al., 2016). Theories of cognitive development propose a cascade in which developmental increases in processing speed lead to increases in working memory, that are further associated with improvement in other cognitive and academic abilities. In contrast, slowed processing speed and/or reduced working memory capacity is associated with reduced efficiency in an array of cognitive and academic outcomes (Gorman et al., 2016). In this sample, children with dyslexia appeared to have an impairment in the ability to process information quickly, as evidenced by their poor reading fluency, slow naming speed, and slower response time on the interference control measure. Slowed processing speed is thought to create a bottleneck that reduces both reading fluency and reading comprehension and decreases working memory capacity (Gorman et al., 2016). The group with AD were also slow on the speed of processing measures (RAN and the Flanker task) and had working memory deficits. However, this group was not impaired on the reading measures (reading fluency, phonological decoding and reading comprehension). This might suggest that their processing speed impairments are the result of inattention and distraction, rather than a genuine reduction in processing efficiency. The group with ADHD also showed working memory deficits, without any evidence of impaired processing speed. However, this group made more errors on both interference control conditions suggesting their responses may have been quick and impulsive, rather than controlled and considered. The distinctions between the three groups suggest that working memory impairments may be manifestations of different underlying processes.

Overall, the finding that difficulties with phonological decoding, rapid naming, interference control, working memory and reading comprehension are associated with dyslexia is consistent with research that has found each of these processes to be integral to reading and reading comprehension. The finding that interference control is significantly
associated with poor reading fluency, independent of co-occurring ADHD or AD, potentially identifies this EF as a shared cognitive deficit that may explain the co-occurrence between these disorders. It also provides tentative support for the hypothesis that executive dysfunction is not a sufficient cause for the expression of ADHD or AD (Nigg et al., 2005; Willcutt, Doyle, et al., 2005). This study identified at least two cognitive deficits to be associated with dyslexia, ADHD and AD, supporting a multiple deficit model of these developmental disorders. In particular, the findings clearly identify five cognitive difficulties in the dyslexia profile. The finding that a phonological deficit was associated with dyslexia alone is in contrast to the multiple deficit model, as it does not support a complete dissociation between disorders. However, in Chapter 3 we demonstrated that the majority of children with AD and ADHD also had subclinical reading difficulties, suggesting the dissociation is only partial, and this is consistent with the MDM. Based on the risk factors assessed in this study, we were unable to find any unique predictors for ADHD or AD. However, this is consistent with the research literature, which has not yet identified a core deficit underlying the disorder (Andreou et al., 2007). Further, the classification process for AD and ADHD did not include a clinical diagnosis, and there is a possibility that some children were classified with a disorder they did not have. The cognitive constructs included in the battery were not all-inclusive of previously identified predictors of ADHD or AD, and one possible unique predictor for future research of AD is organisational skills. For example, a recent study found that 38% - 57% of WM’s effect on organisational problems was conveyed by WM’s association with inattentive behaviour, and that attentional difficulties uniquely predicted poor organisational skills (Kofler et al., 2017). The researchers proposed that although hyperactive behaviour is characterised as intrusive and detrimental, the overt verbalisations (e.g., self-talk, talking through steps out loud, blurring out answers) associated with the hyperactive subtype of ADHD may in fact be a protective factor in working memory
processes, as they decrease WM demands by externalising thoughts and re-engaging sensory encoding and storage buffers (Kofler et al., 2017).
Chapter 7: General Discussion

This research had seven aims: (1) investigate the prevalence of single and multiple developmental disorders in a sample of children with a either a previous diagnosis of dyslexia, ADHD or AD, or who had been identified by parents as underachieving in school; (2) determine whether the severity of the deficits found would be greater in children with more than one disorder compared to a single disorder; (3) validate an adult self-report protocol used to screen for dyslexia and ADHD, and use this measure to estimate family risk of dyslexia, as well as for ADHD and AD separately; (4) determine the prevalence of symptoms of dyslexia, ADHD and AD in children with and without a family risk of dyslexia, ADHD or AD; (5) determine whether a single, double, multiple, or an intergenerational multiple deficit model is the best predictor of reading fluency; (6) examine the extent that difficulties with phonological decoding and rapid automatic naming differentiate dyslexia, and the extent that difficulties with interference control differentiate ADHD and AD; and (7) examine the multiple deficit model as an explanation of the co-occurrence between dyslexia and ADHD or AD using cognitive variables identified as common to both disorders.

