

Predictors of Criminality and Substance
Use in Adolescents with ADHD:
A 23-Year Follow-up Study

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Abstract

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Title: Predictors of Criminality and Substance Use in Adolescents with ADHD: A 23-Year Follow-up Study

Supervisor I: Merete Glenne Øie; **Supervisor II:** Jens Egeland

Background: The literature shows a strong link between the diagnosis of Attention-Deficit/Hyperactivity Disorder (ADHD) and criminality and substance use. However, less is known about the potential predictors of criminality and substance use within the ADHD population. The first aim of this study is to investigate the longitudinal criminality and substance use outcomes in adolescents with ADHD followed over 23 years. The second aim is to investigate potential predictive effects of neuropsychological impairment, ADHD symptoms and global symptom load, co-occurring externalizing and internalizing problems, and comorbid Oppositional Defiant Disorder (ODD) in adolescence on criminality and substance use after 23 years follow-up.

Methods: Forty-five individuals between 12-18 years of age, 19 males with ADHD and 26 healthy controls (M= 13, F=13), were investigated 13 and 23 years after an initial baseline study. At baseline they underwent comprehensive neuropsychological testing. Emotional and behavioral problems were measured with the Attention problems, Externalizing, Internalizing, and Total Scale of the Child Behavior Checklist/4-18 (CBCL). Symptom load was measured with the Global Assessment Scale of Symptoms (GAS), and comorbid ODD was diagnosed by a clinician at baseline. The participants in the ADHD group were asked about criminal convictions at 13- and 23-year follow-ups. Substance use was measured in both groups using the Alcohol Use Disorder Identification Test (AUDIT) and the Drug Use Disorder Identification Test (DUDIT) in the 23-years follow-up. Simple linear regression analyses, Analyses of variance, Chi square analyses of independence and Spearman's rank-order correlations were used to investigate the longitudinal associations between the baseline and the outcome measures.

Results: A substantial proportion of adolescents with ADHD (47%) had committed crimes by the 23-years follow-up. Two baseline measures predicted criminality in the ADHD group: overall symptom load measured with the GAS, and externalizing problems measured with the CBCL Externalizing scale. Neuropsychological functioning and comorbid ODD in the adolescents with ADHD did not predict later criminality. There were no statistically

significant differences between the ADHD group and the healthy control group regarding substance use after 23 years. However, numerical differences similar to those reported in other studies were found, with the ADHD group had higher scores on all substance measures than the healthy control group. None of our baseline measures predicted substance use through the course of the 23-year study.

Conclusion: A substantial amount of adolescents with ADHD were convicted of crimes and reported problematic substance use in adulthood. In individuals with ADHD, overall symptom load, and externalizing problems in adolescence, predicted criminality in adulthood. These findings indicate that it is possible to predict the risk of criminality in adulthood for adolescents with ADHD. The findings of this study highlight the need for long-term treatment planning from adolescence and into adulthood, especially in cases with high symptom load and externalizing problems.

Preface

This project was based on data from the research project, “Clinical, Neurocognitive and Functional Outcome in Early-Onset Schizophrenia and Attention-Deficit/Hyperactivity Disorder: A 23 Year Follow-up Study”. We are grateful to our main supervisor Merete Glenne Øie for the opportunity to participate in such an interesting project, and for believing in us from the moment we came to her with our hypotheses. She has been an important sparring partner, and we have benefited greatly from both her wealth of knowledge and her quick responses to our questions. Our co-supervisor Jens Egeland has given us valuable advice when it comes to methodology, while supporting our exploration into the world of statistics. He has also asked important questions and given helpful input to text.

