Neuropsychological and Symptom Predictors of Diagnostic Persistence, Symptom Severity, and Executive Dysfunction in ADHD:

A 23-Year Predictive Follow-up Study

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Abstract

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Title: “Neuropsychological and Symptom Predictors of Diagnostic Persistence, Symptom Severity, and Executive Dysfunction in ADHD: A 23-Year Predictive Follow-up Study”
Main supervisor: Professor Merete Glenne Øie. Co-supervisor: Professor Jens Egeland

Background: Attention-deficit/hyperactivity disorder (ADHD) is currently understood as a neurodevelopmental disorder known to persist into adulthood in a significant subset of cases, but recent large community-based studies have revealed that the rate of persistence may be lower than previously assumed. There is a lack of studies predicting persistence that include both comprehensive neuropsychological test batteries and longer follow-up intervals. This study extends long-term predictive research on ADHD by including both a broad neuropsychological test battery and symptom measures at baseline in adolescence as predictors of diagnostic persistence, symptom severity, and ecologically measured executive dysfunction in ADHD 23 years later.

Methods: Forty-five individuals, 19 with ADHD (M/F=19/0) and 26 healthy controls (HC; M/F=13/13), were assessed in adolescence and 23 years later. Measurements at baseline included a comprehensive test battery measuring eight neuropsychological domains, an IQ estimate, the Child Behavior Checklist (CBCL), and the Global Assessment Scale of Symptoms (GAS). Outcome measures included diagnostic status, the Adult ADHD Self-Report Scale (ASRS), the Attention and ADHD subscales of the Adult Self-Report (ASR) from the Achenbach System of Empirically Based Assessment (ASEBA), and the Behavior Rating Inventory of Executive Function (BRIEF). Group differences between ADHD persisters, ADHD remitters, and HC was calculated by use of ANOVAs in SPSS, and potential predictions of differences in the ADHD group by linear regression analyses.

Results: Eleven (58%) participants retained their ADHD diagnoses at follow-up. The remitters did not significantly differ from healthy controls on measures of symptom severity or executive dysfunction at follow-up. Motor Coordination and Visual perception at baseline predicted diagnostic status at follow-up, but not symptom severity or executive dysfunction. The CBCL Attention problems subscale at baseline in the ADHD group predicted variance in diagnostic status, attention symptoms in the ASR and the ASRS, and the BRIEF Global Executive Composite and Working Memory subdomain.

Conclusion: The persistence rate found in the present study was higher than other long-term estimates, and the predictive value of baseline attention symptom severity was confirmed. The predictions of lower-order neuropsychological functions related to motor function and perception on diagnostic persistence are an important new finding of consequence, suggesting the continued relevance of the early theoretical concept of DAMP. These findings carry implications for future research on interventions, theoretical models, and the lifespan of ADHD.

Preface

It is with feelings of awe, pride, gratitude, and a smidge of trepidation that I hereby submit my final thesis.

When I first jumped at the opportunity to collaborate with Professor Merete Glenne Øie, a role model of mine, on her project "Clinical, Neurocognitive and Functional Outcome in Early-Onset Schizophrenia and Attention-Deficit/Hyperactivity Disorder: A 20-Year Follow-up Study", we took some time to figure out which sample and data I'd work on. Looking back on it all now, eight months later, I am in awe at both the process that has transpired and the final result. I have learned so much about ADHD, neuropsychology, myself, and conducting research – all of it invaluable in my coming career as a scientist-practitioner.

I must convey my most heartfelt gratitude to Merete, my main supervisor. It is my firm belief that had anyone else filled her shoes, the product would be less than it is now, and the process much more costly. It is ironic that in a project concerning self-regulation, I have alternated between being paralysed and overly captivated by the work. Merete has provided gentle encouragement, clear expectations, and boundless patience when my own executive functions have staggered. She shares so generously of both her time and considerable expertise, whether it be on ADHD, neuropsychology, or academic writing.

Many thanks must go to my co-supervisor as well, Professor Jens Egeland. His enthusiasm for this project was immediate and has revitalised it time and again; we have together only grown steadily more excited by its findings. His insight in the field has elevated the quality of this paper, and his guidance on statistics and methods has made SPSS far less intimidating.

I thank every one of the 45 unknown individuals I have analysed here for their two-decade-long loyalty to this project. Your generosity in sharing of your lives may improve others'.

Last, but not least, for all the hugs, fun, input, love, assistance, and support, I shout a great "Thank you!" at my friends, family, and boyfriend. It takes a village.

Oslo, 09.04.19

Tor Amund V. Storaas

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1 Introduction

1.1 Conceptual development of ADHD

One hundred and seven years ago, George Still (1902), a medical doctor at the King's College Hospital in London later referred to as the "father of British paediatrics" (Lange et al., 2010), argued that the "occurrence of a defective moral control as a morbid condition in children" was worth studying more closely. Since then, the issues of abnormal deficits in motivation, learning, attention, and activity levels in children have been conceptualised in various ways (Gillberg, 2003a). As neurological research gained prominence as an academic field, the term "minimal brain dysfunction" (MBD; Clements, 1966; Paul H Wender, 1971) arose based on assumptions that the causal factors of the disorder resided in the brain. The diagnosis of attention deficit disorder was introduced in the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III) published by the American Psychiatric Association as part of their directional change from theoretically oriented diagnoses to clearly operationalised behavioural disorders (American Psychiatric Association, 1980; Epstein & Loren, 2013). The name of the diagnosis was changed to the currently in-use term attention-deficit/hyperactivity disorder (ADHD) in the revision of the DSM-III, the DSM-III-R (American Psychiatric Association, 1987). Hyperkinetic disorder became the corresponding diagnosis of the International Classification of Diseases (ICD) published by the World Health Organization (World Health Organization, 1992).

While the ICD diagnosis of hyperkinetic disorder does not have a subgroup structure, subgroup distinctions were added to the ADHD diagnosis of the DSM in its fourth revision (American Psychiatric Association, 2000), namely the predominantly Hyperactive-Impulsive, predominantly Inattentive, and Combined subtypes. Research has suggested that hyperkinetic disorder is a more severe subset of the combined subtype of ADHD (Asherson, 2016; Gillberg, 2003a), and that as few as only a quarter of children with ADHD qualify for hyperkinetic disorder (Santosh et al., 2005). However, the subtype structure of the DSM has shown limited validity and clinical value, as there exists considerable intra-individual change across subtypes over time (Thapar & Cooper, 2016; Willcutt et al., 2012). This led to the change in the fifth instalment of the DSM from the concept of subtypes to instead including presentation types; for instance, a primarily inattentive or primarily hyperactive presentation

of ADHD (American Psychiatric Association, 2013). Parallel to the development of the ADHD concept, the term deficits in attention, motor control and perception (DAMP) was established in Scandinavia, also intended to replace the then-dominant MBD-term (Gillberg, 1983, 2003b; Kadesjö & Gillberg, 1998). When introduced, the DAMP diagnosis was used to describe cases in which attentional deficits and hyperactivity symptoms co-occurred with motor and perceptual problems called developmental coordination disorder (DCD). Later studies confirmed how these problems negatively impacted behaviour cross-situationally through interactional effects with each other and made a prevalence estimate of 1.5% in 7-year-olds (Gillberg, 2003b). Reflecting the focus of the research literature, the present study will be primarily preoccupied with the DSM diagnosis of ADHD. In this introduction section, the characteristics of ADHD will be deliberated leading up to the research aims of the present study.

1.2 Characteristics of ADHD

ADHD is defined as a childhood-onset mental disorder characterised by attention deficits, hyperactivity, and impulsivity of a developmentally inappropriate nature (Thapar & Cooper, 2016). It is typically seen as a neurodevelopmental disorder that starts in early development and follows a persistent course. The diagnostic criteria of ADHD in the DSM-III-R are that at least eight behaviour disturbances have taken place throughout the last six months, usually cross-situationally, causing clinically significant impairment (American Psychiatric Association, 1987). These eight behaviours, or diagnostic criteria, include, but are not limited to, behaviours such as fidgeting and restlessness, difficulties sustaining attention, or being impulsive and disruptive. These disturbances must have had an onset before the age of seven, must not co-occur with a pervasive developmental disorder, and may only be counted as a criterion for the disorder if the behaviour is considerably more frequent than what typically occurs in most children of the same age.

As is the case for several other mental disorders, comorbidity is widespread in ADHD, with some estimating that two-thirds of people with ADHD have other comorbid mental disorders as well (Killeen, Russell, & Sergeant, 2013). The most common comorbid disorders include oppositional defiant disorder, learning disorders, and conduct disorder in childhood, with anxiety, depression, and substance abuse disorders becoming more prominent in adulthood (Asherson, 2016; Franke et al., 2018). Comorbid pervasive developmental disorder has also

been broadly documented, leading to the removal of the respective exclusion criterion in either diagnosis in the fifth edition of the DSM (American Psychiatric Association, 2013). Gender differences in prevalence have traditionally been reported to be approximately 2.5:1 (Hinshaw, 2018; van Lieshout et al., 2017), matching other neurodevelopmental disorders such as autism and schizophrenia (Hinshaw, 2018; Willcutt, 2012). The case has been made that these prevalence differences may partly stem from referral and diagnostic biases (O'Brien, Dowell, Mostofsky, Denckla, & Mahone, 2010; Rucklidge, 2010), as girls with ADHD are reported to experience both similar and dissimilar symptoms (Hinshaw, 2018). Teachers are often the first to detect ADHD symptoms, but may report stronger symptoms in boys than girls, despite similar levels of impairment (Derks, Hudziak, & Boomsma, 2007). Interestingly, the gender prevalence ratio narrows considerably in adult ADHD (Hinshaw, 2018).

The worldwide prevalence rate among children range around 5%, with reports varying between 3-12% (Polanczyk, Willcutt, Salum, Kieling, & Rohde, 2014). This was partially replicated in a Norwegian study of 12-year-olds, which found a national prevalence estimate of hyperkinetic disorder of 5%, but with substantial intranational variation across counties (Surén et al., 2018). The adult prevalence rate is estimated to be approximately half of that found in children, around 2.5%-3%, although this might realistically be higher due to the childhood-oriented diagnostic criteria not accurately catching impairment experienced by adolescents and adults with the disorder (Epstein & Loren, 2013; Franke et al., 2018). These variations in prevalence estimates reported across studies seem to stem from methodological variation in diagnostic and screening practices in either clinical settings or epidemiological surveys, and not to true variance in the occurrence of the disorder across geographical locations (Polanczyk et al., 2014; Thapar & Cooper, 2016).

Like many mental disorders, ADHD can be understood as the extreme end of a continuum of traits that are normal parts of the human condition (Asherson, Buitelaar, Faraone, & Rohde, 2016; Demontis et al., 2019; Salum et al., 2014). While the diagnosis has been reported to have good sensitivity, specificity, and positive and negative predictive power (Faraone, 2005), it is nevertheless based on an arbitrary and artificial cut-off. This is evidenced partly by reports that people with subthreshold ADHD symptoms report clinically significant impairments in need of treatment, similar to those reported by people with the full ADHD diagnosis (Asherson, 2016; Hinshaw, 2018). These subthreshold effects show that ADHD

pathology can be seen as a dimensional phenomenon. In their systematic review of long-term prediction of ADHD persistence, van Lieshout, Luman, Buitelaar, Rommelse, and Oosterlaan (2013) concluded that using only categorical diagnostic measures as outcome measures was a considerable limitation in the research field up to that point. This is due to the increased risk of Type II errors, i.e. not uncovering effects that are actually there, when not including dimensional measures complementary to recording diagnostic information. Due to the evident dimensional nature of ADHD and other mental disorders, including quantitative measures when operationalising mental disorders was expressly included in the latest research strategy published by the National Institute of Mental Health in the USA, called the Research Domain Criteria (Insel et al., 2010).

1.3 Etiology

1.3.1 Genetics

Genes are a major causal factor in the development of ADHD, with heritability estimates from twin studies ranging between 70-80% (Asherson, 2016). Additionally, heritability estimates for continuous ratings of ADHD symptoms in the general population are similar to those found in for categorical diagnosis (Asherson, 2016; Hinshaw, 2018), further supporting the dimensional nature of the disorder. These genetic contributions to the disorder seem to originate in both common and rare genetic variants (Thapar & Cooper, 2016). Few single genes have been identified as causes of ADHD, and those identified seem to be of limited clinical significance. This is partly due to being indicated in other mental disorders as well, such as schizophrenia, and partly due to their small effect sizes when analysed individually. This makes sense considering the evident heterogeneity and dimensionality of ADHD. It appears that ADHD likely follows a multifactorial polygenetic threshold model of inheritance in which multiple genes, both rare and common, act additively or interactively with each other and environmental factors to produce the manifest phenotype of the disorder (Cortese, 2012; Demontis et al., 2019; McAuley, Crosbie, Charach, & Schachar, 2014).

1.3.2 Neurobiological substrates

When looking at the brain correlates of ADHD, both structural and functional neuroimaging techniques have been used to document abnormality. Structurally, decreased brain volume of

particularly the basal ganglia, but also the prefrontal cortex, has been robustly associated with ADHD (Friedman & Rapoport, 2015). These structural abnormalities correspond with the clinical presentation of the disorder, as the basal ganglia are thought to underlie reward processing, and the prefrontal cortex is central to neuropsychological functions deficient in ADHD, such as working memory and other executive functions (Norman et al., 2016). Structural abnormalities have also been investigated longitudinally, showing delayed prefrontal cortical development both in thickness and surface area (Shaw et al., 2007). Functional analyses have found ADHD-related dysfunction in multiple neuronal systems spanning both fronto-cortical and fronto-subcortical areas, extending previous pathophysiological theories of ADHD focused on exclusively prefrontal-striatal circuits (Castellanos & Proal, 2012; Cortese, 2012; Cubillo, Halari, Smith, Taylor, & Rubia, 2012).

On the neurophysiological level of analysis, dopamine is seen as the most central neurotransmitter in ADHD pathology. This aligns with several convergent research findings on genes, neuroanatomy, and medication effects (Wu, Xiao, Sun, Zou, & Zhu, 2012). Due to this, the early "dopamine hypothesis" claimed that dopamine was a central causal mechanism in ADHD, but has since become less prominent as ADHD likely has multiple causes (J. M. Swanson et al., 2000). More recent theories include the Behavioral Neuroenergetics Theory, which combines neuropsychological research findings on ADHD with detailed neurophysiological insight to posit that a lack of neuronal "energy" caused by reduced revitalisation of neurons by astrocytes is the underlying neural mechanism behind many evident deficits (Killeen et al., 2013).