To achieve these aims, a sample of children ($N = 72$) with dyslexia and/or ADHD, or parent-reported academic difficulties, were assessed on a range of abilities. The participants were a convenience sample that consisted predominantly of males (64/18), which is in accordance with studies that have used clinical criteria for dyslexia.

Contrary to the hypothesis that the majority of children would show evidence of more than one developmental disorder, the overall prevalence rates for single and multiple disorders were similar using clinical criteria. However, the prediction was supported when subclinical symptoms were included, as most children diagnosed with one disorder had subclinical symptoms of at least one other disorder. A series of one-way ANOVAs were conducted to determine whether the severity of each disorder was influenced by the number
of disorders diagnosed in each child. The results indicated that the groups with co-occurring
dyslexia + AD, or dyslexia + ADHD were not more impaired than the groups with either
dyslexia, ADHD or AD alone.

Family risk of dyslexia, ADHD or AD was estimated using a self-report questionnaire
asking parents to report on their own reading, attention and hyperactivity behaviours. The
parent sample \((N = 117)\) included 64 mothers and 53 fathers who completed the Adult
Reading Questionnaire (ARQ: Snowling et al., 2012), and the short-form of the Adult ADHD
Self-Report Scale (ASRS: Kessler, et al., 2005). The factor structures for the ARQ and
ASRS were validated separately and found to be consistent with those previously reported.
These scales were used to estimate family risk of dyslexia, ADHD and AD in the child
sample. The influence of parental reports of reading and attention difficulties on child
reading and attention difficulties was determined using correlation, regression, logistic
regression analyses and odds ratios. The results indicated that 70.4% of children at family
risk of dyslexia had scores in the clinical range on the reading measures, compared to only
43.2% of children without family risk of dyslexia. Parent-reported symptoms of ADHD
indicated that 42.9% of the children at family risk met criteria for a clinical diagnosis,
however, more than half (57.1%) the children at family risk showed no symptomatology of
the disorder. When subclinical symptoms were also considered, the children of parents with
self-rated symptoms of ADHD were more likely to have clinical or subclinical symptoms
(64.3%) than children from a family with no risk of ADHD (38%). In the current sample the
majority (81%) of children with AD were from families with no reported history of AD
symptoms, indicating there was no significant association between parent-reported symptoms
and a child diagnosis of AD.

The best predictors of reading fluency were identified using correlation, and multiple
regression analyses to evaluate single, double, multiple, and the intergenerational multiple
deficit model of dyslexia. Based on the variables assessed, children were found to have a combination of single, double and multiple deficits (phonological decoding, rapid letter naming, and expressive language), indicating there are various pathways to dyslexia. Parental reports of reading difficulties were examined using the intergenerational multiple deficit model, and consistent with the hypothesis, they were found to account for a substantial proportion (18.6%) of the variance in children’s reading fluency.

Finally, mixed ANOVAs, and moderated regression analyses were conducted within a multiple deficit framework to determine the extent that impairments in phonological decoding and rapid naming were unique to dyslexia, and impairments in interference control were unique to ADHD and AD. The contribution of working memory and reading comprehension to each dyslexia, ADHD and AD was also examined. As predicted, phonological decoding was a unique predictor of dyslexia, however, contrary to the hypothesis impaired interference control was found to be a shared cognitive deficit. Further, the study found that the relationship between reading fluency and naming speed was moderated by AD, although the effect was weak. This moderating effect was not present for ADHD. However, ADHD and AD were each found to moderate the relationship between reading fluency and working memory. For children with severe symptoms of ADHD or AD there was no significant association between reading fluency and working memory, but for those children within the normal range of ADHD or AD symptoms, as reading fluency improved so did working memory. The results and implications of the findings for the diagnosis of these disorders, as well as educational interventions targeted at reading and attention difficulties, are discussed within the context of each study. The limitations of this thesis and future research directions are addressed within each section.
7.1 The Prevalence of Single and Multiple Developmental Disorders in Children with Dyslexia, ADHD and AD.