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Mathilde Solberg Jensen & Karethe Hustad Torgersen

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1 Introduction

1.1 Background

The associations between Attention-Deficit/Hyperactivity Disorder (ADHD), criminality and substance use disorders (SUD) are well documented (Erskine et al., 2016; American Psychological Association, 2013). Several studies find higher incidence of ADHD in prison populations than in society as a whole (Knecht, de Alvaro, Martinez-Raga, & Balanza-Martinez, 2015). Longitudinal and cohort studies show that ADHD populations have a higher conviction rate than people without this diagnosis (Moffitt et al., 2015; Fletcher & Wolfe, 2009; Rasmussen & Gillberg, 2000). The same is evident for alcohol consumption (Rasmussen & Gillberg, 2000) and drug use (Franke et al., 2018), as well as more severe cases where the individuals meet the diagnostic criteria for SUDs (Erskine et al., 2016). ADHD in childhood is associated with early initiation of substance use, more frequent substance use, and SUDs (Groenman, Janssen, & Oosterlaan, 2017; Molina et al., 2018). There is also a higher prevalence of ADHD in patients in treatment for substance problems than in the general population (van Emmerik-van Oortmerssen et al., 2012), with the prevalence of ADHD increasing with the severity of the addiction (Lugoboni et al., 2017). However, few longitudinal studies have explored the variation within the population of those with ADHD.

Recently, there has been an increased interest in understanding the nature of ADHD in adults and the trajectories from childhood to adulthood with a focus on functional outcomes in individuals with ADHD (Caye et al., 2016c). Knowledge about predictors of negative outcomes within the ADHD group will be vital in the work to develop, implement and improve targeted preventative measures. This would be beneficial for the individuals affected, their next of kin, and society as a whole.

Some researchers have found that in ADHD populations, neuropsychological impairments, ADHD symptoms, and co-occurring Conduct Disorder (CD)/Oppositional Defiant Disorder (ODD) predicted later substance use and crime (Molina & Pelham, 2014, and Knecht et al., 2015; Mohr-Jensen & Steinhausen, 2016). However, few studies have included neuropsychological impairments, ADHD symptoms, as well as more general emotional and behavioral problems, and comorbid Oppositional Defiant Disorder in the same

study. Furthermore, even among longitudinal studies, the follow-up period is usually no longer than 10 years, which means that some development may be missed. In the following section, we will present literature on ADHD, as well as the main associated neuropsychological impairments, ADHD symptoms, internalizing and externalizing problems, and comorbidity. We will then review research on the predictive value of such measures in adolescence on later criminality and substance use outcomes in later life.

1.2 ADHD

1.2.1 Diagnosis

ADHD is a persistent and impairing neurodevelopmental disorder characterized by symptoms of inattention, hyperactivity and impulsive behavior (Fitzgerald, Bellgrove, & Gill, 2007). According to the fifth edition of the American Psychiatric Association's Diagnostic and statistical manual of mental disorders (DSM-V), ADHD begins in childhood, and the symptoms of the disorder have to appear before the age of 12 (American Psychiatric Association, 2013). The symptoms must be maladaptive and inconsistent with the child's developmental level, be present in at least two settings (Goldman et al., 1998), and be associated with substantial functional impairment (American Psychiatric Association, 2013). ADHD is one of the most common psychiatric disorders in childhood (Froehlich et al., 2007). However, ADHD is no longer solely viewed as a childhood disorder, as in many instances it continues into adulthood (Goldman, 1998; American Psychiatric Association, 2013).

The equivalent to ADHD in the International Classification of Diseases, 10th Revision (ICD-10) is Hyperkinetic Disorder (HKD) (World Health Organization, 1992). All three symptoms of impaired attention, hyperactivity and impulsiveness need to be present to fulfill the diagnosis, and the symptoms need to be evident in more than one situation, making the diagnosis narrower than the equivalent ADHD in DSM-V (Taylor et al, 2004). A re-analysis of an American study of treatment efficacy found that only a quarter of the children diagnosed with ADHD qualified for the HKD diagnosis (Santosh et al, 2005). This can complicate comparisons between studies from countries using different diagnostic manuals in healthcare. However, other research has found no difference when comparing the ADHD and HKD diagnoses (Remschmidt, 2005).