1.3.3 Non-biological perspectives and controversies

ADHD has long been a controversial disorder (Hinshaw, 2018; Mayes, Bagwell, & Erkulwater, 2008; Pajo & Cohen, 2013; Visser & Jehan, 2009). While the current thesis is situated in the biomedical model of conceptualising the disorder, it is worthwhile to acknowledge alternative perspectives from other fields of science (Pajo & Cohen, 2013). Chief among concerns regarding the nature of the disorder is its rapid growth to become one of the most frequently diagnosed mental illnesses in children on a global scale (Conrad & Bergey, 2014; Singh, Filipe, Bard, Bergey, & Baker, 2013), with a high rate of receiving psychopharmacological treatment – around 50% (Visser, Lesesne, & Perou, 2007). Arguments have been made that ADHD is socially constructed by societal factors such as

high demands to perform well in a highly competitive and fast-paced society (Nielsen, 2017; Pajo & Cohen, 2013) and an increasing cultural intolerance in the educational sector of natural variation in children's behaviour (Singh, 2008). Additionally, some argue that the artificial, arbitrary, and dichotomous boundary between "healthy" and "ill" is particularly problematic with regards to ADHD (Mayes et al., 2008; Visser & Jehan, 2009), especially considering that some theoretical reviews find insufficient scientific grounds for cataloguing ADHD as a biomedical disorder of the brain (Sjöberg & Dahlbeck, 2018).

It is a less contentious fact that a degree of over-diagnosis, and related over-medication, has indeed been found in several Western countries, including Norway (Surén et al., 2018) and particularly the USA (Hinshaw, 2018). In their paper "The youngest get the pill", German researchers reveal that boys born later in the year are more likely to be diagnosed with ADHD and subsequently medicated, assumedly due to being somewhat unfairly compared to their older peers (Schwandt & Wuppermann, 2016). There is also considerable cross-national variance in medication rates, which in itself calls for considering the socio-culturally contextual nature of the disorder (Singh, 2008). Despite the criticism outlined here, there seems to exist a broad cross-disciplinal consensus, with strong research evidence, that ADHD is a valid and impairing disorder causing significant distress to patients and families, cross-culturally evident and appropriately diagnosed in the majority of cases (Bauermeister, Canino, Polanczyk, & Rohde, 2010; Faraone, 2005; Fayyad et al., 2017). It is important to avoid needless polarisation and strive for a holistic and integrated understanding of ADHD (Hinshaw, 2018; Lee & Neuharth-Pritchett, 2008; Singh, 2002).

1.4 Neuropsychology of ADHD

Neuropsychology is the branch of psychology studying cognition, emotion, and behaviour to elucidate underlying brain functioning, primarily by use of psychometric testing. Deficits in neuropsychological functions in ADHD have been widely documented (Frazier, Demaree, & Youngstrom, 2004; van Lieshout et al., 2013), with many theoretical models of the disorder seeing neuropsychological dysfunction as a core factor (Barkley, 1997; Sergeant, Geurts, Huijbregts, Scheres, & Oosterlaan, 2003; Sonuga-Barke, Bitsakou, & Thompson, 2010). Earlier models hypothesised the presence of a single underlying deficit (Barkley, 1997), while later models propose that there are distinct and separable pathways to dysfunction, not limited

to deficits in only a few neuropsychological domains (Castellanos, Sonuga-Barke, Milham, & Tannock, 2006; Sonuga-Barke et al., 2010).

A common delineation in neuropsychological taxonomies is the distinction between lower-order or bottom-up mental processes on the one side, and higher-order or top-down mental processes on the other. This distinction is based on the degree of conscious awareness and/or control one has over the mental activity being performed, and subsequently how effortful it is (Diamond, 2013; Halperin & Schulz, 2006). Measuring, or even conceptualising, these functions separately is challenging due the task-impurity problem, namely that most tests of higher-order functions involve lower-order processes as well due to their overlapping and hierarchical nature (Toplak, West, & Stanovich, 2013). Despite this, the distinction between lower- and higher-order cognition is a useful one and will be used as a framework here when deliberating the neuropsychology of ADHD and its inherent interrelatedness. As the higher-order executive functions are of particular interest in ADHD research, they will be discussed more in-depth.

Before delving into the current evidence of neuropsychological deficits characteristic of ADHD, it is appropriate to consider the fact that ADHD is best regarded as a neuropsychologically heterogeneous disorder (Coghill, Seth, & Matthews, 2014; Luo, Weibman, Halperin, & Li, 2019; Mostert et al., 2015). Only a proportion of ADHD subjects demonstrate deficits on any one particular neuropsychological task, with substantial overlap between ADHD and typically developing children on various tasks heavily implicated in ADHD (Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005). Some of this heterogeneity may also be "nested" in normal variation of neuropsychological diversity (Fair, Bathula, Nikolas, & Nigg, 2012). Due to this evident heterogeneity, it is unclear whether these neuropsychological deficits can be seen as endophenotypes of ADHD symptoms or if they have an epiphenomenal relationship (van Lieshout et al., 2018). This means that while neuropsychological deficits have been posited to function as potential mediators of the underlying causal effects of genes on behaviour (endophenotype), it could be the case that they instead share a common underlying cause with symptoms (epiphenomenal). The fact that some people with ADHD exhibit no deficits on tests also highlights the difference between cognitive deficits as expressed in manifest behaviour, which serves as the basis of diagnosis, and as shown on tests. One does not necessarily implicate the presence of the other.

1.4.1 Intelligence

General cognitive ability, often called intelligence, is not a type of lower-order cognition, but rather the end result of the interplay between various both lower- and higher-order neuropsychological functions (McGrew, 2009). Lower intelligence has been robustly reported in children with ADHD, with an average discrepancy corresponding to approximately 9 IQ points (Frazier et al., 2004; Hervey, Epstein, & Curry, 2004; Moffitt et al., 2015; van Lieshout et al., 2013). Since measures of overall cognitive ability are known to rely heavily on executive functions, interpretation of the source of these IQ deficits is difficult (Frazier et al., 2004). IQ has also been indicated to have a potential moderating role in ADHD throughout development, for instance making lower-IQ children with ADHD more susceptible to preschool language delays (Rohrer-Baumgartner et al., 2014) and compensating for executive dysfunction in adults with ADHD (Milioni et al., 2017).

1.4.2 Memory

Memory deficits have been evidenced in ADHD (Skodzik, Holling, & Pedersen, 2017). The human memory system can be divided into separate, but partially overlapping, subdomain functions including the short-term, episodic, procedural, verbal, visuospatial, and working memory systems. The respective roles of lower- and higher-order memory functions in memory deficits seen in ADHD are unclear, and they are often insufficiently distinguished from each other in the literature. For instance, the fact that short-term memory is a prerequisite of working memory, and not analogue to it, is often overlooked (Diamond, 2013). A portion of memory deficits in ADHD may come from employing less effortful learning strategies, and not to underlying storage dysfunction (Egeland, Nordby Johansen, & Ueland, 2010). This would constitute an interaction between lower-order storage systems and deficient higher-order self-oriented functions connected to the successful conscious application of strategies. But higher-order dysfunction is unlikely to account for all the memory impairments evidenced in ADHD, as deficits have been found in both short-term memory, long-term memory, and visual memory, while verbal memory seems surprisingly unaffected (Rhodes, Park, Seth, & Coghill, 2012).

1.4.3 Attention

As one would expect, deficient attention has been robustly documented in neuropsychological testing of ADHD samples (Hervey et al., 2004). Especially the subdomain of selective attention has been implicated (Huang-Pollock, Nigg, & Carr, 2005), which is the ability to consciously choose what stimulus you wish to attend to, while excluding other stimuli from conscious awareness (Diamond, 2013). Many individuals with ADHD have the ability to become completely engrossed in something they find interesting and struggle to detach themselves from it, which is sometimes called "hyperfocus" (Hinshaw, 2018). These issues would be related to the ability to self-regulate one's attention and would therefore fall under the umbrella of the higher-order functions. Some have indeed proposed that the attention impairments seen in ADHD are most plausibly explained by top-down regulatory deficits (Wilding, 2005). On the other hand, there is evidence that dysfunction in lower-order early visual processing system may create negative cumulative effects on higher-order attention processes (Lenz et al., 2010; Ríos, Periáñez, & Muñoz-Céspedes, 2004). Deficits in early visual perception have also been documented electrophysiologically occipital brain areas in children with ADHD (Nazari et al., 2010), and abnormalities in sensory processing may be more common in preschool children with ADHD symptoms (Cheung & Siu, 2009). These examples highlight the possible interplay of lower-order neuropsychological functions and higher-order executive functions in the complexity of attention.

1.4.4 Motor skills

These indications of more "primitive" neurologically-determined functions also being implicated in ADHD is supported by evidence of noteworthy deficits in processing speed and motor control as well (Faraone et al., 2015). These often seem to be intertwined with deficits of a sensory nature as well. Approximately half of all cases of childhood ADHD may have motor and sensory difficulties consistent with developmental coordination disorder (Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005), making them eligible for inclusion under the DAMP term (Gillberg, 2003b). It is standard psychiatric practice that so-called "neurological soft signs" such as clumsiness, poor balance and poor sensory integration can support a diagnosis of ADHD, despite not being included in the core diagnostic criteria (American Psychiatric Association, 1987; Chan et al., 2010). A recent review noted that although there is a strong association between motor impairments and ADHD, the underlying

mechanisms and their causal relationships are unclear, calling for longitudinal research for clarification (Goulardins, Marques, & De Oliveira, 2017). Additionally, Goulardins and colleages (2017) argued for the importance of such deficits in ADHD, as they may lead to significant academic, social, and emotional consequences.

1.4.5 Executive functions

Executive functions (EFs) is the umbrella term used to encompass a broad collection of top-down mental processes needed to self-regulate oneself in order to perform flexible, goal-oriented behaviour, such as concentrating and paying attention. The EFs are employed at times when following automatic habits or relying on instinct or intuition would be insufficient or inappropriate, and are therefore broadly recognised as essential mental functions in diverse areas of functioning (Castellanos et al., 2006; Diamond, 2013). While the current study utilises the model outlined by Diamond (2013), another theoretical framework of note is the unity-diversity framework (Miyake & Friedman, 2012). It efficiently highlights the duality of EFs. They are interrelated enough that one can hypothesise an underlying common construct and simultaneously separable enough to warrant discussing them as separate, although overlapping, entities.

Deficits in EFs have been so strongly associated with ADHD that several hypotheses have been posited that ADHD is a disorder of the EFs (Biederman et al., 2009; Castellanos et al., 2006; Nigg et al., 2005). Their importance is shown by their associations with multiple adverse outcomes related to ADHD, such as obesity (Smith, Hay, Campbell, & Trollor, 2011), increased externalizing and internalizing problems (Brunnekreef et al., 2007), and lower academic achievement (Martinussen et al., 2005; Martinussen & Major, 2011). At the same time, the EFs are “fragile” in the sense that they are easily affected negatively by detrimental situational factors, such as sleep deprivation (Barnes, 2012), low physical fitness (Chaddock et al., 2012), and stress (Qin, Hermans, van Marle, Luo, & Fernández, 2009). This lays the groundwork for considerable negative spiralling effects and implies that the EFs could have a potentially mediating role of the association between ADHD and adverse outcomes in the lifespan. Despite cognitive flexibility (i.e. the capacity to efficiently shift one’s perspective and focus) being an important subgroup of EFs (Diamond, 2013), inhibition and working memory (WM) will be covered in detail in this section due to their strong

relationships with ADHD (Alderson, Kasper, Hudec, & Patros, 2013; McAuley et al., 2014; Nigg et al., 2018).

Inhibition

Inhibition used to be at the centre of the understanding of ADHD as a potential core deficit (Barkley, 1997; Nigg, 2001), particularly because of the strong research support of deficits on neuropsychological tests measuring primarily inhibition, such as Go/No-Go tasks and the Stop task. This has since been expanded into a more nuanced view (Castellanos et al., 2006; Sonuga-Barke et al., 2010). Nevertheless, inhibition deficits are arguably a central dysfunction in ADHD, and more so than in other mental disorders of childhood (Sonuga-Barke et al., 2010; Willcutt, Sonuga-Barke, Nigg, & Sergeant, 2008). Inhibition can be divided into separate parts, with behavioural inhibition being a salient theme in ADHD with its conceptual connection to hyperactivity-impulsivity. A segment of inhibition that is more easily overlooked is cognitive inhibition, the ability to focus on whatever mental task your mind is performing (Diamond, 2013). The part of our mind performing such mental tasks is usually referred to as the WM's central executive, as discussed below. As cognitive inhibition in turn needs the WM to hold information and goals in mind to direct inhibition, this illustrates that the EFs are closely interrelated and co-dependent. Recent research has focused on the subjective experiences of excessive mind-wandering in ADHD (Asherson et al., 2016), which could possibly be interpreted as the subjectively experienced counterpart of neuropsychologically documented deficits of cognitive inhibition.

Working memory

Definitions of working memory (WM) are contentious (Rhodes, Coghill, & Matthews, 2004), but one of the most common conceptualisations is Baddeley's multicomponent model (Baddeley, 2003, 2007; Baddeley, Logie, Bressi, Della Sala, & Spinnler, 1986). This model posits that WM is the capacity to hold information in mind in two storage systems, the phonological loop and visuospatial sketchpad, and manipulate it by use of a central executive (Martinussen et al., 2005). Martinussen and colleagues (2005) attempted to integrate and simplify earlier models by renaming the central executive as the "manipulation WM" and the memory subsystems as the "simple storage", which is more comparable to the concept of short-term memory reviewed above. More simply and generally put, one can say that WM is the capacity of working with information no longer perceptually present (Diamond, 2013). WM deficits in ADHD have been documented in meta-analyses to exist in children

(Martinussen et al., 2005; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005) and adults (Alderson et al., 2013) with ADHD. WM deficits seem to be in closer association with ADHD symptoms of inattention than hyperactivity (Martinussen & Tannock, 2006), and ADHD deficits in WM are stronger in the visuospatial than verbal modality (Martinussen et al., 2005; Rapport et al., 2008). The centrality of WM in ADHD was recently explored further in findings showing that WM deficits mediated 43% of the polygenic risk score from common genetic variants, showing that unlike other neuropsychological functions, deficits in WM may be a promising endophenotype for the disorder (Nigg et al., 2018).