Developmental research has highlighted the high rates of co-occurrence between the developmental disorders (e.g., dyslexia, ADHD, AD, SLI and DCD). This has led to theoretical models (e.g., Neuroconstructivism; Karmiloff-Smith, 2009) that propose a deficit in one region of the brain has cascading effects that result in multiple cognitive weaknesses (e.g., reading and attention deficits), and is supported by neurological studies that have demonstrated the interconnected nature of the brain’s cortical systems. Consequently, some researchers now propose that single disorders are uncommon, and that prevalence estimates would be higher for co-occurring disorders (Gooch et al., 2014). Based on these findings it was expected that multiple developmental disorders (e.g., dyslexia + ADHD or AD, ADHD + DCD or dyslexia + ADHD + SLI/DCD) would be more prevalent in a group of children with dyslexia (or symptoms of) + ADHD or AD, than the occurrence of single disorders (e.g., dyslexia, ADHD, AD, SLI or DCD alone). In contrast, most children were observed with a single disorder (dyslexia, ADHD or AD) or dyslexia + ADHD or AD, with few children in the sample showing evidence of SLI or DCD. Considering the large number of children identified with AD or ADHD, as well as the considerable overlap reported in the literature between dyslexia and ADHD, it might be expected that the high number of children diagnosed with ADHD or AD would result in a higher likelihood of co-occurring disorders. However, this was not the case. Using clinical criteria 56.5 % (13) of children had a single diagnosis of ADHD and 45.7 % (21) of children had a single diagnosis of AD. In contrast to the findings for AD and ADHD, dyslexia was much less likely to present as a single disorder. Only 25 % (9/36) of children with dyslexia had this disorder alone. These findings fail to support the neuroconstructivist view (Karmiloff-Smith, 2009), and indicate that single
theoretical explanations of dyslexia and ADHD should be considered, as well as those that explain the co-occurrence between the developmental disorders.

One important issue that arises from these findings is the high number of children in the sample showing evidence of AD or ADHD. Despite there being few children with a prior clinical diagnosis of ADHD combined type, and none with a prior diagnosis of ADHD predominantly inattentive type, the classification procedure identified 23 children who met criteria for ADHD, and a further 23 who met criteria for AD alone. There are several explanations for these findings. First, the prevalence of ADHD and AD is influenced by the method used to make the diagnostic decision, and a clinician based assessment has been found to produce the lowest estimates, while teachers have been observed to report more symptomatology in students than do parents (Angold et al., 2000; Thomas et al., 2015). In the current project the ADHD and AD subtypes were determined using the mean from the parent and teacher reports (other than 10 participants who were classified based on a parent or teacher report only). There was a very high level of parental concern regarding the children’s academic and/or reading performance which may have influenced their responses. Inspection of the data file showed that for the inattention domain, 69% of parents provided ratings on the AD scale within the clinical range. In contrast, teachers placed 48% of the sample in the clinical range. For hyperactivity, parent-ratings placed 51.5% of the children in the clinical range, with teachers placing 24.2% of children in the clinical range. These findings indicate that for this sample, higher clinical ratings occurred on parental reports, rather than for teachers. Due to their anxiety, parents may have over-reported symptoms, particularly AD, as most of these cases were found to be single disorders.

A further explanation for the high prevalence of AD is that the inattentive symptoms identified in the sample are symptomatic of more general reading and academic difficulties found to occur in children in this age group. This could have been more obvious to parents
than to teachers, who work with larger numbers of children. Attention difficulties have also been observed to be more obvious as the demands of reading and school work become more challenging during this period of schooling (Ebejer et al., 2010). For example, reading is an effortful, complex task, and there is evidence to indicate that attention mechanisms are critical for translating print to speech and for fluent reading (Reynolds & Besner, 2006).

Ebejer and colleagues (2010) found that the association between dyslexia and inattention in children aged 6 to 8 years could be explained by a common genes account, however, inattention was found to impact reading ability more directly from 8 to 9 years of age, which they attributed to the greater cognitive demands associated with reading and reading comprehension at that age. As learning demands increase and text becomes more complex, so too does the reliance on attentional mechanisms and systems. However, the argument for the over-estimation of AD or ADHD based on inattention as symptomatic of greater academic demands is diminished when the implications of the observed impairments in executive function domains are considered. For example, in Study 4 (chapter 6) we found that although an additional diagnosis of AD or ADHD with dyslexia did not result in poorer working memory, the presence of AD or ADHD was associated with reduced working memory capacity for children with average reading fluency. Further, the performance of these groups was impaired on the interference control measure, providing some evidence that the difficulties experienced by these children are genuine, and suggest the presence of an underlying disorder.