1.2.2 Epidemiology

Most studies find that ADHD has a prevalence of around 5% in children and adolescents worldwide (Polanczyk, Willcutt, Salum, Kieling & Rohde, 2014; Faraone, Sergeant, Gillberg & Biederman, 2003). A meta-analysis of prevalence studies, which only included studies investigating the prevalence of three or more diagnoses, found an ADHD prevalence of only 3.45%, which is significantly lower than the more typical estimate of 5% (Polanczyk, Salum, Sugaya, Caye, & Rohde, 2015). This could be due to the substantial overlap in symptoms between neurodevelopmental disorders, which complicates differential diagnostics (Thapar, Cooper & Rutter, 2017) and leads to an overestimation of prevalence in studies that only examine ADHD. Another study also found evidence indicative of overdiagnosis, this time due to heuristics and gender bias (Bruchmüller, Margraf & Schneider, 2012). Clinicians diagnosed ADHD even when lacking information about required criteria, and vignettes with a boy received diagnoses twice as often as the same vignette with a girl. Some studies find that the prevalence is higher in higher-income countries and claim that more high-quality research is needed to know the true prevalence of ADHD worldwide (Damiano & Forssberg, 2019). In Norway the prevalence of HKD is 3% in children aged 6-17 (4.3% for boys and 1.7% for girls) (Ørstadvik et al., 2016).

While ADHD was long thought to be a childhood disorder only, this has been disproved, as studies have shown that 2.5% of adults worldwide fulfill the diagnostic requirements (Fayyad et al., 2017; Simon, Czobor, Bálint, Mészáros, & Bitter, 2009). Recent findings from three longitudinal studies in New Zealand, Brazil and the United Kingdom indicate that adult ADHD may be a separate clinical entity from childhood ADHD (Moffitt et al., 2015; Caye et al., 2016a; Agnew-Blais et al., 2016).

In children and adolescents, the disorder affects more males than females, with a rate of approximately 2.4-1 (Polanczyk et al., 2007). This gender difference is not found in adulthood, but this can possibly be due to referral bias (Bruchmüller, Margraf, & Schneider, 2012). This can also be explained by the finding that males have a much higher increase in dopamine receptor density in puberty than females, a difference which is eliminated by adulthood due to heavier pruning (Andersen & Teicher, 2000).

1.2.3 Etiology

Despite extensive research on ADHD over the past decades, the exact causes of the functional problems associated with ADHD are still not known (Thapar, Cooper, Eyre & Langley, 2013). Converging evidence suggests that the disorder is complex and heterogeneous, with both genetic and non-genetic factors playing a role in its etiology, as well as an interplay between the two (Tarver, Daley, & Sayal, 2014; Thapar et al., 2013; Rutter, Moffitt, & Caspi, 2006). This multifactorial explanation of the etiology of ADHD corresponds to the heterogeneity of the disorder, including the psychiatric comorbidity, range of neuropsychological impairment, and structural and functional brain abnormalities associated with it (Faraone et al., 2015).

Family, twin, and adoption studies yield a heritability estimate of around 70–80% in both children and adults, making ADHD one of the most heritable psychiatric disorders (Faraone et al., 2005). Genetic analyses find both common and rare genetic variants with small associations with ADHD (Faraone & Larsson, 2018). Genome-wide association studies have implicated the role of dopamine, noradrenaline and serotonin neurotransmitter systems in ADHD (Faraone et al., 2015), which aligns with the dopamine deficit theory of ADHD (Levy, 1991).

A large number of twin and family studies have found genetic overlaps between ADHD and ODD, CD, antisocial behavior, and substance use problems, as well as internalizing disorders such as anxiety and depression (Faraone & Larsson, 2018). There is also evidence of shared genetic risk factors for behavioral inhibition/impulsivity, which is a core component of externalizing disorders such as ADHD and ODD/CD (Young et al., 2009).