Rating measures of executive functions

It has become increasingly evident that neuropsychologically operationalised test measures of EFs only correlate weakly with actual real-life outcomes or informant ratings, either given by the clinician, parent or individual with ADHD (Barkley & Murphy, 2011; Roth, Isquith, & Gioia, 2014). In other words, EF test measures seem to have limited ecological validity. The reason for this could be that neuropsychological testing happens in highly structured settings that, by design, put only limited demands on EFs to impose organisation and structure to the task at hand (Salthouse, Atkinson, & Berish, 2003). In addition to their scaffolding removing the need for self-structuring, EF tests lack the affective significance experienced in everyday life (Toplak et al., 2013). This makes them less sensitive to "hot" subdomains of EFs related to motivation, decision-making, and reward processing. These are posited to be dissociable deficits in ADHD from "colder" EF deficits that are measured by standardised test measures in less emotionally salient testing contexts (Skogli, Egeland, Andersen, Hovik, & Øie, 2014; Sonuga-Barke et al., 2010). This is an important area of functioning to be cognisant of, as emotional dysregulation has emerged as a potentially central but previously overlooked impairment in ADHD (Graziano & Garcia, 2016), especially in adults (Hirsch, Chavanon, Riechmann, & Christiansen, 2018). In other words, it is in everyday life that executive dysfunction, both the "cool" structuring kind and "hot" emotional kind, is naturally expressed, and measuring it in calm, quiet testing laboratories is therefore not always optimal.

To address these issues, the Brief Inventory of Executive Functions (BRIEF) was created as an ecological rating inventory with an explicit focus on the everyday challenges that arise from EF deficits (Gioia, Isquith, Guy, & Kenworthy, 2000b). While not the only rating measure of EFs, it is the most frequently used, with the strongest empirical support (Roth et al., 2014; Toplak et al., 2013). Its inherent structure includes eight subdomains discovered by

use of factor analysis, which create the meta-indices Behavioral Regulation Index (BRI) and Metacognitive Index (MI). These indices can be seen as measures of "hot" and "cool" EFs, respectively, although later factor analyses showed that a three-factor model is more accurate, dissociating emotional and behavioural regulation (Egeland & Fallmyr, 2010). Elevated rates on especially the Working Memory and Inhibit subdomains in ADHD have consistently been reported since the BRIEF's conception (Isquith & Gioia, 2000). Because of this, the BRIEF has shown good clinical and diagnostic utility (McCandless & O' Laughlin, 2007) across various age groups of ADHD (Mahone & Hoffman, 2007; Toplak, Bucciarelli, Jain, & Tannock, 2008).

Behaviour ratings and test measures of EFs correlate only to a small extent, or not at all, but seem to explain separate parts of functional outcomes related to executive dysfunction (Toplak et al., 2013). While test measures show how the participant can perform under optimal conditions, ratings more realistically illustrate how well the respondent is actually doing in their day-to-day life (Burgess et al., 2006). This serves as an illustration of the trade-off between internal and external validity researchers must consider when selecting assessment instruments. This mirrors the parallel trade-off between sensitivity and specificity: test measures of EFs appear to have low sensitivity, making false negatives likely when assessing people with probable executive dysfunction (Egeland, 2010), but 25% of healthy controls measured by the BRIEF receive a T-score above 65 one any one subdomain (Gioia, Isquith, Guy, & Kenworthy, 2000a), showing reduced specificity. While this means that the BRIEF has limited utility as a screening instrument, it is less of an issue in clinical settings, where assessment is conducted indicatively. BRIEF scores are predictive of later quality of life, occupational functioning, and antisocial behaviour in ADHD, more so than test measures of EFs (Barkley & Murphy, 2010; Barkley & Murphy, 2011; Stern, Pollak, Bonne, Malik, & Maeir, 2013). This makes the BRIEF a potentially interesting outcome measure in its own right due to its potential mediating effects of adverse outcomes seen in ADHD.

1.5 Treatment of ADHD

A vast amount of research has consistently and robustly supported stimulant medication treatment for ADHD, especially short-term and up to 3 years (Rajeh, Amanullah, Shivakumar, & Cole, 2017). There is some evidence of sustained benefits in the long run (Faraone et al., 2015; Fredriksen, Halmøy, Faraone, & Haavik, 2013), but this is not universally found in

large longitudinal studies (Molina et al., 2009). It is also yet unclear whether medication has a beneficial impact on cognition, and if so, whether such effects are direct or indirect through alleviating symptoms (Coghill, Seth, Pedroso, et al., 2014; Mostert et al., 2015; Uchida, Spencer, Faraone, & Biederman, 2018).

A wide range of psychological and psychosocial interventions have been developed for the treatment of ADHD and are supported by research (Molina et al., 2009; Sibley, Kuriyan, Evans, Waxmonsky, & Smith, 2014), while not to the same extent as medication, and with some inconsistencies (Rajeh et al., 2017; Sonuga-Barke et al., 2013; Thapar & Cooper, 2016). Research evidence also suggests that behavioural interventions may have superior effects on functional impairment measured by other parameters than symptoms, such as parental functioning or comorbid conduct problems (Daley et al., 2014; Pfiffner, 2014). European international treatment recommendations (National Institute for Health and Care Excellence, 2018) advise a stepwise therapeutic approach so that psychosocial interventions are tried ahead of, or in combination with, pharmacological treatment, and that combined therapy is implemented in cases that need medication (Asherson, 2016). This is similar to the content of the national recommendations ("Nasjonal retningslinje") for ADHD in Norway, which lists numerous types of interventions that may be attempted in ADHD, and highlights the potential negative sides to utilising medication (Helsedirektoratet, 2018). Despite this, a 2016 report by the Norwegian Institute of Public Health reported that as many as 80% of children with a diagnosis of ADHD had been given stimulants on at least one occasion (Ørstavik et al., 2016). Such potential overreliance on medical treatment contrary to recommended policies has been shown in the USA as well, and may be related to whether patients are in the care of primary care providers or mental health specialists (Walls, Allen, Cabral, Kazis, & Bair-Merritt, 2018).

1.6 ADHD in the lifespan

1.6.1 Adverse life outcomes

The research literature of longitudinal follow-up studies measuring various outcomes in ADHD is extensive (Franke et al., 2018). Studies have reported a host of various adverse outcomes of ADHD in the lifespan on physical, psychological, social, academic, economic, criminal and occupational domains, of severe consequence to society and the individual

(Halmøy, 2011; Hinshaw, 2018; Instanes, Klungsøyr, Halmøy, Fasmer, & Haavik, 2018; Jangmo et al., 2019; Klein et al., 2012; Mohr-Jensen & Steinhausen, 2016; Øie, Sundet, & Ueland, 2011). Although whether these effects are causal or only correlational is yet unclear regarding several of these associations (Erskine et al., 2016), it is robustly documented that a diagnosis of ADHD in childhood is associated with numerous severe adverse life outcomes. More research is needed to elucidate the respective contributions of various factors and the mechanisms underlying developmental pathways leading to said outcomes.

As a developmental disorder, ADHD is manifested differently during different stages of the lifespan (Franke et al., 2018). In pre-school, ADHD symptoms include abnormal development of gross motor skills, language (Rohrer-Baumgartner et al., 2014), and neurological soft signs (Chan et al., 2010). In school age, impairments become more readily apparent through trouble coping with the demands of school, both when it comes to sitting still (hyperactivity) and following the content of the schoolwork (attention deficits). In adolescence, struggling to keep up with gradually more complex social hierarchies and interactions might become evident. At all stages of life, as different developmental stages are reached, with corresponding developmental tasks to be solved, ADHD pathology interacts uniquely with each stage (Asherson, 2016).

1.6.2 Course of the disorder

Since research consistently ties ADHD to adverse outcomes through the lifespan, understanding the different courses of ADHD, and possibly even their underlying developmental pathways, becomes a matter of importance. While ADHD symptoms in general seem to decline with age (Cheung et al., 2015), it is a consistent finding that this seems to apply to symptoms of hyperactivity to a larger degree than to symptoms of inattention (Asherson et al., 2016), which seem relatively stable with advancing age (van Lieshout et al., 2013). Thus, many children with a combined subtype of ADHD present with predominantly inattentive symptoms as adults. Despite the clear evidence of the central role of neuropsychological deficits in ADHD, the course of the disorder seems to be almost entirely independent from the course of these deficits in longitudinal studies (McAuley et al., 2014; van Lieshout et al., 2018). This means that ADHD symptoms and neuropsychological deficits may improve or deteriorate independently from one another (Biederman et al., 2009).

As mentioned previously, estimates of the persistence rate of the disorder vary widely, between 5-80% (Asherson et al., 2016; van Lieshout et al., 2013). While a meta-analysis of follow-up studies found a rate of only 15% (Faraone, Biederman, & Mick, 2006), a recent 6-year follow-up study found that as many as 84% persisted from adolescence into adulthood in a large clinical sample (van Lieshout et al., 2016). They cited severity and combined subtype as potential reasons for this higher estimate. This continued irregularity has been pointed out as a weakness in the literature (Franke et al., 2018). Part of this variation can be attributed to differences in defining remission, since it can be operationalised and measured as either diagnostic or functional, full or partial (Biederman, Petty, Clarke, Lomedico, & Faraone, 2011). Variation in the subtype and severity level of the ADHD studied may also affect persistence rates found (Asherson et al., 2016). Also, follow-up intervals vary widely, with some studies investigating persistence from childhood into adolescence and others from adolescence into early or middle adulthood. Increased knowledge of the longitudinal course of ADHD has led to some theorists arguing that adult ADHD is an underdiagnosed and undertreated disorder (Asherson et al., 2016). Reports have suggested that individuals with remittent ADHD still experience clinically significant impairment (Thapar, Cooper, & Rutter, 2017), including a meta-analysis of outcome studies (Faraone et al., 2006).

Influential and controversial new findings have recently come from three large, independent, longitudinal, and statistically powerful cohort studies: the Dunedin Multidisciplinary Health & Development Study (Moffitt et al., 2015), the Pelotas (Brazil) Birth Cohort Study (Caye, Rocha, et al., 2016), and the Environmental Risk (E-Risk) Longitudinal Twin Study (Agnew-Blais et al., 2016). Their findings are two-fold. Firstly, they all found much lower persistence rates of childhood ADHD into adulthood than most studies do, namely 5%, 17%, and 22%, respectively. Secondly, the vast majority of adult cases of ADHD did not evidence ADHD symptoms as children (87%, 87%, and 67%). These findings have severe implications for the central assumption in the research field that ADHD is a child-onset neurodevelopmental disorder at all and thus opens up the discussion on the developmental trajectory of ADHD with renewed force. For instance, it has been suggested that adult and childhood ADHD may be separable disorders with distinct developmental pathways (Caye, Rocha, et al., 2016). However, one literature review argues concisely for why such conclusion would be pre-emptive (Franke et al., 2018). Also, as these three studies are population-based with large cohorts, the amount of information of symptoms and neuropsychological deficits among the

participants are limited, which calls for longitudinal studies with more extensive information at both baseline and follow-up.

1.6.3 Predicting the persistence of ADHD

The research field is seemingly still in the process of understanding the long-term persistence rate of ADHD. It is clear, however, that for some individuals, ADHD is a persistent disorder with important social, vocational and health-related ramifications. Additionally, such outcomes are more prominent in individuals whose ADHD persists (Agnew-Blais, 2017). As such, being able to identify which cases of ADHD in childhood are at risk of a chronic course is of strong clinical relevance – in other words, the research field needs to identify statistical predictors of persistence and remission. Unfortunately, the currently available knowledge base from research attempting to answer this question is limited and largely inconsistent (Franke et al., 2018). The only systematic review performed on predictive studies on ADHD persistence identified this field as an "overlooked question" constituting only 0.08% of the published literature on ADHD (Caye, Spadini, et al., 2016). Cayes and colleagues' review and meta-analysis is the first to review the entire field of predictive studies of ADHD persistence, and it is the author's impression that van Lieshout and colleagues' review (2013) is the only one specifically targeting neuropsychological predictors.

Symptom severity and comorbidity predict persistence

One of the most consistent predictors of ADHD persistence has been symptom severity (Kessler, Adler, Barkley, et al., 2005), in addition to comorbidities of conduct disorder and depression (Caye, Spadini, et al., 2016). These predictors were all found by Biederman and colleagues (2011) in their well-described longitudinal ADHD sample in their 11-year predictions, in addition to maternal mental health, a family history of ADHD, and psychosocial adversity. An international World Health Organization study made similar findings, but did not find evidence of the predictive effects of psychosocial adversity (Lara et al., 2009). The predictive effects of symptom severity found by both the Cheung (2015) and Biederman research groups on persistence included both baseline parental rating measures and objective actigraph measures. Cheung and colleagues also found socioeconomic factors to be predictor, but the largest prospective study of ADHD in the world, the Multimodal Treatment (MTA) study, found no predictive effect of household income after 16 years. It did, however, confirm the predictive effects of childhood comorbidity and parental mental health.

Neuropsychological predictors of persistence

Both Caye and colleagues (2016) and van Lieshout and colleagues (2013) reached the conclusion that as of yet, there is little evidence of predictive effects of neuropsychological functions on ADHD persistence, apart from across timespans of only a few years within childhood. Only IQ may have a protective role (Cheung et al., 2015; Gao et al., 2015), but this was not supported by the MTA study (Roy et al., 2016). However, studies often include only few, or zero, neuropsychological measures apart from IQ. Two recent studies included more comprehensive test batteries (Sjöwall, Backman, & Thorell, 2015; van Lieshout et al., 2017). Sjöwall and colleagues had a follow-up period of 13 years (ages 5-18) and found that only WM was a significant predictor of ADHD symptoms. This was replicated by van Lieshout and colleagues, although their study only a 6-year follow-up interval and had a more diverse age range (5-19 years of age at baseline). While the impressive Dunedin, Pelotas, and E-Risk research projects covered long time periods, they do not appear to have included extensive neuropsychological test batteries. To the author's knowledge, no study has thoroughly investigated the predictive value of neuropsychological functioning over more than two decades and into adulthood. There is a lack of convincing studies of the role of neuropsychological measures in predicting long-term persistence, as baseline measures are too general or simple. This calls for studies in which the participants go through extensive examinations at baseline.

Limitations in the evidence of neuropsychological predictions of ADHD persistence

In addition to the lack of detailed neuropsychological assessments, there are several methodological shortcomings in the literature on neuropsychological predictors of ADHD persistence (van Lieshout et al., 2013). For one, many studies include only one or a few tests, which leads to limited internal validity as neuropsychological functions may overlap. Also, focusing only on diagnosis (a dichotomous "yes" or "no" question), and not also dimensional symptom measures, when predicting pathological outcomes reduces sensitivity to subthreshold effects. Omitting functional outcomes related to symptoms may also reduce the clinical significance of research findings. Additionally, many studies use ADHD cohorts with substantial internal age differences. Even when statistically controlling for the effect of age, the fact that developmental maturation processes relevant to ADHD outcomes have occurred in subsets of a sample, and not in others, remains an issue (van Lieshout et al., 2017). This is because the substantial neuropsychological development in adolescence sometimes occurs in leaps and bounds rather than in a linear fashion (Geier, 2013).