Another explanation for the overestimation of AD in the current sample is that these children were less likely to be formally identified (Rowland et al., 2015). Children with inattention are often overlooked in school because they tend to be quieter and less disruptive than children with co-occurring hyperactivity and impulsivity. It is possible that the children identified with AD in the current study have a genuine attention deficit but were overlooked
at school because they lack the behavioural disturbances that frequently result in the identification and referral of children with the combined ADHD subtype. In addition, this group of children were recruited from the community, where AD is noted to be more common than ADHD (Milich et al., 2011). The age-of-onset criteria may also be a contributing factor, as inattentive symptoms have been found to emerge later than hyperactive/impulsive symptoms (Rowland et al., 2015). Finally, this was a self-selecting sample specifically targeted towards children with symptoms of dyslexia and/or AD, so the estimates for these disorders are expected to be higher. However, due to the high level of parental concern regarding the reading performance of this sample, the majority of children (65%) were receiving remediation at school, and/or undertaking private tuition, and it should also be considered that this may have resulted in the amelioration of the severity of symptoms for at least some children in the sample.

The prediction that dyslexia and ADHD or AD would co-occur more frequently when sub-clinical symptoms were reported was supported, with more than two-thirds of children in the current sample found to have dyslexia and an additional attention deficit. This finding supports previous research that has observed high rates of co-occurrence between dyslexia, ADHD and AD (e.g., Bental & Tirosh, 2007; Czamara et al., 2013; Del’homme, et al., 2007; Miranda, et al., 2011). Importantly, in the current study the prevalence of dyslexia + AD was substantially higher than that found between dyslexia + ADHD and is consistent with research that has also found a strong association between attention difficulties and dyslexia (Heikkila et al., 2016).

The present research provided extensive evidence of subclinical dyslexia in children with ADHD or AD, and subclinical ADHD or AD in children with dyslexia. This is consistent with studies that have investigated the association between dyslexia and ADHD and found that even when clinical criteria were not met for both disorders, subclinical
elevations of the other disorder are often evident (e.g., Willcutt et al., 2007). The high rates of subclinical symptomatology found in the current study provides support for the proposal that dyslexia and ADHD are continuously distributed disorders (e.g., Boets et al., 2010; Pennington, 2006; Snowling & Melby-Lervag, 2016), and has important implications for the diagnosis and remediation of these disorders. In the total sample there were only 6 (8.3%) children without clinical or subclinical evidence of any disorder, and this is consistent with the concerns reported by parents that their children were not performing as well as expected academically. The high rates of subclinical symptomatology suggest there are many children with undiagnosed difficulties that negatively impact their learning, and this research highlights the importance of assessing children on a range of ability measures. Although one primary difficulty such as dyslexia may be the most obvious, it is clear from these results that there is likely to be at least one other subclinical impairment contributing to the problem. When a child is assessed for a reading difficulty they should also be assessed for an ADHD, and vice versa. When all the difficulties experienced by a child are identified, interventions can be tailored to address each child’s specific needs and weaknesses.

In the current study the children with dyslexia + AD or dyslexia + ADHD were not more impaired than children with either disorder alone. These findings are not consistent with other research that has found children with co-occurring developmental disorders (e.g., DCD + ADHD + SLI) have more severe symptomatology and are more pervasive underachievers. Several explanations are proposed for the more severe impairments found in these prior research studies. First, these samples included many children who were more likely to be clinically impaired across a range of measures including language, motor, perceptual organisation, and verbal comprehension. These children were receiving specialist assistance (e.g., speech and language therapy, occupational therapy), and many were recruited from these specialist units (e.g., Dyck & Piek, 2010; 2014) making them more severely impaired
than the participants in the current research. Second, in many of these other studies the majority of children presented with more than two disorders (e.g., Piek & Dyck, 2004), and this may indicate that children with more than one additional disorder may form that clinically impaired group found in studies of children with severe difficulties (e.g., Dyck & Piek, 2010; 2014). There were few children in the current sample with evidence of more than two disorders, either clinically or sub-clinically, and although many were receiving remediation for literacy and/or numeracy none were receiving specialised clinical treatment.