Findings from neuroimaging studies propose that ADHD is a disorder of early brain development (Nagel et al., 2011; Batty et al., 2010; Gray, Korczyk, Andrews, & Bélanger, 2018). The areas implicated are involved in networks associated with neuropsychological functions, such as attention and executive functions (Cortese, 2012; Moreno-Alcázar et al., 2016). A developmental twin study found that genes not only contribute to the onset of ADHD, but also to the persistence and remittance of the disorder (Chang et al., 2013).

While there is a strong genetic component, non-genetic neurological factors have also been implicated in the etiology of ADHD (Gray et al, 2018; Tarver et al, 2014). Examples are factors known to affect brain development, such as exposure to alcohol and tobacco during pregnancy, low birth weight, hypoxic-anoxic brain injury, epilepsy and traumatic brain injury. Socio-environmental variables such as parenting strategies and family adversity can also

affect symptom severity, extent and type of impairment, and presence of co-occurring problems (Villodas, Pfiffner & McBurnett, 2012; Counts, Nigg, Stawicki, Rappley, & Von Eye 2005).

1.3 Variation within ADHD

ADHD is a heterogeneous disorder with a wide spectrum of symptom presentation and severity (Gray et al., 2018; Franke et al., 2018). The disorder has generally been conceptualized as a discrete entity with clear diagnostic thresholds, but recent genetic, neurobiological, and neuropsychological research indicates that it is better viewed as the extreme end of normal variation (McLennan, 2016). Wåhlstedt, Thorell and Bohlin (2009) argue that this heterogeneity is empirically evident in three different respects: in neuropsychological impairments, in the ADHD-specific symptom domains of hyperactivity/impulsivity and inattention, and in comorbid behavioral problems. We will use this framework, but will also include other psychiatric symptoms that are common in ADHD.

1.3.1 Neuropsychological impairment

ADHD is characterized by deficits in multiple neuropsychological domains (Coghill et al., 2018), which have been found to be associated with ADHD symptoms (Thaler, Bello & Etcoff, 2013; Wåhlstedt et al., 2009). The core neuropsychological feature of the disorder is deficits in executive functions, which are high-level cognitive processes that control lower-level processes in order to facilitate goal-directed behavior (Friedman & Miyake, 2017). Executive functions are moderated primarily by the frontal lobe, particularly the Prefrontal Cortex (Miyake & Friedman, 2012), which is central in inhibitory control and working memory, behavioral regulation, temporal processing, reinforcement processing, and delay aversion (Rutter, Kim-Cohen, & Maughan, 2006; van Lieshout, Luman, Buitelaar, Rommelse, & Oosterlaan, 2013; Willcutt et al., 2010). Deficits in executive functions inhibit the control and organization of behavior, manifesting in forgetfulness and difficulty in planning and coordinating everyday tasks (Tarver et al., 2014; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005).

One of the most comprehensive models of ADHD, historically, is Russell Barkley's theory of self-regulation. It emphasizes the impairments in the prefrontal lobes and executive

inhibitory processes in individuals with ADHD (Barkley, 1997). In line with this theory, there have been findings of inhibitory deficits in about 60-70% of individuals with ADHD (Nigg, 2005). More recently, a framework of executive functions, the unity/diversity framework, posits that executive functions can be divided into three domains (Miyake & Friedman, 2012). These include the modifying of content in the working memory by attending to relevant information and the suppression of irrelevant information; shifting/switching between tasks; and the inhibition of dominant responses.

Several domains other than executive function have also been found to be impaired in ADHD (Faraone et al., 2015), highlighting its neuropsychological heterogeneity and the importance of including comprehensive neuropsychological test batteries in research. These domains include reward processing, decision making, speech and language, memory span, processing speed and response time variability, arousal and activation, and motor control (Coghill 2018; Sonuga-Barke, Taylor, Sembi, & Smith, 1992; Luman, Oosterlaan, & Sergeant, 2008; Faraone et al., 2015). To account for the heterogeneity of neuropsychological impairment, recent models of ADHD have been proposed with a number of pathways that may affect children with ADHD differently (Sonuga-Barke, Bitsakou, & Thompson, 2010).