Another inconsistency in the research field seems to be lie in the use of the word "long-term". Studies investigating and/or predicting outcome and/or persistence rates in ADHD seem to include follow-up intervals between four to 40 years, the majority seemingly between six and ten. The developmental period studied seems to be either between childhood ADHD and adolescence (Sjöwall, Bohlin, Rydell, & Thorell, 2017) or between adolescence and early adulthood (Biederman et al., 2011). While understandable given the prohibiting costs and challenges inherent to longitudinal research projects, this nevertheless constitutes a hole in the literature. There seems to be a paucity of studies with longer follow-up intervals predicting the adult outcomes of children and youth with ADHD.

1.7 Research aims and hypotheses

To summarise, much is known about the somatic, psychological, and social outcomes of ADHD across the lifespan. Recent studies have shed new light on the disorder in the long-term, bringing considerable uncertainty regarding its persistence rates. Being able to predict the long-term persistence rates of the disorder is of strong clinical interest, as it could enable individually tailored long-term interventions and treatment planning. Despite this, there is a paucity of long-term predictive studies of persistence with follow-up periods of more than 10 years. While the evidence of the role of symptom severity and comorbidity seems clear, the role of neuropsychological predictors seems less so, as older studies have included few neuropsychological test measures. To the author's knowledge, no study has included executive dysfunction as an outcome measure parallel to diagnostic and symptomatic outcome in a long-term predictive study of ADHD, despite the considerable research evidence showing the centrality of EFs in the disorder. The research presented in the current thesis serves as a continuation of studies on a well-described and thoroughly neuropsychologically tested clinical cohort of ADHD (Øie & Rund, 1999; Øie, Sundet, & Rund, 1999, 2010; Øie et al., 2011). This 23-year follow-up is the first to investigate predictors of persistent ADHD pathology and executive dysfunction in this research sample.

The goal of the current study is to expand the research literature examining the predictive abilities of neuropsychological and symptom measures by investigating a longer time period, including a larger neuropsychological test battery, and examining the long-term effects of these predictors on executive dysfunction as well as diagnosis and symptoms. Executive dysfunction is worth including as an outcome measure as it is associated with adverse

outcomes disproportionately experienced by people with ADHD, particularly when ecologically measured by use of ratings. ADHD persistence is examined as both continuous measures of symptoms and function as well as a categorical diagnosis, to both account for the dimensional nature of ADHD and include the clinical value of a dichotomous diagnosis.

The research aims of the present study are to investigate:
1) What proportion of the sample retains their ADHD diagnosis after 23 years and how the remitters fare compared to both the retainers and the healthy control group.
2) To what extent neuropsychological measures and symptom severity in adolescence can predict diagnostic persistence after 23 years.
3) To what extent neuropsychological measures and symptom severity in adolescence can predict ADHD symptom severity after 23 years.
4) To what extent neuropsychological domain measures and symptom severity in adolescence can predict executive dysfunction after 23 years.

Due to the discrepancy between the thoroughness in earlier predictive studies and the present study when it comes to the baseline neuropsychological assessment, no hypotheses are stated on these predictors. The literature supplies a broad range of persistence estimates but indicates that more severe samples may persist more. The stated hypotheses are therefore:
1) The persistence rate in adulthood will be around 50%. Remitters will report fewer symptoms and less executive dysfunction than persisters, but more than healthy controls.
2) Baseline symptom severity will predict diagnostic persistence in adulthood. No hypothesis is made regarding baseline neuropsychological predictors.
3) Baseline symptom severity will predict symptom severity in adulthood. No hypothesis is made regarding baseline neuropsychological predictors.
4) Baseline symptom severity will predict executive dysfunction in adulthood. No hypothesis is made regarding baseline neuropsychological predictors.

2 Methods

2.1 Participants

The present study is part of a larger research project initiated in 1992 (T1) which, at the time, aimed to compare neuropsychological functions in adolescents with early onset schizophrenia with an ADHD sample and healthy controls (Øie & Rund, 1999). The study was later expanded to longitudinally investigate the course of neuropsychological deficits in the two patient groups and their relation to functional outcomes (Øie et al., 2010; Øie et al., 2011). The whole research sample was reassessed 13 years (T2) and 23 years later (T3). The focus of the current study will be on the ADHD group at T1 and their outcomes at T3, while also looking at the healthy control group for comparison. Nineteen of the original 20 subjects in the ADHD sample were available for assessment at T3. One subject was deceased before T2 (information obtained from the Norwegian Cause of Death Registry). Twenty-six of the original 30 healthy controls were available for re-testing at T3. Of the four who were not available, one was deceased from medical issues (information obtained from the Norwegian Cause of Death Registry), two no longer wished to participate, and one had developed an illness incompatible with participation as a healthy control.

2.1.1 Baseline

Thorough descriptions of the demographic information of the research sample at T1 and T2 can be found in earlier publications (Øie & Rund, 1999; Øie et al., 2010; Øie et al., 2011). The ADHD sample was mostly recruited from another research project started by psychiatrist Pål Zeiner at the National Centre for Child and Adolescent Psychiatry (NCCAP) in Oslo, while the rest were recruited from other outpatient clinics in Oslo. Diagnoses were made based on fulfilling the required eight diagnostic criteria of the DSM-III-R (American Psychiatric Association, 1987), by mental health professionals using semi-structured clinical interviews and standardized rating scales. Their ADHD symptoms occurred both at home and at school and had occurred between the ages of six and 10 as assessed by the Parent's Rating Scale (P. H. Wender, Reimherr, Wood, & Ward, 1985). Diagnoses of ADHD subtypes were not made at T1, as they were first introduced in the DSM-IV. Comorbidities included oppositional defiant disorder (N=9), developmental reading disorder (N=2), and concurrent

oppositional defiant disorder and developmental reading disorder (N=3), corresponding with frequent comorbid diagnoses in the patient population (Franke et al., 2018). The mean age of the ADHD group at first assessment was 14.1 years, with a standard deviation of 1.5. The ADHD sample was significantly younger than the healthy controls ($P<0.05$). The ADHD group was exclusively male, which reflects the fact that the gender disparity seen in clinical and research practice was greater at this time than it is today (Biederman & Faraone, 2004). Twelve of the participants with ADHD received stimulant medication (11 used methylphenidate and one used dextroamphetamine) which was discontinued at least 24 hours ahead of testing. One of the subjects with ADHD received a small dose of haloperidol (1mg/day) due to tics.

The participants placed in the healthy control group (HC group) were recruited from schools from the local area and attended regular schooling at normal grade level. All research participants underwent the Child Behavior Checklist (CBCL; Achenbach & Edelbrock, 1991) to screen for mental health problems, with mothers acting as informants. Healthy controls with a raw score over 45 were excluded from the study (Øie & Rund, 1999), which was a cut-off at the 90th percentile set according to American norms and corrected for sex and age (Øie, Rund, Sundet, & Bryhn, 1998). The mean age of the healthy control group at T1 was 15.8 years, with a standard deviation of 1.7. At T3, all the 26 individuals in the healthy control group available for reassessment at T3 still fulfilled criteria to serve as healthy controls after being screened with the Mini-international neuropsychiatric interview (M.I.N.I.; Sheehan et al., 1998) and through the use of an unstructured clinical interview investigating whether they had a treatment-needing mental disorder. The gender distribution of the HC group was evenly male and female.

Both the ADHD and HC groups were screened at T1 by use of questionnaires and medical records to exclude participants with a history of substance abuse, head injury with loss of consciousness, and medical disease that was likely to affect central nervous system functions. They were also all screened for intellectual disability, with a score below 70 on the Wechsler intelligence scale for children revised (WISC-R; Wechsler, 1974) serving as the exclusion criteria threshold, computed by use of four WISC-R subtests.

2.1.2 Follow-up

At T3, one member of the ADHD group used stimulants (Ritalin), three used a small dose of atypical antipsychotics (Seroquel), and one used antidepressants (Venlafaxine). While 19 of the original 20 participants were retained in the study, only 11 of them retained their ADHD diagnosis. Four of these 11 individuals fulfilled DSM-IV diagnostic criteria for only ADHD, combined or inattentive type, while seven of them also fulfilled criteria for other mental disorders; five for depression or anxiety, one for a bipolar disorder, and one for Tourette's. As data regarding ADHD subtype was not collected at T1, comparisons of subtypes across time cannot be made. As reported in Øie and colleagues (2010; 2011), at the 13-year follow-up (T2) the ADHD group was characterised by poor social and global adaptive functioning compared with the HC group. They were largely unemployed and less educated, and more likely to be single. Seventy-four percent reported substance abuse having occurred in the 13 years before T2, and 42% had been convicted of a crime. Furthermore, four participants received diagnoses of antisocial personality disorder, and two received diagnoses of comorbid bipolar disorder. These findings regarding comorbidity is in line with empirical evidence stating that ADHD is associated with an elevated risk of being diagnosed with bipolar or personality disorder in adulthood (Franke et al., 2018). At T2, four participants in the ADHD group no longer qualified for a diagnosis.

2.2 Neuropsychological measures

A comprehensive neuropsychological test battery was used at T1, using known, standardized tests. While they also underwent the same testing at T2 and T3, potential learning effects are irrelevant since no neuropsychological measures obtained at T3 are used in the present study. The neuropsychological tests from T1 included in this study were the Wisconsin Card Sorting Test (Heaton, Chelune, Talley, Kay, & Curtiss, 1981), Digit Span Distractibility Test (Oltmanns & Neale, 1975), Kimura Recurring Figure Test (Kimura, 1963, 1967), California Verbal Learning Test (Delis, Kramer, Kaplan, & Thompkins, 1987), Trail Making Test A and B (Reitan & Wolfson, 1985), Grooved Pegboard (Matthews & Klove, 1964), Seashore Rhythm Test (Lezak, Howieson, Loring, & Jackuns, 1995; Seashore, Lewis, & Saetveit, 1960), Dichotic Listening (Hugdahl & Andersson, 1986; Øie et al., 1998), Backwards Masking (Rund, Øie, & Sundet, 1996), and the Digit Span, Coding, Similarities, and Block Design tests from WISC-R (Wechsler, 1974). Various indices and measures from this battery

were used to construct eight composite scores to represent eight neuropsychological domains, as described in Øie et al. (2010). In short, z-scores were computed for all tests using the original HC group's scores' means and standard deviations, only including subtests showing adequate psychometric properties in the retest sample and modified according to Saykin et al. (1991; 1994). These eight composite scores were named Executive function, Visual memory, Verbal memory, Visuomotor processing, Motor coordination, Auditory attention, Selective attention, and Visual attention. In cases where higher scores indicated dysfunction, their values were inverted to assure that high scores on the composite scores always indicated better function. An IQ estimate was constructed using results from the Similarities and Block Design tests as a ninth neuropsychological domain. The z-scores were based on the original HC group at T1, consisting of 31 people, meaning that the current HC group has slightly deviating scores on three domains. All tests included in this neuropsychological test battery have shown good reliability, as that was an inclusion criteria when selecting test measures at the outset of the research project (Bakker, Van Der Vlugt, & Claushuis, 1978; Charter & Webster, 1997; Goldstein & Watson, 1989; Harper & Kraft, 1986; Kimura, 1980; Spreen & Strauss, 1991; Wechsler, 1974; Øie & Rund, 1999).

2.2.1 Auditory attention

The Seashore Rhythm Test (Lezak et al., 1995; Seashore et al., 1960), the Digit Span subtest from the WISC-R (Wechsler, 1974), and the Digit Span Distractibility Test (Oltmanns & Neale, 1975) were used to construct the Auditory attention domain. The Seashore Rhythm Test is a measure of nonverbal auditory perception and attention/concentration deficits, in which the participant is presented with 30 similar and dissimilar pairs of musical beats. The measure collected is the number of correct identifications of similarity/dissimilarity made by the participant out of the 30. In the Digit Span test from the WISC-R, participants are auditorily presented increasingly longer series of digits which they are asked to repeat back to the test technician. In the second part of the test the participant needs to repeat the listed numbers in reverse order. The maximum number of digits the participant can correctly repeat, both forwards and backwards, are the measures from this test used here. The last test to be included in this domain variable was from the Digit Span Distractibility Test, during which short strings of digits are read to the participant, who is asked to repeat them back in numeric order. The test has two conditions: one in which only the target digit string is presented, and one in which distractor digits are interjected and must be ignored. The proportion of correctly

repeated digits with and without distractor digits read in between targets was the measure included in the computation of this domain variable.

2.2.2 Executive function

The Wisconsin Card Sorting Test (WCST, PC version; Heaton et al., 1981) is a test of the cognitive processes necessary to form hypotheses and try them out when solving a task. The participant is asked to sort a series of cards with various shapes on them into the correct categories according to unknown matching rules set by the computer. Feedback is given after each response. After 10 consecutive correct responses, the computer changes the matching rule without notice. Responses in which the participant continues to use the previous matching rule, instead of realising that the rule has been changed and subsequently attempting to discover the new rule, are called perseverative responses. The number of the participants' perseverative responses were used here to construct the Executive function composite score in this study. This entails that the neuropsychological domain Executive function is here conceptualised as the participants' ability to continuously monitor their responses and subsequently change their strategy when receiving negative feedback. This would constitute a functional integration of all three of the subdomains of EFs discussed previously: inhibition, working memory, and cognitive flexibility (Diamond, 2013).

2.2.3 Motor coordination

Grooved Pegboard is a commonly used measure of fine motor speed and agility (Matthews & Klove, 1964; Skogan, Oerbeck, Christiansen, Lande, & Egeland, 2018). In this test, the participant is asked to place 25 small pegs into a pegboard with slots angled in different directions. The pegs must be placed from right to left with the left hand and left to right with the right hand, starting with the dominant hand. The mean time in seconds it took to complete the task for the dominant and nondominant hand combined was the measure used to compute the Motor coordination variable.

2.2.4 Selective attention

The Dichotic Listening Test (DL; Hugdahl & Andersson, 1986) is an auditory test of selective attention and brain laterality, during which the participant receives auditory stimuli in the form of six stop-consonants (*b, d, g, p, t,* and *k*) paired with the vowel *a* to form six basic

consonant-vowel syllables (CV-syllables): *ba, da, ga, pa, ta, ka*. The participant is asked to report the input being received in one ear at the time, which requires them to selectively attend to it and ignore information received by the other ear. The test is administered under three conditions, which differ regarding to which ear the stimulus is introduced: forced-right condition (FR), forced-left condition (FL) and the non-forced condition (NF). In this study, the mean number of correct right ear answers for the FR condition and left ear answers from the Forced left condition was used to construct the Selective attention composite score.