Another explanation proposed is that the combination of deficits may have had an impact on the severity of difficulties. For example, these prior studies contained larger numbers of children with SLI and DCD, whereas our sample contained children with predominantly dyslexia and AD or ADHD. In the current study it was expected that the groups with dyslexia + AD or dyslexia + ADHD would show poorer performance than the single disorder groups. In contrast, no differences in severity were found between the multiple and single disorder groups, indicating that the presence of this particular combination of disorders (e.g., dyslexia + AD or dyslexia + ADHD) did not result in more severe symptoms. However, there was a small group of children (n = 5) with language impairments who had poorer reading fluency, greater symptoms of ADHD or AD, and poorer non-verbal ability than the other groups. Despite the small group size, the addition of SLI with dyslexia + AD had an association with the language, literacy, and non-verbal skills of this group and highlights the importance of including language measures in a developmental assessment, as the additional language deficits in the current sample appeared to have an impact on the performance of these children.

7.2 Family Risk and the Prevalence of Dyslexia and ADHD or AD

There is considerable evidence to show that the risk of a child developing reading difficulties is significantly higher if he or she has an affected relative (Shaywitz et al., 2008).
An important aim of family risk studies is to identify the precursors of dyslexia before formal reading instruction commences (Snowling et al., 2012), and to understand the contribution of parental risk to children’s reading skills (van Bergen, van der Leij et al., 2014). Studies have shown that children with a family history of dyslexia are at an increased risk for the co-occurrence of reading and attention problems from kindergarten onward (Costa et al., 2013). Longitudinal studies have also presented evidence that inattentive behaviour during kindergarten predicts reading achievement by the end of primary school (Duncan et al., 2007; Rabiner & Coie, 2000), and that inattention reported in middle school significantly predicts long-term educational attainment in early adulthood (Pingault et al., 2011). These research findings are supported by studies that have found a strong genetic association between reading and inattention (e.g., Ebejer et al., 2010; Greven, et al., 2012; Willcutt, Pennington, et al., 2007), and emphasize the importance of accurately identifying children at family risk and assessing them for an ADHD, as well as for reading difficulties.

In Study 2 (Chapter 4) the scores from the ARQ and ASRS were used to estimate family risk of each disorder, and an overlap between the symptoms of dyslexia and ADHD or AD was found in the parent sample. For example, parents reporting greater reading difficulties had higher scores on both the reading and attention scales, and 35.5% of this group were more likely to report symptoms of AD or ADHD than parents without reported reading difficulties. Similarly, parents reporting symptoms of AD or ADHD reported poorer reading and word finding skills and were more likely to report the potential presence of dyslexia than the group without AD or ADHD. These findings are consistent with Study 1 (Chapter 3), where a significant overlap was found between the clinical and subclinical symptoms of dyslexia and ADHD or AD in the child sample, and supports other genetic and behavioural research that has identified an association between reading difficulties and inattention (e.g., Ebejer et al., 2010; Gayán et al., 2005; Greven et al., 2011; Greven et al.,
A strong association was found between parent-reported symptoms of dyslexia, and the prevalence of symptoms of dyslexia in their children. The majority (70.4%) of children at family risk of dyslexia had scores in the clinical range on the reading measures and supports the use of the ARQ for the identification of children at family risk of dyslexia. The identification of children at family risk is important for several reasons. For example, as demonstrated in the current research, as well as other studies (e.g., Hulme, Nash, Gooch, Lervåg, & Snowling, 2015; Nash, Hulme, Gooch, & Snowling, 2013), literacy outcomes are continuously distributed, and although some children at family risk did not meet clinical criteria for a diagnosis of dyslexia, they were observed to have subclinical symptoms, such as poorer reading fluency and pseudoword decoding skills. This research demonstrates the significant number of children at family risk who present with clinical as well as subclinical reading impairments and highlights the critical importance of reliable assessment for liability to dyslexia. A protocol such as the ARQ offers schools a simple format for gathering family information that could be used to identify those children likely to develop a reading difficulty. Ideally, children should be assessed for risk at a young age, as the implementation of early reading intervention prior to reading failure has been found to be more effective than later remediation, once children’s reading skills lag (e.g., Compton, Miller, Elleman, & Steacy, 2014; Ozernov-Palchik et al., 2017). The finding that the ASRS was not effective in identifying children at risk of AD further underscores the importance of assessing a child for an attention deficit if they have been identified to be at family risk for dyslexia, as there is a high risk that they will be overlooked for this additional disorder.