Meta-analyses have found significantly lower Full Scale IQ in children (Frazier, Demaree & Youngstrom, 2004) and adults (Bridgett & Walker, 2006) with ADHD than in the general population. However, most of individuals with ADHD have a normal IQ score, but the test scores can be affected by impaired working memory and processing speed (Barkley, 2014), as well as disruptive behavior during testing (McConaughy, Ivanova, Antshel & Eiraldi, 2009). The same is evident in other neuropsychological domains, as some individuals with ADHD show few or no neuropsychological impairments, and few individuals show deficits in all domains (Coghill, Seth, & Matthews, 2014; Wåhlstedt et al., 2009). It is possible that this is due to the poor ecological validity of test situations, as most neuropsychological tests are construct-driven rather than based on everyday functioning (Burgess, 2006). For this reason behavior ratings such as the Behavior Rating Inventory of Executive Function (BRIEF) are commonly used to assess executive impairments in everyday life situations (Gioia, Isquith, Guy, & Kenworthy, 2000).

As it is evident in the literature that neuropsychological deficits are prominent features in the expression of ADHD, longitudinal studies on the impact of neuropsychological impairments on future outcomes are necessary in order to discern then the consequences of such impairments.

1.3.2 Symptoms

The DSM-V describes three presentations of the disorder; predominantly inattentive, predominantly hyperactive–impulsive, and combined (American Psychological Association, 2013). A review and meta-analysis found overwhelming support for the concurrent, predictive and discriminant validity of the subtypes (Willcutt et al., 2012). However, presentations can change over time (Nigg, Tannock, & Rohde, 2010), and individuals with the same presentation vary greatly in symptom profiles, as individuals with one presentation often have sub-threshold symptoms of a different presentation (Faraone et al., 2015). While this means there is little support for classifying the presentations as distinct forms of ADHD, information about the presentation is of clinical relevance (Willcutt et al., 2012) and could be important to furthering our understanding of the intra-group variation of negative outcomes associated with ADHD.

As the presentation of symptoms in ADHD is so variable, it is important to include this variation in research investigating outcomes in this population. Moreover, it may be useful to study the continuum of the different symptoms of ADHD as predictors of future outcomes.

1.3.3 Comorbidity

ADHD comorbidity is complex, and varies in relation to the different manifestations of the disorder and developmental stages (Weissenberger et al., 2017). Recent reports replicate earlier findings that about 2/3 of children with ADHD in the US have a co-occurring psychological disorder (Danielson, Ghandour, Holbrook, Kogan & Blumberg, 2018; Cantwell, 1996). Different subgroups of ADHD as delineated by co-occurring disorders might have different risk factors, clinical courses, neurobiology and pharmacological responses. Therefore, investigating the potential long-term effect these factors have on future outcomes is important (Biederman, Newcorn & Sprich, 1991), as it may provide insights on ways to improve preventative measures by tailoring interventions to the subgroups different needs.

As a discussion of all possible comorbid disorders is too extensive for this study, there will be a focus on the most frequent co-occurring disorders. About half of the children with ADHD also fulfill the diagnostic criteria for ODD or CD, making it the most common comorbid disorder (Danielson et al., 2018). ODD and CD are defined by disruptive and antisocial behaviors that violate the rights of others or social norms (American Psychiatric

Association, 2013). The European clinical guidelines for HKD conclude that CD should often be regarded not as a comorbid disorder, but rather as a complication of a concurrent HKD (Taylor et al., 2004), and the ICD-10 has a subtype of HKD with comorbid CD. This comorbidity is more common in boys than girls and has been suggested to cause a referral bias responsible for part of the difference in the prevalence of ADHD between the genders, as the increased symptom load in boys, which is partly caused by this comorbidity, increases the chance of referral (Biederman et al., 2002).