2.2.5 Verbal memory

In the California Verbal Learning Test (Delis et al., 1987) 16 words (List A) are read to the participant, who is subsequently asked to repeat back as many as they remember. This is repeated five times. Following one presentation and recall of an "interference" list (List B), the subject is immediately asked to freely recall the first list, and then given a cued recall opportunity. Twenty minutes later free and cued recall are tested again. Finally, the subject is presented a recognition trial where the words from List A are embedded in a 44-word list. The total amount of correct recollections in trial 1-5 was used to represent Verbal memory.

2.2.6 Visual memory

The Kimura Recurring Figure test (Kimura, 1963) presents the participant with 20 cards with geometric or nonsensical figures shapes. One hundred-forty cards are then presented individually for three seconds each, with the participant indicating whether each one was amongst the 20 cards originally presented. Only eight of the 20 original cards are among the 140 presented, and each of them is shown seven times. This gives a maximum possible score of 56, with 84 distractor cards. False positive responses award negative points to correct for guessing.

2.2.7 Visual perception

The Backwards Masking task (Green, Nuechterlein, & Mintz, 1994) was the test used to construct a variable to represent the domain originally called Visual attention. In the present study, however, this domain is called Visual perception to better reflect that the measures included here as described below are better thought of as measures of sensory-perceptual, pre-attentive processes (Green, Nuechterlein, & Breitmeyer, 1997; Saccuzzo, Cadenhead, &

Braff, 1996). In visual masking paradigms, masking occurs by presenting different stimuli in close temporal association to make them "compete" to be recognised explicitly in our conscious attention (Bachmann, Luiga, & Põder, 2005). The target stimulus is of an informational nature (e.g. a two-digit number) while the masking stimulus is non-informational (e.g. a pair of X's). Backwards masking occurs when the masking stimulus is presented after the target stimulus. The time interval between the offset of the target stimulus and onset of the masking stimulus is called the interstimulus interval (ISI) (Green, Nuechterlein, Breitmeyer, Tsuang, & Mintz, 2003), and typically vary from 0 to 100 milliseconds (ms). ISIs of less than 60 ms are thought to represent pre-attentional processes, with detection of the target stimulus occurring without conscious thought, and where longer ISIs reflect one's susceptibility to attentional disengagement (Green, Nuechterlein, Breitmeyer, & Mintz, 1999). In the present study, a traditional backward masking paradigm was used, originally developed by Sperling (1965). After being shown both the target and the masking stimulus, the respondents were asked to report the digits presented on the screen, even if they were not certain, making it a forced response. ISIs of 33 and 49 ms were chosen, with each stimulus being presented for 16 ms. The mean number of correctly identified digits in both the 33 and 49 ms ISIs were computed to create the Visual perception domain score.

2.2.8 Visuomotor processing

The neuropsychological domain of Visuomotor processing is here constructed by combining the Trail Making Test A and B (Reitan & Wolfson, 1985), measured as seconds to complete, with the Digit Symbol-Coding subtest from the WISC-R (Wechsler, 1974), measured as number of symbols correctly coded in 120 seconds. The Trail Making Test is a task where the participant must connect a series of circles arrayed on a sheet of paper by tracing a line between them, but in a specific order. In part A the participant must connect the circles in numeric order matching their designated numbers. In part B there are both circles with numbers and circles with letters, and the participant must connect them in an alternatingly numerical and alphabetical order (so A-1-B-2-C-3 etc.). The combination of visual scanning and motor ability in this test makes it apt to be part of the Visuomotor processing domain. The Digit Symbol-Coding subtest from the WISC-R is a test where participants must exchange a lengthy sequence of numbers ranging between 1-9 with a set of symbols, with one symbol corresponding to each number. The number-symbol exchange key is presented at the top of the page. The number of correct number-symbol exchanges the participant makes in the

allotted 120 seconds is recorded. The motor coordination necessary to fill in the symbols interacts with the visual scanning necessary to continually browse the symbol-number chart.

2.2.9 IQ estimate

IQ stands for the historic term of "Intelligence Quotient". At T1, the Wechsler Intelligence Scale for Children was one of the most generally accepted way to estimate the general cognitive ability (IQ) of children and adolescents under 16 years of age (WISC-R; Wechsler, 1974). The WISC-IV and, increasingly, the WISC-V are the currently in use editions of WISC (Wechsler, 2008, 2014). In the WISC-R a full-scale IQ (FSIQ) is computed from the scores on 10 subtests. A standard research and clinical practice is to use only certain subtests from the whole Wechsler test battery to estimate FSIQ due to the large scope of test (Sattler & Dumont, 2004). Short form versions aiming at measuring general intelligence usually include reasoning subtests of both a verbal and visual nature and exclude speed and attention subtests (Wechsler, 1999). Of the four WISC-R subtests conducted at T1, only the Similarities and Block Design subtests were used to compute an IQ estimate in the present study. The Similarities subtest measures abstract thinking, in the sense that the participant has to extract an overarching abstract concept uniting phenomena that differs on a concrete level. It is thus a measure of verbal intelligence. The Block Design subtest measures the participant's affinity for recognizing and re-organising visual patterns. The participant is asked to rearrange blocks with sides that are red, white, or both to match a presented picture, making it a measure of perceptual intelligence.

Two issues arise from using subtests to directly estimate IQ. Firstly, small deviations on the subtest level will amount to larger deviations when aggregated. For example, less than 16 percent of the population will achieve scores on the 16^{th} percentile on *two* tests. When computing general intelligence from two instead of 10 tests, this could artificially reduce variance somewhat. Secondly, as all tests with non-perfect reliability are affected by measurement errors, the true score will statistically regress towards the mean, and the degree to which this phenomenon occurs will depend on the subtests' reliability. To correct for these issues, the IQ estimate was not created by simply computing the mean of the two subtest scores, but by using Sattler and Dumont's table chart for estimating full-scale IQ from combinations of subtests (2004).

2.3 Symptom measures

While the Child Behavior Checklist (CBCL; Achenbach & Edelbrock, 1991) is technically a measure of problem behaviour, certain subscales of it is used here as ADHD symptom severity measures. This is not without precedence, as the MTA study used the Swanson, Nolan and Pelham (SNAP) rating scale as a baseline measure of symptom severity, and its content is reminiscent of these subscales of the CBCL, albeit more narrow (Roy et al., 2016; James M Swanson, 1981). The CBCL is part of the Achenbach System of Empirically Based Assessment (ASEBA), and its subscale structure was derived from Achenbach's (Achenbach, 1966) factor-analytical approach to structuring children's psychopathological symptoms. The CBCL assesses various behavioural and emotional problems that have taken place in the last 6 months in children from 4 to 18 years of age, with parents acting as informants. Each item is a general statement of possible behavioural problems and/or mental health symptoms, and the informant rates the degree to which the statement holds true for their own child. The scale of agreement ranges from 0 = "Not true" to 3 = "Very true or Often true". In total, the CBCL consists of 113 such items, which are divided into eight syndrome scale scores: Attention problems, Thought problems, Withdrawn, Somatic complaints, Anxious/depressed, Delinquent behaviour, Aggressive behaviour, and Social problems. The Somatic complaints and Anxious/depressed scales are combined to form the Internalizing subscale, and Delinquent behaviour and Aggressive behaviour scales form the Externalizing subscale. In this study, the participants' mothers were used as the informants, and the Attention problems and Externalizing subscales were selected for use as predictors due to their close association with ADHD symptoms, especially the Attention problems subscale (Lampert, Polanczyk, Tramontina, Mardini, & Rohde, 2004). The CBCL Total score was also included to account for the total load of potential problem behaviours. Previous findings have confirmed the CBCL as a valid screening instrument of ADHD, especially its Attention problems subscale (Hudziak, Copeland, Stanger, & Wadsworth, 2004), and its validity has been replicated in a Norwegian sample (Nøvik, 1999).

The Global Assessment Scale of symptoms (GAS) was also used to evaluate the symptoms of the ADHD group at T1 (Endicott, Spitzer, Fleiss, & Cohen, 1976). The treatment provider is asked to rate their patient's symptoms on a scale of 0 to 100, divided into 10 even intervals, with higher scores implicating stronger symptoms and dysfunction. The clinician treating each respective research participant with ADHD completed the GAS in the present study.

2.4 Outcome measures

2.4.1 Diagnostic status

Diagnostic reassessment was performed at T3 as at T2 (Øie et al., 2011). The ADHD diagnoses were determined by professor and clinical neuropsychologist Merete G. Øie using the Mini-International Neuropsychiatric Interview (MINI; Sheehan, Lecrubier, Janavs, Knapp, & Weiller, 1994) and information from patient case records. The diagnostic criteria used were those of the DSM-IV. The DSM-IV corresponds well with the DSM-III-R, implicating good diagnostic continuity between the two sets of diagnostic criteria used in this study at T1 and T3 (Biederman et al., 1997).

2.4.2 The Adult ADHD Self-Report Scale and Adult Self-Report form

To investigate ADHD psychopathology dimensionally, measures assessing ADHD symptoms as a continuous variable were used at follow-up. Two self-report forms were utilised, namely the Adult ADHD Self-Report Scale (ASRS) published by the World Health Organization (Kessler, Adler, Ames, et al., 2005) and the ADHD and Attention Subscales of the Adult Self-Report (ASR) from the ASEBA adult forms (Achenbach & Rescorla, 2003).

The ASRS was developed by Kessler and colleagues on behalf of the WHO to ensure that instruments screening for ADHD would be included in epidemiological surveys (Kessler, Adler, Ames, et al., 2005). It consists of 18 questions divided evenly between focusing on attention and hyperactivity/impulsivity symptoms, and this division construes two separate subscales. Each item is scored from 0 to 4, with higher scores indicating stronger symptoms. Empirical studies have indicated the ASRS test to have strong reliability and validity (Adler et al., 2006; Kessler et al., 2007). The ASRS was collected in the ADHD group only, which does not enable cross-group comparison with the HC group. While the ASRS does include an informant report which was collected by the research project, only self-report ratings were included in the present analyses.

The ASR from the ASEBA is not a symptom measure per se, but a measure of adaptive functioning. It is a self-report scale with items concerned with vocational, educational and social function. Its total score, ASR Adaptive Functioning, can be divided into subscales based on the underlying themes of items. The Attention problems and ADHD subscales are

used in the present study as ecological impairment-oriented measures of ADHD symptoms. Both the HC and ADHD groups were assessed with this instrument, which allows for cross-group comparison.

2.4.3 The Behavior Rating Inventory of Executive Function (BRIEF)

The BRIEF is a self-report assessment instrument developed specifically with ecological validity in mind, to focus on the everyday challenges faced by people with executive dysfunction (Gioia et al., 2000b). It was originally developed for use with children, with separate patient, parent, and teacher forms, but has later been expanded with an adult form, the BRIEF-A (Arch, 2005). Despite using the BRIEF-A, the acronym "BRIEF" will be used here for simplicity's sake. The inventory consists of 86 items comprised of statements of various challenges the participant may have faced the last 6 months that might pertain to executive dysfunction, such as having a messy living space, being forgetful, tardiness etc., which are answered on a three-point scale ("Never, Sometimes, Often"). These item scores make up eight subdomains of executive dysfunction, with higher scores indicating stronger dysfunction. The Inhibit, Shift, and Emotional Control subdomains together result in an additional composite Behavioral Regulation Index (BRI), while the subdomains Initiate, Working Memory, Plan/Organize, Organization of Materials, and Monitor together create the composite Metacognition Index (MI). The BRI and MI together construe the Global Executive Composite score (GEC), which is one of the three BRIEF measures used as outcome variables in this study. Additionally, the two subdomains Inhibit and Working Memory are also used separately as outcome variables, due to the evidence of their strong relationship with ADHD pathology (Isquith & Gioia, 2000), as mentioned above. The BRIEF's internal structure has been replicated and validated in a Norwegian study with a translated version (Fallmyr & Egeland, 2011), which was used in the present study.

2.5 Procedures

All participants received the same battery of neuropsychological tests in a fixed order at both T1 and T3: first the Wisconsin Card Sorting Test, then the Covert Visual Attention Test, followed by the Digit Span (WISC-R), Dichotic Listening Test, Digit Symbol (WISC-R), California Verbal Learning Test trials 1-5, Kimura Recurring Figures Test, Seashore Rhythm Test, Digit Span Distractibility Test, California Verbal Learning Test long delay recall, Trail

Making Test, Span of Apprehension Task, Similarities (WISC-R), Degraded Stimulus Continuous Performance Test, Block Design (WISC-R), Backwards Masking Test, Sustained Attention Test, and Grooved Pegboard. Note that this list includes more tests than what is utilised in the present study, but all are included in the list reported here to account for the total testing situation that the research participants went through. The total time of the tests amounted to between three to four hours, including breaks. Two breaks of 5-10 minutes and one break of 30 minutes were provided. The visual acuity of all participants was examined before testing at T1, allowing for the use of glasses if necessary. The neuropsychological tests were performed by the same individual at T1 and T3, Merete G. Øie, who was a PhD fellow at T1 and an experienced clinical neuropsychologist and professor at T3. As an exception to this, some of the healthy controls were tested by a test technician at T1 and by a clinical psychologist and PhD fellow at T3. Discontinuation of stimulant medication in the ADHD group was set to 24 and 15 hours before testing at T1 and T3, respectively. Psychological and diagnostic assessments came in addition to the neuropsychological test battery and were not performed in a standardised sequence at T1 and T3, affording some flexibility in the execution of the research data gathering. For instance, for a few participants at T3 it was more convenient to divide the total testing time into two days, in which case the neuropsychological test battery was prioritised to be performed in a standardised manner and sequence, while the rest could be flexibly adjusted. The testing intervals between T1 and T3 varied between 22 and 25 years, with an average follow-up time of 23 years.

2.6 Data analyses

All analyses were conducted using the statistical package SPSS, version 25.0. First, the ADHD group was divided into two groups, namely diagnosis retainers and remitters. Preliminary ANOVAs were performed to investigate between-group relationships between all three groups including the HC group. This would address the research question regarding the impairment levels reported by the persisters and remitters compared with the healthy controls at T3. Thus, the between-group comparisons needed to be performed for three groups. Due to the limited sample size, three pairwise ANOVAs were chosen as a method rather than a three-way ANOVA. Group differences between ADHD retainers and remitters at T1 were then further investigated as candidate predictors of the outcome measures by using linear regression analyses. No regression analyses included the HC group, as the goal of this study

was to predict in-group variation in only the ADHD sample at follow-up. When predictive effects between ADHD retainers and remitters were detected, hierarchical multiple regression analyses were performed. The largest variable was entered first, to see if the second one accounted for more variance. Due to the limited sample size, this was only done with up to two variables at a time. When testing for significance, the alpha-level threshold was set to 0.05, in accordance with conventional research practice. Predictions that reached a significance value between 0.1 and 0.05 are deliberated despite being statistically insignificant due to the limited sample size and increased risk of Type II errors.