It is a limitation of this study that parental education was not considered, as parents’ educational attainment has been found to be associated with a number of variables, such as
the frequency that parents read themselves, and to their children, as well as the availability of reading material in the home (Hamilton et al., 2016; van Bergen Zuijen et al., 2017). This information could be utilised as another potential risk indicator, for use in educational settings to identify families who may benefit from information highlighting the importance of the home literacy environment.

7.3 Single, Double, Multiple, and Intergenerational Explanations of Reading Fluency

When examining single deficit models of dyslexia, phonological decoding and rapid naming explained equal amounts of variance in children’s reading fluency, and these variables were again found to contribute equally to the double deficit model of dyslexia. It was found that 75% of the group with dyslexia had an impairment in phonological decoding, rapid naming or both, supporting a considerable body of research that has consistently found children with dyslexia to have pronounced deficits across these skills (e.g., Boets et al., 2010; Caravolas, Volín, & Hulme, 2005; Carroll, Solity, & Shapiro, 2016; Menghini et al., 2010; Moura, Moreno, Pereira, & Simões, 2014; Ramus, Rosen, et al., 2003; van Bergen et al., 2011). For example, phonological processing deficits and decoding capabilities have been found to be core difficulties for many individuals with dyslexia (Lyon, Shaywitz, & Shaywitz, 2003), and tend to be identified using accuracy measures such as the phonological decoding measure used in the current study. In addition, many studies have found that RAN performance is the most important predictor of reading fluency across all orthographies in both typical and dyslexic readers (Kirby, Georgiou, Martinussen, & Parrila, 2010; Norton & Wolf, 2012). Rapid naming involves a wide range of cognitive processes. These include the integration of visual and orthographic information with stored phonological representations, access and retrieval of phonological labels, attentional processes, as well as processing speed (Kirby et al., 2010; Norton & Wolf, 2012). It has been suggested that RAN tasks represent an index of how well children with dyslexia are able to establish the word-specific orthographic
representations that underpin reading (Clarke, Hulme, & Snowling, 2005; Ehri, 1995). However, in the current study a number of children with dyslexia were observed without an impairment in phonological decoding, rapid naming or expressive language. This suggests the involvement of other cognitive deficits, which were not assessed in this study.

Although consistent with the study by Pennington and colleagues (2012), the assessment for the presence of dyslexia relied on reading fluency alone and makes it difficult to generalise these findings to other studies that have assessed dyslexia in different ways (e.g., such as those based on poor word identification skills). Future studies should include variables such as single word reading and a broader range of phonological processing tasks. For example, the inclusion of variables such as phonological memory and orthographic coding may have identified more children in the current sample with specific underlying cognitive impairments.

This was the first formal test of the intergenerational model since that of van Bergen, van der Leij and colleagues (2014), who examined the effects of parental literacy skills and children’s skills in a family risk study. The inclusion of parent-reported reading difficulties indicated no significant association between mother’s reports of reading difficulties and children’s reading fluency. The lack of association may have been due to the few mothers in the sample reporting significant reading difficulties, or to the small sample size, as a larger sample could have provided more information. However, children whose fathers reported greater reading difficulties were observed to have poorer reading fluency and pseudoword decoding, as well as slower naming speed. The current analyses indicated that the ARQ had good internal consistency, and the use of self-report reading measures have been found to be a good discriminator of adults with dyslexia in other family risk studies (e.g., Giménez, Luque, López-Zamora, & Fernández-Navas, 2015; Wolff & Lundberg, 2003). However, the
use of an objective measure of parental reading ability may have increased the likelihood of a positive diagnosis and should be considered in future studies.

The findings from the inclusion of the intergenerational variable highlight the important contribution of family risk factors in explaining children’s reading fluency as well as for their inclusion in family risk studies. A key component of the MDM is the proposal that liability for a disorder is continuously distributed. This predicts that at-risk children without dyslexia have also inherited some risk factors that place them at a higher liability than children without family risk. Consequently, the identification of at-risk children without a clinical diagnosis is critical, as subclinical symptoms may go undetected. Children without overt symptoms of reading or attention difficulties, and who do not have parents who are as vigilant as those in the current sample, may not be identified and included in school reading remediation programs, resulting in poorer academic performance throughout their school years.