Children with both ADHD and ODD have more severe symptoms, more impairment, poorer social skills, more comorbidity, and academic underachievement across age ranges than children with only ADHD (Connor, Steeber & McBurnett, 2010; Jensen et al., 2001; Biederman et al., 1996; Carlson, Tamm, & Gaub, 1997). Other prevalent comorbid conditions in children with ADHD are internalizing disorders such as anxiety and depression (Danielson et al., 2018). This is associated with poor treatment response (Al Ghriwati et al., 2017) and poor functioning, including poor academic achievement (Armstrong, Lycett, Hiscock, Care, & Sciberras, 2015; Cuffe et al., 2015). Furthermore, concurrent comorbidities of both internalizing and externalizing nature have been evidenced to occur in up to 22% of all childhood cases of ADHD (Abikoff et al., 2002). Some researchers have found evidence indicating that ADHD with ODD/CD, ADHD with anxiety disorders, and ADHD co-occurring with both ODD/CD and anxiety disorders could constitute separate clinical entities (Jensen et al., 2001).

The findings of such high comorbidity rates highlights the importance of taking such comorbidity into account in research concerning outcomes in ADHD.

1.4 Treatment

While there is no known cure for ADHD, pharmacological (mainly stimulant medications; methylphenidate and amphetamines), psychological and psychosocial treatment methods, as well as a combination of the two, have reported to be efficacious for ADHD symptoms (Faraone et al., 2015; Jensen et al., 2007). Stimulant medications used to treat ADHD target the dopamine system by increasing the levels of dopaminergic neurotransmitters (Faraone et al., 2015). In Norway, a combined treatment approach is recommended (Norwegian directorate of health, 2017). A report from 2004 showed that 77% of children in treatment for ADHD in Norway received medication, with less focus on psychological interventions

(Andersson, Ådnanes, & Hatling, 2004), and the number of adolescents under 19 who used ADHD medication doubled between 2004 and 2017 (The Norwegian Prescription Database, 2019). This preference for pharmacological interventions could be due to early studies reporting superior effect of medication over psychological and psychosocial treatment (Vogt & Lunde, 2018; Hinshaw & Arnold, 2015). However, more recent findings from the multimodal treatment study of ADHD (the MTA-study, Hinshaw & Arnold, 2015) indicated that medication does not have a long-term effect on symptoms (Swanson et al., 2017). Moreover, the findings regarding the effect of cognitive training (Cortese et al., 2015) and medications on neuropsychological functions are conflicting (Coghill et al., 2014; Uchida, Spencer, Faraone, & Biederman, 2018; Maruta, Spielman, Tseretopoulos, Hezghia, & Ghajar, 2014). Some studies in Norway show that clinicians often do not make adequate differential diagnostic assessments or diagnose comorbid disorders (Andersson et al., 2004; Surén et al., 2018), which can result in a lack of treatment for these comorbid disorders.

Inadequate treatment may have negative consequences in relation to the long-term outcomes of these disorders (Vogt & Lunde, 2018), including substance use and criminality in adulthood. Moreover, the evidence regarding treatment's effect on reducing the risk of substance use (Mannuzza, Klein & Moulton, 2003; Molina et al., 2013; Humphreys, Eng & Lee, 2013) and criminality are conflicting and inconclusive (von Polier, Vloet & Herpertz-Dahlmann, 2012).

1.5 Outcome

1.5.1 ADHD across the lifespan

ADHD in childhood has been shown to significantly increase the risk of adverse outcomes later in life, such as comorbid psychiatric and substance related disorders in adulthood, the most common of which being SUDs and antisocial personality disorder (Copeland, Shanahan, Costello, & Angold, 2009; Biederman et al., 2010; Barbaresi, et al., 2013). ADHD also increases the risk of impaired academic and occupational performance, dysfunctional relationships (Barkley, 2016; Fitzgerald et al., 2007), poor physical health (Khalife et al., 2014), and even reduced life expectancy and quality of life (Erskine et al., 2013; Dalsgaard, Østergaard, Leckman, Mortensen, & Pedersen, 2015).