2.7 Ethical considerations

All studies in this research project were approved by the Regional Committee for Medical Research Ethics in Eastern Norway (REK) and conducted in accordance with the Helsinki Declaration (General Assembly of the World Medical Association, 2014). Inclusion of the author into the research project was approved by REK before the work was initiated. The project was also approved by the Privacy Protection Ombudsman for research at the Innlandet Hospital Trust. Participants were assessed using established and standardized instruments, with no known risks associated with the examination. Ample breaks were given during testing, as needed. All participants received feedback on their results in any way that could benefit them. At T1, the ADHD group was given no monetary compensation, as they came to the NCAAP for treatment in any case. The HC group, on the other hand, had their travel expenses covered at T1. At T3, all participants were compensated for travel expenses and given NOK 500 for participating. At T1, signed consent forms were retrieved from parents and the partaking adolescent after being given a complete description of the study. The same procedure for the retrieval of informed, written consent was followed at T3, but this time only from the now-adult participants.

3 Results

All T1 and T3 variables included in the present study are included in Table 1. The ADHD sample is presented as two separate groups, as the predictive regression analyses will be based on the variation between them. Effect sizes are not included in the table because all scores except ASRS scores are given in either the z- or T-score format, which improves readability. Significant between-group differences are reported with “less than/more than”-symbols (<, >).

The ADHD remitters and persisters both had increased psychological impairment as measured by the CBCL at T1 compared to the healthy controls. However, the ADHD persisters had significantly higher T-scores than the ADHD remitters on the CBCL Attention problems and CBCL Externalizing subscales (10 and three T-score points, respectively). The ADHD remitters and persisters did not significantly differ on symptom severity at T1 measured by way of GAS ratings.

3.1 Outcomes of diagnosis, symptom severity, and executive dysfunction

The between-group differences as shown by the ANOVAs are displayed in Table 1. Eleven of the 19 participants with ADHD were re-diagnosed with ADHD after 23 years and eight were not. This gives a persistence rate of 58% with a corresponding 42% remission rate. ADHD remitters did not significantly differ from healthy controls on impairment caused by symptoms or executive dysfunction as measured by the ASR and BRIEF at T3. The ADHD remitters and persisters had significantly different scores on all outcome measures.

Table 1

Characteristics, predictors, and outcome measures of the ADHD and HC groups

	ADHD retainers	ADHD remitters	Healthy controls	Significant effects
Sex (male/female)	11/0	8/0	13/13	
Symptom ratings and BRIEF at T3:				
ASR ADHD	62.5 (10.8)	52.8 (3.3)	52.2 (5.1)	Ret>Rem & HC
ASR Attention	63.6 (9.0)	51.3 (2.9)	52.0 (4.5)	Ret>Rem & HC
ASRS Total	36.7 (9.3)	15.5 (7.8)	-	Ret>Rem
ASRS Attention	21.7 (4.8)	9.4 (4.8)	-	Ret>Rem
ASRS Hyperactivity	15.0 (6.0)	6.1 (3.8)	-	Ret>Rem
BRIEF GEC	61.6 (9.6)	40.1 (5.1)	42.8 (8.7)	Ret>Rem & HC
BRIEF Inhibit	58.5 (11.2)	43.3 (4.9)	44.5 (7.9)	Ret>Rem & HC
BRIEF Work. Mem.	67.1 (10.0)	47.0 (8.6)	44.2 (7.7)	Ret>Rem & HC
Predictor variables at T1:				
Auditory attention	-1.2 (1.1)	-0.7 (.9)	0.0 (0.7)	Ret & Rem<HC
Executive function	-0.5 (1.2)	-0.5 (1.1)	-0.1 (1.0)	No effect
Motor coordination	-1.4 (1.3)	0.7 (0.7)	0.1 (0.8)	Ret<Rem & HC
Selective attention	0.2 (1.0)	0.0 (1.0)	0.1 (0.9)	No effect
Verbal memory	-1.4 (1.1)	-0.9 (1.1)	0.0 (1.0)	Ret & Rem<HC
Visual memory	-0.7 (1.4)	-0.7 (2.0)	0.0 (1.0)	No effect
Visuomotor proc.	-1.2 (1.9)	-0.6 (0.8)	0.0 (0.8)	Ret<HC
Visual perception	-0.9 (0.6)	0.0 (0.9)	0.0 (0.9)	Ret<Rem & HC
FSIQ	105.5 (13.0)	107.1 (13.1)	115.9 (15.7)	No effect
GAS	51.9 (8.1)	50.9 (5.6)	-	No effect
CBCL Total	70.3 (4.6)	64.3 (6.5)	44.7 (8.5)	Ret>Rem>HC
CBCL Attention	71.3 (6.8)	60.9 (4.6)	51.8 (3.3)	Ret>Rem>HC
CBCL Externalizing	69.0 (8.8)	65.9 (4.5)	45.0 (7.7)	Ret & Rem>HC

Note: Neuropsychological domain composite scores given in z-scores based on the original 31 healthy controls. Due to five drop-outs the current HC group does not average 0 on the Executive function, Motor coordination, and Selective attention domain composite scores. CBCL and BRIEF scores given in the T-score format.

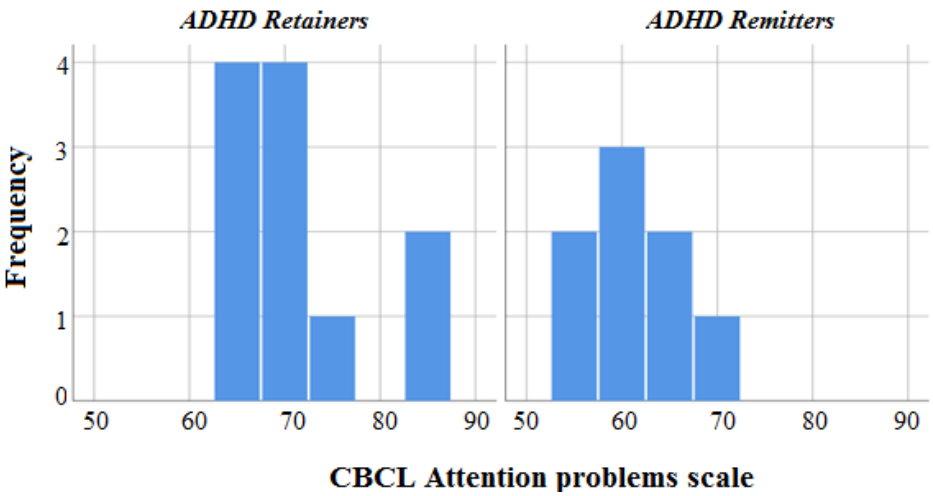
3.2 Predicting ADHD diagnostic persistence

Of the nine neuropsychological predictors, only Motor coordination and Visual perception significantly predicted diagnostic persistence at T3. Motor coordination explained 31% of the variance (Beta=0.559, F=7.730, p=0.013). Visual perception reached an explained variance value of 27% (Beta=0.523, F=6.417, p=0.021). A hierarchical multiple regression analysis showed that when Visual perception was added onto Motor coordination, they explained 44% of diagnostic variance, but the R^2 change failed to reach significance (R^2 change=12.5%, F change=3.56, p=0.078). When the order of the variables was reversed, the change reached significance (R^2 change=16.4%, F change=4.66, p=0.47).

Looking at the CBCL measures, the Attention subscale significantly predicted diagnostic persistence, at T3 with an explained variance of 45% (Beta=-0.672, F=13.976, p=0.002). The Externalizing subscale did not significantly predict diagnostic persistence, while the CBCL Total explained 25% of the variance (Beta=-0.500, F=5.670, p=0.029). When entered after the Attention problems subscale in a hierarchical multiple regression model, the Visual perception domain did not contribute with significant effects of its own. The Motor coordination domain significantly contributed with an additional 17% explained variance on top of the Attention problems subscale, ending up with a total explained variance of 62% (F change=7.37, p=0.015). The other symptom measure collected at T1, GAS, made no significant predictions on T3, on any outcome measure.

Figure 1

Baseline attention symptoms in the ADHD sample



Frequency table of CBCL T-scores, where higher T-scores indicate stronger symptom load.

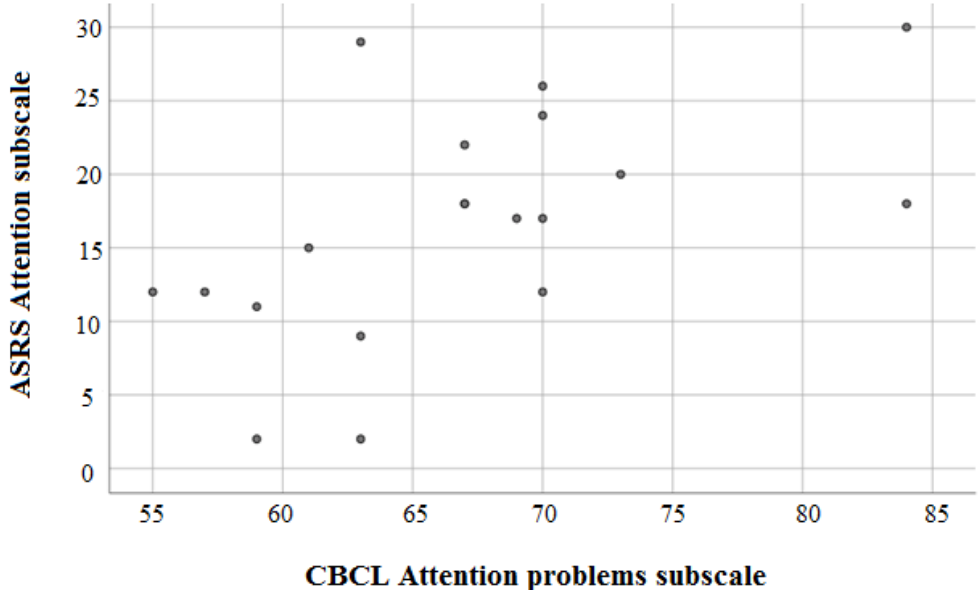
3.3 Predicting ADHD symptoms

No neuropsychological domain measures at T1 significantly predicted variance in the ASRS at T3, but the Motor coordination domain trended towards significantly explaining 17% of the variance in its hyperactivity subscale (Beta=-0.416, F=3.563, p=0.076).

The Attention subscale of the CBCL predicted 32% of the variance in the ASRS attention subscale (Beta=0.562, F=7.840, p=0.012). This effect was significant when looking at the ASRS total score as well, but as there were no significant predictions of the ASRS hyperactivity subscale, the explained variance was reduced to 25%, and the p-value increased to 0.029 (Beta=0.500, F=5.672). The CBCL Total score at T1 made no significant predictions on any ASRS outcome measure at T3. Thus, the CBCL Attention subscale at T3 predicts the ASRS at T3 when looking at both the attention subscale and the total ADHD composite, but the effect exclusively originates on the subscale-level of both the CBCL and the ASRS.

Figure 2

Attention symptoms at baseline and follow-up



ASRS score given in raw scores. CBCL subscale scores given in the T-score format.

No neuropsychological domain measures at T1 significantly predicted variance in the ASR at T3. The ASR subscale of attention deficits at T3 was predicted by the CBCL Attention problems subscale at T1, with an explained variance of 27% (Beta=0.524, F=6.428, p=0.021), while the CBCL Total score reached no significant predictions. No effects were found when

predicting the ADHD subscale of the ASR at T3, which has items targeting attentional problems mixed in with behavioural problems.

3.4 Predicting BRIEF scores

No neuropsychological domain measures at T1 significantly predicted any outcome variance in the BRIEF at T3, but Motor coordination came close to significantly predicting 18% of the variance in the GEC (Beta=-0.427, F=3.797, p=0.068).

The Attention subscale from the CBCL at T1 predicted 33% of the variance found in the GEC (Beta=0.573, F=8.314, p=0.01). While no significant results were found when attempting to predict the Inhibit subdomain from the BRIEF, the CBCL Attention problems subscale at T1 significantly predicted 41% of the variance in the Working memory subdomain on the BRIEF at T3 (Beta=0.638, F=11.684, p=0.003).

4 Discussion

This thesis has reported on a 23-year follow-up study on a clinical sample of adolescents with ADHD, examining the predictive effects of neuropsychological functioning and symptom severity on later diagnostic persistence, ADHD symptoms, and ecologically measured executive dysfunction. The roughly two-thirds of the ADHD sample who retained their diagnoses after 23 years exhibited larger deficits of motor coordination and visual perception in adolescence than their remitted counterparts, with stronger symptoms of attention as rated by their mothers. While attention deficits predicted almost all outcome measures, Motor coordination and Visual perception were the only neuropsychological functions that made significant predictions on any outcome measure. When analysed hierarchically, it was indicated that they explained separate sources of variance, and Motor coordination added explained variance to attention symptoms. The relevance of these findings to the research field and clinical practice is reviewed in the following section. Topics that are relevant to several of the research aims are deliberated continuously, creating differences in the space that is allotted to each aim.

4.1 Contemplating the present findings

4.1.1 Aim 1: Diagnosis, symptom severity, and executive dysfunction

Persistence rate

As described previously, the research field is seemingly unclear on what the actual persistence rate of ADHD is (Franke et al., 2018). This is likely caused by a combination of methodological issues regarding the operationalisation of remission, lengths of follow-up, and age spans studied, as well as to potential true variation in research samples. The present ADHD sample had a persistence rate of 58%. Of particular note is how this rate is seemingly in conflict with those of the Dunedin (Moffitt et al., 2015), Pelota (Caye, Rocha, et al., 2016), and E-Risk (Agnew-Blais et al., 2016) studies, which all showed a substantially lower persistence rate from childhood into adulthood than what had been previously suggested by research. One explanation may be provided by considering the inherent differences between birth cohorts and clinical samples (Caye, Swanson, et al., 2016). The present ADHD sample was recruited from an outpatient clinic where they were diagnosed and received treatment due

to their functional impairment. The prominent difference when studying a birth cohort is that you are assessing "everyone", and not exclusively treatment seeking individuals, which implies that the samples of the aforementioned studies may not have been as clinically impaired or symptomatically severe. Additionally, the present study is from Norway, a country where the diagnostic manual used nationally is the ICD and not the DSM. This implies that everyone in the present ADHD sample were also diagnosed with hyperkinetic disorder, since they received diagnoses and treatment in a Norwegian mental health clinic. As previously mentioned, several theorists argue that the difference between hyperkinetic disorder and ADHD may be one of severity (Asherson et al., 2016), and a reanalysis of the MTA study's sample showed that only 25% of them would have satisfied the more stringent criteria of hyperkinetic disorder (Santosh et al., 2005). Symptom severity has been repeatedly found to predict diagnostic persistence (Caye, Spadini, et al., 2016), including in the present study, providing a potential explanation for the higher persistent rate shown here. The current persistence rate is higher than those found in both a 33-year follow-up of individuals referred by teachers due to ADHD problem behaviour (Klein et al., 2012) and a meta-analysis of follow-up studies, although the studies included in the latter had very mixed interval lengths (Faraone et al., 2006). The persistence rate of 84% found in a large Dutch clinical sample is higher than that of the present study (van Lieshout et al., 2016), and this could possibly be attributed to their shorter follow-up interval of 6 years, a fourth of the length analysed here. Due to its important societal and individual implications, it is important that future research addresses these inconsistencies.