7.4 A Multiple Deficit Explanation of the Co-occurrence between Dyslexia and ADHD or AD

This thesis sought to advance the understanding of dyslexia, ADHD and AD by identifying cognitive deficits that differentiate these disorders, as well as shared cognitive deficits that may explain the association between them. The performance of the group with dyslexia was found to be impaired across all the cognitive variables assessed. This is consistent with previous research that has found phonological (Lyon et al., 2003), rapid automatic naming (Catts et al., 2002; Landerl et al., 2013; Shanahan et al., 2006; Willcutt, Pennington, et al., 2005; Wolf, 1997), reading comprehension (Hulme & Snowling, 2016; Pennington & Lefly, 2001; Snowling et al., 2003), working memory (Kibby & Cohen, 2008), and interference control (Berninger et al., 2006; Brosnan et al., 2002; Purvis & Tannock, 2000; Reiter et al., 2005) deficits to be associated with dyslexia. The groups with ADHD and
AD were impaired on the interference control measure, and each was found to moderate the association between reading fluency and working memory, particularly for good readers. AD was also found to moderate the relationship between reading fluency and rapid naming.

**Dyslexia**

Consistent with a large body of prior research, phonological decoding was found to be a unique predictor of dyslexia (McGrath et al., 2011; Pennington et al., 2012). Poor reading comprehension was also a unique predictor of dyslexia and the finding that these variables were also associated with reading fluency, working memory, as well as naming speed is consistent with research that suggests working memory is an important construct for successful reading, and underlies the capacity to perform more complex processes such as reading comprehension (Friedman & Miyake, 2004; Gathercole, Alloway, et al., 2006; Kibby et al., 2014; Martinussen & Tannock, 2006; Swanson, Howard, et al., 2006; Swanson et al., 2009).

In Study 3 (Chapter 5), poorer reading fluency was associated with slower rapid naming, and in Study 4 (Chapter 6) the group with dyslexia responded more slowly to the incongruent condition on the interference control measure (although accuracy was not impaired). These findings are in accord with a recent study examining the cognitive mechanisms involved in rapid automatic naming. For example, Bexkens and colleagues (2015) found that interference control was significantly related to RAN performance and captured 15% of the variance in the total RAN performance. Those children with dyslexia who had poorer interference control also had poorer performances on rapid naming tasks. The authors concluded that the inhibition of inappropriate response activation is necessary to select between competing response alternatives and is therefore important for the efficient rapid naming of target stimuli (Bexkens et al., 2015). However, in the current research the group with dyslexia were only impaired on response time for the Flanker task, and an
alternative explanation for the group’s slower performance on both the RAN and Flanker
tasks may the involvement of slow processing speed, as there is extensive evidence of
difficulties in speed of processing for almost all stimuli in individuals with dyslexia (Fawcett,
Nicolson, & Maclagan, 2001; Shanahan, Pennington, & Willcutt, 2008).

**AD and ADHD**

No direct associations were observed between AD or ADHD and working memory
scores, however symptoms of AD or ADHD appear to have had a greater impact on better
readers in the current sample. When symptoms of AD or ADHD were low or absent, there
was a strong association between reading fluency and working memory. For example, as
reading fluency improved so too did working memory capacity. However, no significant
association between reading fluency and working memory was observed when symptoms of
AD or ADHD were high. For example, there was no associated improvement in working
memory for children with AD or ADHD when reading fluency scores improved, indicating
that working memory capacity did not improve to a level expected of fluent readers. These
findings suggest that no matter how fluent a child’s reading may be, symptoms of inattention
and hyperactivity/impulsivity each reduce a child’s capacity to encode and store information
for short periods of time, and that these traits interrupt the cognitive processes involved in
working memory. A similar (but much weaker) conditional association was found between
reading fluency, AD and rapid naming. Rapid naming skills improved as reading fluency
improved, however when clinical symptoms of AD were very high the association found
between reading skills and rapid naming (although significant) was not as strong. Rapid
naming performance improved, but to a lesser extent than for children without AD. This is
consistent with other studies that also used combined parent and teacher ratings of inattention
and found that good performance on RAN tasks requires higher levels of attention (Pham et
al., 2011). This suggests that symptoms of inattention and distractibility interrupt the
automatic processing and response requirements involved in RAN tasks (Jacobson et al., 2011).