Several studies have found that in about 50% of cases, ADHD persists into adulthood (Hechtman et al., 2016; Lara et al., 2009), with varying trajectories across individuals (Barkley, 2016). However, a meta-analysis found that only 15% of 25 year olds still had the diagnosis, while 50% of the individuals partly remitted (Faraone, Biederman & Mick, 2006). Symptoms of hyperactivity and impulsivity generally decline with age (Van Lieshout et al., 2013), while symptoms of inattention appear to be relatively stable with increasing age (Hart, Lahey, Loeber, Applegate, & Frick, 1995; Millstein, Wilens, Biederman, & Spencer, 1997). A recent review concluded that available evidence is largely inconsistent with regard to the predictors of persistence versus remittance (Franke et al., 2018). Individuals with persistent ADHD have been shown to have worse outcomes compared to individuals who only had the diagnosis in childhood (Hechtman et al., 2016; Agnew-Blais et al., 2018). However, many individuals with remitted ADHD still have symptoms and functional impairments, and thus have substantial needs in adulthood (Thapar et al., 2017; Huntley & Young, 2012; Faraone et al., 2000).

In summary, having ADHD in childhood is a risk factor for a range of adverse outcomes, and this risk seems to be increased when the disorder persists into adulthood. Even though interest in the research field has increased in the past decades, there is still a lack of knowledge regarding the lifespan aspects of ADHD, and more longitudinal studies are needed (Franke et al., 2018). In the current study, the focus will be on the longitudinal outcomes regarding criminality and substance use.

1.5.2 ADHD and criminality – intergroup variation

Studies have revealed significantly higher rates of ADHD among adolescent and adult criminal offenders than in the general population, with rates ranging from 24-67% (Knecht et al., 2015; Gudjonsson, Sigurdsson, Young, Newton, & Peersen, 2009). Likewise, studies following children with ADHD to adolescence and adulthood have shown higher rates of arrests than in the general population (Dalsgaard, Brøbech, Frydenberg, & Thomsen, 2013; Rasmussen & Gillberg, 2000). The prospective longitudinal study by Dalsgaard et al. (2013) found that 47% of the ADHD group had criminal convictions by adulthood, and were therefore five times more likely to have committed a crime than individuals without ADHD. A recent meta-analysis and systematic review of longitudinal studies found that ADHD in childhood was significantly associated with adolescent and adulthood arrests, convictions and

incarcerations (Mohr-Jensen & Steinhausen, 2016). Moreover, individuals with ADHD had an earlier onset of antisocial behavior and an increased risk of criminal recidivism than individuals without ADHD. Philipp-Wiegmann et al. (2018) conducted a follow-up of former juvenile prisoners 15 years after their first conviction and found that youth with ADHD had a higher rate of recidivism and re-offended 2.5 times faster than youth without ADHD. Contrary to these findings, one study failed to detect a relationship between ADHD and criminal offending (Kolla et al., 2018). However, this study was cross-sectional and used self-report of ADHD symptoms as opposed to a clinical interview, which may have affected the results.

There have been fewer studies that focus on females with ADHD than on males with ADHD, and the findings are conflicting. Some studies have reported no gender differences (Dalsgaard et al., 2013; Knecht et al., 2015), while others find that men have higher rates of criminal offences, as well as gender-specific predictors of criminality (Vingilis et al., 2015; Kolla et al., 2018).

In summary, the majority of available research suggests a strong link between ADHD both in childhood and adulthood, and criminal behavior.