Symptom and executive dysfunction outcomes

Contrary to the evidence that individuals whose ADHD remits in adulthood have substantial subthreshold impairments (Asherson et al., 2016), this was not indicated to be the case for the present sample by either the ASR or BRIEF, and thus defied the expectation set by the stated hypothesis. The ASRS scores were substantially lower than those reported by the ADHD retainers, although some symptoms were reported. The transition from others- to self-ratings is a known methodological challenge in developmental research on ADHD, as individuals with ADHD have been shown to under-report symptoms compared to other informants who know them (Franke et al., 2018). This could have had an effect on the present findings.

That the retainers would report increased dysfunction on the BRIEF than remitters and healthy controls was supported, but not the expectation that remitters' scores would be higher

than the healthy controls. One general explanation of this could be that it stems from error related to the fact that the Norwegian BRIEF uses American norms. However, a psychometric validation of the BRIEF in Norway disproves this, as it showed that the scores of the Norwegian general population were within the 95% confidence intervals of the American norm (Fallmyr & Egeland, 2011). A more recent study on the adult BRIEF showed that Norwegian controls received 0.5-0.75 of a standard deviation lower scores than the American norm group (Løvstad et al., 2016). This means that even if the American norms do not match the Norwegian population, the current scores around 40 in the HC group indicate that they are representative of the Norwegian population in any case. However, it is important to avoid pre-emptively concluding that ADHD remitters experience no impairments. For one, some research has suggested that although ADHD symptoms disappear in adult remittent ADHD, the underlying cause of the disorder may be expressed in other areas of function, contributing to clinically significant functional impairments and adverse outcomes (Franke et al., 2018). In other words, individuals with remittent ADHD may have "outgrown their symptoms, but not their disorder". Additionally, reports from the present research project after the first follow-up after 13 years (T2) documented an elevated rate of adverse outcomes in academic, social, criminal, and marital functional areas (Øie et al., 2011). Functional outcomes at T3 should be given closer scrutiny before concluding that the individuals with remittent ADHD in this sample do not experience clinically significant impairment, although the present findings implicate that if they do, it is probably not caused by ADHD symptoms.

When it comes to the impairments reported by the ADHD retainers, it is interesting to compare the two functionally oriented measures, ARS and the BRIEF. While the ADHD retainers' ASR scores were one standard deviation higher than the remitters and controls, this difference was two standard deviations on the BRIEF. This could suggest that out of two functionally oriented measures, the measure specifically targeting functional impairment related to the EFs was more sensitive than the measure focused on symptoms, despite the group reporting strong symptoms on the ASRS.

4.1.2 Aim 2: Predicting diagnostic persistence

Neuropsychological predictors: positive findings imply the continued relevance of DAMP
It was surprising that the neuropsychological domains of Motor coordination and Visual perception predicted diagnostic persistence, and with a sizeable explained variance as well

(31% and 27%, respectively). When hierarchically including both in the same regression models, their total explained variance rose to 44%. When Visual perception was entered first into the model, the change in explained variance was significant, but was not the case when Motor coordination was entered first (p=0.078). This suggests that the possibility that their predictive effects stem from the same source of variance cannot be altogether disconfirmed in the present small sample. However, a combination of other findings in this study contends with this null hypothesis. Motor coordination still reached significance when included in a regression model with the CBCL Attention problems subscale, to a total explained variance of 62%. When Visual perception was added to the CBCL Attention problems subscale, its added explained variance did not approach significance. Additionally, Motor coordination approached significance as a predictor of the ASRS hyperactivity subscale. The combination of these findings can be interpreted as indication that Motor coordination and Visual perception distinctly correspond to the two separate symptom factors known to exist in ADHD (Asherson et al., 2016). That Visual perception did not approach significance when added onto the Attention problems subscale indicates that its predictive effects are already included in the symptom scale. But their respective places chronologically in the sequence of behaviour is important to notice: the variance shown in Visual perception occurs in the span of milliseconds, while behavioural measures will by nature be an expression of the entire neural sequence of in- and outgoing information. In other words, this lower-order, pre-attentional sensory-perception function may be responsible for a segment of the expressed behavioural attention symptoms shown on the Attention problems subscale. This is in alignment with the findings mentioned previously that indicated that lower-order perception functions may create cumulative effects in higher-order systems (Lenz et al., 2010; Ríos et al., 2004). As reviewed in the introduction section of this text, impairments in motor control and visual perception have been reported previously in the literature. However, it is to the author's knowledge that this study is the first time that the considerable predictive power of such deficits on long-term diagnostic persistence is documented.

These findings call into relevance the concept of DAMP – deficits of motor and perception (Gillberg, 1983). It is defined as the co-occurrence of ADHD with developmental coordination disorder (DCD), in the absence of a severe intellectual disability (IQ<50) or cerebral palsy. Despite its focus on motor skills and development, the coding instructions for the DCD allow for the supplementary coding of sensory deficits such as seen in neurological soft signs (American Psychiatric Association, 2000; Gillberg, 2003b). DAMP is associated

with substantial long-term impairments, possibly to a larger extent than ADHD without motor and perception deficits (Gillberg, 2003a). Systematic literature reviews show that approximately half or more of all individuals with ADHD show impairments in motor control and motor skills, probably satisfying the conditions for concurrent DCD (Damme, Simons, Sabbe, & van West, 2015; Kaiser, Schoemaker, Albaret, & Geuze, 2015). These estimates have also been known to the research field for a long time (Martinussen et al., 2005). It is confounding that such strong prevalence rates have not been given more attention in the theoretical literature at large on ADHD or on predicting its long-term outcomes, much less in the diagnostic criteria themselves. One reason for this discrepancy may be that motor difficulties in ADHD are often interpreted as expressions of hyperactivity-impulsivity rather than "true" motor difficulties in large parts of the literature (Egeland, Ueland, & Johansen, 2012). For an illustrative example of this trend, see Brown (2008). Egeland et al. (2012) stipulated that there may be different causes of such motor difficulties depending on subtypes, with the combined subtype's being caused by hyperactivity-impulsivity (i.e., higher-order regulation deficits) and the inattentive subtype's being caused by genuine motor difficulties (i.e., lower-order motor deficits). This would not coincide completely with the present findings: although the ADHD persisters, who had apparent substantial motor skills impairment, exhibited strong symptoms of inattention, they also had high Externalizing symptoms.

These findings carry implications for future research. The potential predictive relationships of these DAMP-like symptoms with diagnostic persistence would make strong candidates for therapeutic and preventive interventions. One example of the development of such interventions are the promising results of physical therapy on the motor impairments exhibited by children with concurrent ADHD and DCD (Watemberg, Waiserberg, Zuk, & Lerman-Sagie, 2007). The theoretical implications of this research would also need to be examined further. For instance, Halperin and Schulz' (2006) model on ADHD stipulates that it is caused by subcortical remittance from ADHD will occur in line with the development of the EFs. The present findings could be seen as partial support of this, at least regarding the prominent role of subcortical dysfunction. However, estimations of the improvement of neuropsychological functions are not possible due to a lack of T3 measurements. Although, an interpretation in line with the model could be formulated, namely that the current ADHD persisters had a stronger subcortical dysfunction to compensate for to begin with, and thus their EF development was not up to the task, resulting in diagnostic persistence and executive

dysfunction. This could also align with hypotheses made by behavioral neuroenergetics theory (Killeen et al., 2013), that stronger subcortical dysfunction uses up the neural reserve, evidencing stronger executive dysfunction.

Neuropsychological predictors: negative findings

Most neuropsychological predictors included in this study made no significant predictions. Negative findings are important in clinical research (Easterbrook, Gopalan, Berlin, & Matthews, 1991), but in a small sample such as this, the lack of statistical power creates an increased likelihood of Type II error (Banerjee, Chitnis, Jadhav, Bhawalkar, & Chaudhury, 2009). This lends reduced credibility to the negative findings.

If one were to interpret the negative findings of the majority of neuropsychological domains studied here as legitimate, certain domains would warrant commentary. For instance, both the domains of visuomotor function and selective attention are associated with EFs such as working memory and inhibition (Diamond, 2013), and their lack of predictive potential here could signify that EFs are not predictive of persistence, unlike the lower-order functions discussed previously. Contrary to this, some studies have found WM deficits to be predictive of persistence after 6 and 13 years (Sjöwall et al., 2017; van Lieshout et al., 2017). Another of the negative findings that would be of note would be the lack of any predictive potential in IQ, despite previous findings in certain studies (Cheung et al., 2015; Gao et al., 2015), but in support of others (Roy et al., 2016). However, while such negative findings would have contributed meaningfully to the research literature in their own right, they are not very robust or trustworthy in this small sample.

Symptom predictors

While the findings among the neuropsychological predictor variables were perhaps the most surprising, the most consistent and strong predictions across almost all outcome variables in this study were made by the Attention problems subscale of the CBCL. These predictions included diagnostic persistence (R^2=45%, p=0.00), the ASR attention subscale (R^2=27%, p=0.02), the ASRS attention subscale (R^2=32%, p=0.01), and the BRIEF General Executive Composite (GEC; R^2=33%, p=0.01) and WM subdomain (R^2=41%, p=0.00). One perspective would be that it is hardly surprising that ratings of attention deficits in adolescence predicts the outcome measures included here, as both the ADHD diagnostic criteria, symptom severity measures, and the BRIEF includes content similar to the CBCL Attention subscale. In other words, the present findings have merely shown that attention deficit ratings in adolescence

predict attention deficit ratings in adulthood. As past behaviour is known as a possible predictor of future behaviour (Harris, Lee, Thompson, & Kranton, 2016), these would not be very noteworthy findings, but only reflect trait stability. The author would contend with such a perspective, as this nonetheless serves as a useful replication of the predictive power of symptom measures in adolescence, even if it was in accordance with the existent literature. Additionally, that the effect is so strong is a robust finding in itself, where close to half the diagnostic variance at T3 is being explained by a single maternal rating measure in adolescence 23 years earlier. The literature is also clear on the fact that there resides considerable complexity in the multifactorial causes of behaviour, insofar as trait stability may not always be expected over longer periods of time (Montano & Kasprzyk, 2015).

Another relevant finding is the fact that in all instances of the CBCL Total score predicting an outcome measure, the Attention problems subscale's predictions were stronger and thus more significant. This is a partly contrary finding to the general principle of the increased predictive power of aggregation in psychology (Rushton, Brainerd, & Pressley, 1983), and shows specificity in the predictive value of specifically attention symptoms. This is also interesting since the current clinical sample was not selected to have no comorbidities – indeed, half the ADHD sample had either a diagnosed developmental reading disorder, oppositional defiant disorder, or both of these commonly comorbid disorders in ADHD. Given the premises that 1) the CBCL Total detects a broader range of symptoms of mental illness than just the Attention problems subscale, and 2) that comorbidity predicts persistence of ADHD pathology (Caye, Spadini, et al., 2016), one could have expected the CBCL Total to make stronger predictions than just a single subscale.

Another notable finding is the lack of any significant predictions made by the Externalizing subscale. Before making any speculative interpretations of this, it must be mentioned that it is very possible that this subscale was simply not a sensitive stand-in measure of hyperactive-impulsive symptoms of ADHD. It is constructed by combining the Delinquent behaviour and Aggressive behaviour subscales, which would probably correlate to some extent with hyperactivity-impulsivity, but not completely. The rest of this paragraph will discuss various interpretations one could consider to the extent that the Externalizing subscale is sensitive to hyperactivity-impulsivity. First of all, the differences in predictive power between the Attention problems and Externalizing subscales highlights the importance of evaluating these two dimension separately, since aggregating them might obfuscate their specific contributions

(Gao et al., 2015). Also, the present results from the Externalizing subscale is seemingly in conflict with several previous studies that have found that both inattentive and hyperactive-impulsive symptom ratings possess predictive power on persistence (Biederman et al., 2011; Gao et al., 2015). There are several possible interpretations of this conflicting finding. First of all, the finding might be "true" in its own right, that there are no differences in the childhood levels of externalizing behaviour in children with remittent or persistent ADHD, at least not when investigating a longer timespan than previous studies. This would indicate that despite their disruptive qualities exasperating teachers and parents alike, externalizing behaviours are not what clinicians should look to when attempting to make prognostic evaluations of the diagnostic outcomes of their clients, but attention symptoms. It is also possible that the negative finding is a Type II error, either due to unsystematic error, i.e. coincidence, or because of a lack of statistical power to yield significant effects when the group differences are small. Contrary to the large difference between ADHD retainers and persisters on the Attention problems subscale at baseline, which constitutes a standard deviation, there is only a 3 point difference on the Externalizing subscale. If this trend were replicated in a larger sample, it could yield a significant effect, even if the variance explained would be small.

4.1.3 Aim 3: Predicting symptom severity

Neuropsychological predictors

While no hypothesis regarding potential predictions from the neuropsychological variables was stated. No neuropsychological domain measures predicted ADHD symptoms in either the ASR or the ASRS, although Motor coordination's prediction on the ASRS hyperactivity subscale trended towards significance (R^2=17%, p=0.08). Several of the points raised above on the lack of prediction of neuropsychological measures on diagnostic outcome apply here as well. Additionally, the inconsistency regarding the predictive potentials of Motor coordination and Visual perception is worth addressing. When it comes to the ASR, the difference between ADHD retainers and remitters was relatively small, only one standard deviation. This was due to low scores amongst the retainers, and not high scores among remitters, as compared to the healthy controls. This could be in accordance with reported underestimations of self-reported symptoms by adults with ADHD (Franke et al., 2018). The ASRS scores showed larger between-group differences, and Motor coordination almost significantly predicted hyperactivity symptoms in adulthood. In any case, the diagnostic assessment by an experienced clinical neuropsychologist is, by definition, more conceptually valid and sensitive

than a screening instrument. Although the negative findings shown here cast some doubt on the significant findings on diagnostic persistence, one finding could be said to "trump" the other.

Symptom severity predictors

The stated hypothesis was that symptom severity in adolescence would predict symptom severity in adulthood. This was largely confirmed by the findings. That the subscale level was more predictive than the aggregate level was discussed above and so was the lack of predictive effects of the Externalizing subscale.