These results differ from other studies that found direct associations between ADHD or AD, working memory and RAN. However, the impact of AD or ADHD may be more obvious for children with moderate to low reading skills but makes no additional contribution in a sample such as this, where reading is already severely impaired. These findings are interesting and highlight the serious impact of symptoms of AD and ADHD, particularly for children who are average readers. However, it should be noted that these results relate only to reading fluency and should be replicated using additional measures such as word identification, and a wider range of phonological processing measures.

The AD and ADHD groups also demonstrated impairments on the interference control measure, although they were observed to have different types of difficulties on this task. The AD group responded more slowly and made more errors on both the congruent and incongruent conditions, suggesting that the distractibility and mind wandering associated with AD slowed response times. These findings may also indicate that the observed errors were the result of guesses due to lapses in focused attention. The group with ADHD also made more errors on both the congruent and incongruent conditions but responded as quickly as the groups without ADHD. The responses of this group were likely based on impulsivity, resulting in more errors because they responded before evaluating the correct response. These findings suggest that the interference control deficits found for the AD and ADHD groups are the result of different underlying causes, and supports the current movement toward investigating the ADHD subtypes separately.
7.5 Conclusions

This thesis sought to advance theoretical understanding of the association between dyslexia and ADHD within a sample of children identified predominantly with reading and attention difficulties. The results of the four studies contribute to research building a case for the utility of conceptualising the symptoms of dyslexia, AD and ADHD as continuously distributed, rather than discrete and categorical disorders (Pennington, 2006). The use of categorical groupings leaves many children with symptoms outside the arbitrary cut-offs, and the data from this research indicates that a substantial number of children with diagnosed with dyslexia, AD or ADHD have additional unidentified subclinical symptomatology that has a negative impact on their academic performance. Although the children in this sample have proactive parents who recognised these difficulties and have ensured that they receive additional remedial assistance, this is not always the case. Many children fail to receive adequate treatment and support services for their difficulties. Of course, as previously noted, it is possible that this proactive sample of parents may have over-identified the symptoms of inattention and hyperactivity in their children, as parent-rated symptoms were substantially higher than those of teachers.

In this sample of children, the overlap between the symptoms of dyslexia and ADHD or AD was substantial and made the use of continuous groups plausible. In addition, this sample contained few good readers, and most children in the dyslexia group were severely impaired, with reading fluency scores in the very low range (standard scores 60 to 70). This may explain why symptoms of ADHD or AD had no additional effect on the severity of difficulties for children with dyslexia.

The much higher prevalence of dyslexia among the children of parents with dyslexia indicates that children at family risk inherit genetic risk factors (van Bergen, van der Leij et al., 2014), and children with a family history of reading difficulties are at increased risk for
the co-occurrence of dyslexia and attention problems. This identifies family risk as an important variable for inclusion in dyslexia research. A recent review of interventions for dyslexia has highlighted their limited success, and noted that a significant number of children receiving these interventions remain poor readers (Compton et al. 2014). Considering the high percentage of children with dyslexia who also have clinical or subclinical attention deficits, the limited success observed for reading intervention programs may be the consequence of unidentified attention deficits that hinder their success. A recent study observed that executive processes such as attention and inhibition had greater power than the typical behavioural indicators of inattention and hyperactivity/impulsivity in predicting learning behaviours in children with ADHD. Consequently, the addition of cognitive training programs (e.g., Rapport, Orban, Kofler, & Friedman, 2013; Tamm et al., 2017) as an adjunct to reading remediation may be critical. These types of interventions address problems that are highly relevant for inattentive behaviour, as well as executive dysfunction, and aid in strengthening learning behaviours (e.g., Colomer, Berenguer, Rosello, Baixauli, & Miranda, 2017; Lundervold et al., 2017).

The main conclusions derived from this research have both clinical and educational implications. The findings highlight the importance of assessing children with dyslexia across a broad range of learning domains, particularly for an attention deficit and/or hyperactivity/impulsivity disorder. In addition, identifying children at family risk enables clinicians and educators to prevent or ameliorate early reading and/or attention difficulties by ensuring these children have access to remediation, and that appropriate intervention plans are developed and implemented. It is imperative that children diagnosed with one developmental disorder be assessed for other co-occurring disorders, both for accurate research methodology and to ensure that appropriate remedial interventions can be timed and implemented to achieve optimal academic outcomes for these children.
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