It is also an intriguing, but easily overlooked, finding that the GAS at baseline predicted no outcome measure at T3. When inspecting the scores of the ADHD group when divided into diagnosis retainers and remitters, it is clear that this lack of a predictive effect stems from a complete lack of a between-group difference. This is in turn an interesting finding on its own when considering the clear group distinction in maternal ratings. So, while the mothers of the adolescents with an eventual persistent ADHD course rated their children to have attentional difficulties around a T-score of 70, mothers of eventual remitters from the disorder rated their sons' inattention to range around a T-score of 60. This discrepancy constitutes a whole standard deviation, while the evaluations of clinicians who treated the adolescents were that there were no apparent differences between the groups at that time. It is possible that clinicians put greater stock in hyperactive-impulsive symptoms when evaluating the GAS, as mothers reported reduced between-group differences on the Externalizing subscale. In any case, this negative finding of the predictive potential of the GAS, as well as the strong predictive validity of the mothers' ratings, brings a clear message to clinicians of the importance of the parental perspective. This does not entail that the GAS or similar clinician rating measures are without worth, but that their long-term predictive value is limited, at least in the present data. One reason for this discrepancy in predictive accuracy may be the difference in the amount of information each respective informant has regarding the patient. It could have been useful to include teacher ratings of the adolescent with ADHD, as evidence shows that teacher and parent ratings correlate only weakly to moderately, but each contribute meaningful clinical information (Narad et al., 2015). Despite the fact that teachers report more externalizing symptoms than parents (Tripp, Schaughency, & Clarke, 2006), diagnoses based on either parents or teachers are equally accurate (Bied, Biederman, & Faraone, 2017). These research indicate that including teacher reports in the present study could have yielded useful,

complementary information, and perhaps more variability in reports of externalizing behaviour.

4.1.4 Aim 4: Predicting executive dysfunction

Neuropsychological predictors

No neuropsychological measures significantly predicted BRIEF scores. However, that Motor coordination came close to significance with a p=0.068 and an explained variance of 18% in the GEC is a substantial finding in itself, despite being on the trend level. That the p-value is above the predetermined alpha threshold of 0.05 means that the null hypothesis cannot be discarded, i.e. that it cannot be ruled out that this effect was an artefact occurring by chance. If one were to interpret it as a genuine finding, however, it would be highly noteworthy that the Grooved Pegboard test in adolescence were to predict almost a fifth of the variation in executive dysfunction as expressed in the complexity of everyday life 23 years later. An intriguing statistical implication would also follow; since Motor coordination's predictions of the Inhibit and Working memory (WM) subdomains did not approach significance, the almost-significant effect on the GEC likely comes from significant predictions of other subdomains. It would be interesting to find out which of the subdomains are predicted by Motor coordination to the extent that its prediction of the aggregate of nine subdomains is almost significant. When it comes to the negative findings in the rest of the neuropsychological predictors, including tests of EFs such as the WCST, TMT, or Dichotic Listening, this was expected due to the evidence that shows limited overlap between behavioural ratings of executive dysfunction and neuropsychological test measures (Fallmyr & Egeland, 2011; Toplak et al., 2013). As Toplak and colleagues' review of the relationship between ratings scales and test measures of EFs posits (2013), the "extremely weak" (r=0.15) concurrent correlations seen between ratings scales and test measures of EFs may be due to them tapping different "cognitive levels". In other words, that ratings scales tap into EFs as a broader concept than the narrower focus of standardised test measures. This could arguably be the case, or even more so, when the relationships examined span across 23 years.

Symptom predictors

The predictions of the Attention subscale on the BRIEF were discussed earlier. Shortly mentioned, the expectations stated in response to the research aim was met. That no predictions were made by the Attention problems CBCL subscale on the Inhibit subdomain is

intriguing, due to the close association between selective attention and cognitive inhibition (Diamond, 2013). Moreover, this subscale predicted 41% of the variance in the WM subdomain, which is surprising given the "unity" of the EFs described by the unity-diversity model (Miyake & Friedman, 2012). One the other hand, this contrast lends support to the dissociation (or "diversity") of the two EF domains.

4.2 Strengths and limitations

While there are clear and significant limitations to the present study which necessitate caution when interpreting its findings, there are some remarkable strengths as well. Its three main strengths are its inclusion of a comprehensive neuropsychological test battery, its long time frame of 23 years from adolescence into middle adulthood, and its focus on dimensional and ecologically valid outcome measures concurrently with diagnostic persistence. All of the neuropsychological test measures and symptoms rating measures included in the study are psychometrically validated and frequently in use in clinical practice, such as the GAS, the CBCL, the BRIEF, and the ASRS. Another strength of the study is its high retention rate, with only one of the original 20 participants in the ADHD sample not included at T3, and that was due to death.

4.2.1 Statistical power

The most severe limitation of this study is its small sample size, making it clearly underpowered and vulnerable to Type II errors, i.e. falsely accepting the null hypothesis. It is thus quite possible that some of the null findings presented here are reflections of this lack statistical power more so than actual lack of significant relationships. One instance where this could be the case is the lack of a group difference found between the ADHD and HC groups on the IQ estimate from T1. This difference trended towards replicating the dissimilarity of around half a standard deviation that is reported in previous publications (Frazier et al., 2004). Such a risk of Type II errors could possibly have been mitigated by allowing for a higher alpha threshold value than 0.05. However, this would have raised the probability of false positives, which was unwanted due to the large amount of analyses being performed. With an alpha threshold level of 0.05 in such a small sample, one can be more confident in its findings (although the actual sizes of the effects could still vary according to chance). The lack of statistical power in this study also directly relates to its reduced capacity to withstand

statistical analyses that could clarify relationships of a more complex nature than what linear regression is capable of – for instance including more than two predictive variables in one regression model, as the analyses of the MTA did (Roy et al., 2016). This would have been in line with arguments that since ADHD severity in childhood is the strongest known predictor of outcome, predictive variables are only useful to the degree that they add to the variance explained above symptom severity (Sjöwall et al., 2017). On the other hand, more complex analyses could have reduced the clinical applicability and utility of the conclusions made due to their abstract nature. One could say that a linear regression analysis more closely reflects the prognostic evaluations that professionals are asked to make in their clinical practice.

4.2.2 Measures and participants included in the study

The question of which measures to include in the study always carries implications as to its strengths and limitations. Although a broad array of neuropsychological functions was included, there are other functions that are regarded as important in ADHD today, such as delay aversion, reward-related functions, emotional dysregulation or reaction time variability. All outcome measures in this study were self-report in one way or another, as the clinician making the diagnostic reassessments at T3 talked only to the participants. Evidence shows that including ratings from other informants such as parents, partners, friends, or clinicians may increase accuracy (Asherson, 2016). For instance, it could be the case that under- or overestimation of impairment by the ADHD respondents led to inflated or deflated remission rates in the current study. This could be a source of both systematic and random error. As such, including teacher reports at baseline, or ratings from family, partners, or friends at follow-up could have been beneficial. On the other hand, using exclusively self-report outcome measures could also be seen as a corresponding strength in this study, in the sense that it has a clear client-oriented focus based on the participants' own subjective experience. To carry such a perspective further, it would have been interesting to include baseline self-report measures from the research participants as well. Another research strength regarding the measures included is the fact that mostly all of them are tests and rating scales that are frequently used in clinical practice. Choosing to include the BRIEF rather than EF test measures significantly contributes to the applicability of the present findings to clinical practice.

As this is a study reporting on a clinical, all-male, Norwegian sample of ADHD, there are obvious limitations to the generalisability of its findings, for instance to ADHD in the general population, other cultures and/or ethnicities, and gender. While this may reduce somewhat the study's ability to shed light on the core questions of the research field on ADHD as a general phenomenon, the fact that it is a clinical sample allows for the assumption that the sample is reasonably representative of what Norwegian clinicians encounter in outpatient clinics. When considering gender and methodological limitations, one might argue that the HC group being evenly divided between the genders when controlling for an all-male ADHD sample constitutes a limitation. However, it is the author's view that this is a research strength contributing to the external validity of the study, as it more accurately reflects the societal context that the all-male ADHD sample is situated in; their achievements and challenges are relative to the whole population, not just other men. An additional strength of the study is the small age variability in the ADHD sample, with a standard deviation of only 1.4 years. This reduces the likelihood that developmental effects are obfuscated by a too wide age group.

4.3 Clinical implications

The findings of the present study are clinically relevant in several ways. First of all, it shows that when ADHD persists into adulthood, it is associated with considerable impairment both when it comes to symptoms and executive dysfunction. The ASR, ASRS, and BRIEF scores of the ADHD persisters were all significantly higher than those of the ADHD remitters and healthy controls. This is important knowledge to all clinicians and researchers working with ADHD. Second, neuropsychological assessment is often employed in the process of diagnosing ADHD, and often includes the Grooved Pegboard test. Ninety-one percent of Norwegian clinical neuropsychologists already include this test in their standard testing procedure (Egeland et al., 2016), and the current findings adds value to their interpretations made when working with individuals with ADHD. This could also inform their treatment decisions. Third, knowledge of the strong predictive validity of maternal ratings of symptoms is useful to all clinicians, especially when complimented by humility that, at least in the present study, clinicians' evaluations measured by the GAS made no significant predictions on outcomes. Fourth, prognostic evaluations are a central part of clinical practice. Knowing what to expect from the future is important to both the adolescent patient with ADHD (Honkasilta, Vehmas, & Vehkakoski, 2016) and their parents (Craig et al., 2016). The

knowledge of the multitude of adverse outcomes associated with ADHD, and the persistence findings shown in this clinical sample, also highlights the need for therapeutic interventions over time in the lifespan with regular follow-ups. A challenge here is also the transition from childhood and youth services to adult services, which is reputedly often challenging to conduct smoothly (Franke et al., 2018). These issues arise in the same time period that individuals with ADHD face increased risk of outcomes with more severe consequences than in childhood and early adolescence, such as traffic accidents and substance abuse (Dalsgaard, Østergaard, Leckman, Mortensen, & Pedersen, 2015; Øie et al., 2011). This calls for the detailed planning and implementation of long-term treatment plans, and such tasks may be informed by research such as the present study.

Additionally, while the national public policy for admittance criteria into adult specialist mental health services state that ADHD can be constitute a right to assessment and treatment (Helsedirektoratet, 2015), anecdotal clinical experience indicates that this is not standard practice in many institutions, which often face considerable budgetary constraints when it comes to their time (Friberg, 2015). If that were the case, it would be truly unfortunate when there are documented interventions readily available for such a high-cost disorder (Doshi et al., 2012). Though only speculation, this could be due to lack of knowledge about the limitations of long-term ADHD medical treatment or confidence in psychological interventions aimed at ameliorating symptoms or dysfunction.

4.4 Future research

Several potential research questions could have been included in this study but were excluded due to consideration of size constraints. Three of the most central considerations for future research would be to expand the sample size, the baseline measures included, and the outcome measures included. Firstly, if future research projects were to quite simply replicate the current research finding, but with a larger sample size, it would be a strong contribution to discount the (albeit unlikely) possibility that the present findings are simply an artefact of a small sample. Secondly, despite having collected baseline data on family history of mental illness, including ADHD, and comorbidity, it was beyond the scope of the current study to include this data in the analysis. Doing so would strengthen the research findings, as evaluating all candidate predictors with empirical support could potentiate clearer delineation of which effects stem from where. Third, as there is ambiguity regarding the adult outcomes

of childhood ADHD, expanding the current study to also include other outcome measures could enhance the conclusions. This could include functional outcomes, such as marital and occupational status, as some reports indicate that ADHD remitters outgrow their diagnoses, but not their functional impairments (Franke et al., 2018). Another outcome worth closer scrutiny is emotional dysregulation, an area of dysfunction that is being increasingly investigated as a potential central impairment in ADHD (Shaw, Stringaris, Nigg, & Leibenluft, 2014; Surman et al., 2015). Such functional outcomes were recorded at T3 but were outside of the scope of the present study and will be included in future reports on the project.

Methodologically speaking, this study only used simple linear regression analysis to evaluate the predictive relationship from baseline to follow-up, and did not, as van Lieshout and colleagues request (2013), include neuropsychological test measures at follow-up to evaluate how the *course* of neuropsychological functioning predicts the *course* of ADHD pathology. Doing so would drastically improve the study's ability to discuss the developmental trajectories of ADHD, and potentially its underlying mechanisms and etiology.

As mentioned previously, the present findings carry implications for research on ADHD therapy as well. For instance, while interventions targeting EFs may be somewhat effective (Adler et al., 2014; Diamond, 2013), it is unclear whether ADHD patients with DAMP-related dysfunction would benefit from interventions oriented towards the EFs. On one hand, it could be useful to expand the cognitive reserve available for compensation of such subcortical deficits (Halperin, Trampush, Miller, Marks, & Newcorn, 2008), on the other it could possibly be more useful to address the motor impairments directly (Watemberg et al., 2007). Additionally, it is of consequence to the development of therapies that the present sample's ADHD retainers report the strongest impairments on executive dysfunction measures (the BRIEF), and not symptom impairment (the ASR). This could inform the focus of therapeutic interventions. When it comes to psychopharmacological interventions, future research should investigate more closely how their effects on ADHD symptoms relate to motor- and perception symptoms, and whether this could mediate a mechanism of change on likelihood of diagnostic persistence. Due to the heterogeneity of ADHD, and that the current findings implicate that individuals with DAMP-like symptoms are a subgroup of the disorder with a higher likelihood of a persistent course, investigating these matters on samples consisting only of individuals with DAMP could be worth consideration.

4.5 Concluding remarks

The aims of this study were to investigate the long-term clinical outcomes from adolescence into adulthood in a clinical sample of ADHD, and the predictive relationships of neuropsychological functions and symptom severity on said outcomes. In alignment with expectations, symptom measures in adolescence strongly predicted both diagnostic persistence, symptom severity, and executive dysfunction in adulthood, but this was the case only for attention symptoms, and not externalizing symptoms. More surprising was the finding that, out of nine neuropsychological domains constructed from a comprehensive assessment, Motor coordination and Visual perception were significant predictors of diagnostic persistence. This suggests that traits closely related to DAMP, i.e. the co-occurrence of ADHD with motor skill and perception deficits, may be a fruitful area to search for predictors of persistence. The present study thus confirms the superiority of clinical rating measures of impairments over neuropsychological test measures in predicting ADHD persistence, but also introduces renewed relevance to certain lower-order neuropsychological functions. Additionally, the present study found that in a longitudinal clinical sample, nearly two thirds of individuals with ADHD had a persistent diagnosis in middle adulthood, challenging recent controversial findings of much lower persistence estimates in longitudinal birth cohort studies. While limited by its small number of participants, the present study makes significant contributions to the research field on current questions regarding the nature, outcomes, and prediction of ADHD.

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