1.5.3 ADHD and criminality – intragroup variation

1.5.3.1 Neuropsychological impairments

Longitudinal studies have shown that low IQ in children with ADHD predicts later criminality (Mohr-Jensen & Steinhausen, 2016). Furthermore, abnormalities in the prefrontal cortex associated with impaired executive functions (e.g. impaired behavioral inhibition/impulsivity) could explain why individuals with ADHD sometimes exhibit antisocial behavior (Knecht et al., 2015; Mannuzza, Klein & Moulton, 2008). In addition, the influential General Theory of crime proposes that offending is a function of opportunity and impulsivity (Hanoch, Gummerum, & Rolison, 2012). In line with these hypotheses, meta-analytical reviews of cross-sectional studies have found robust associations between executive function deficits and antisocial behavior in general, and increased impairments in executive functions in inmates with ADHD (Morgan & Lilienfeld, 2000; Ogilvie, Stewart, Chan, & Shum, 2011; Ginsberg, Hirvikoski, & Lindfors, 2010). However, whether executive deficits in children and adolescents with ADHD are predictive of later criminality is, to our

knowledge, largely unexplored. However, the cross-sectional research described makes it plausible that neuropsychological impairments in adolescents with ADHD could predict later criminal outcomes. This further emphasizes the need for more longitudinal studies that investigate these associations.

1.5.3.2 Symptoms

The symptoms of ADHD, such as impulsivity and hyperactivity, may be directly associated with criminality (Erskine et al., 2016; Knecht et al., 2015). Cross-sectional evidence has found that hyperactive/impulsive and combined subtypes have a significantly higher risk of crime than the predominantly inattentive subtype (Cahill et al., 2012). A meta-analysis by Mohr-Jensen & Steinhausen (2016) of related longitudinal studies found that the severity of ADHD predicted later criminal behavior. Barkley, Fischer, Smallish, & Fletcher (2004) found that hyperactive children had a higher rate of antisocial behavior and arrests at 21 years of age than the community control group. This study found that the hyperactive group primarily differed from the control group on drug-related offences, and that this group difference was related to the severity of ADHD in childhood, adolescence, and adulthood. However, more studies on the longitudinal prediction of the risk of criminality in adulthood based on ADHD symptoms are needed.

1.5.3.3 Comorbidity

Having ADHD in childhood has been shown to increase the risk for developing both antisocial personality disorder and SUDs in adolescence, which increases the risk for criminal behavior in adolescence and adulthood (Mannuzza et al., 2008; Gudjonsson, Sigurdsson, Sigfusdottir, & Young, 2012). Some researchers however, have found that substance use is not associated with criminality after controlling for comorbid antisocial personality disorder (Einarsson, Sigurdsson, Gudjonsson, Newton, & Bragason, 2009).

Other comorbid diagnoses that have been extensively studied are ODD and CD. A substantial amount of evidence indicates that conduct problems in childhood and adolescence are strong predictors of later antisocial and criminal behavior among individuals with ADHD (Mordre, Groholt, Kjelsberg, Sandstad & Myhre, 2011; Mohr-Jensen & Steinhausen, 2016). For instance, Pardini & Fite (2010) found that having ADHD in childhood predicted CD and ODD over time, and that CD and ODD robustly predicted antisocial and criminal behavior.

Some longitudinal studies have found that compared to controls, children with ADHD have an increased risk of evidencing antisocial personality traits and criminal behavior in adulthood, even without a co-occurring CD or ODD in childhood (Rasmussen & Gillberg, 2000; Mannuzza, Klein, Abikoff, & Moulton, 2004). Contrary to these findings, studies have reported that ADHD has no unique effect on criminality without the presence of conduct problems (e.g. Satterfield et al., 2007; Mordre et al., 2011). ODD has been associated with an earlier age of initiation into delinquent behavior, a greater variety of offending and higher prevalence of severe delinquency (Sibley et al., 2011). However, the association is less comprehensive than that found with CD, which has stricter diagnostic criteria than ODD (Pardini & Fite, 2010). One longitudinal study found individuals with ADHD and CD had significantly higher rates of youth delinquency, while no significant difference was found between individuals with ADHD and ODD, and ADHD only. All the ADHD groups had increased risk for criminality compared to healthy controls (Sibley et al, 2011). These findings highlight the importance of diagnostic specificity when studying crime in ADHD. In summary, comorbid conditions such as SUDs, CD, ODD and antisocial personality disorder have been found to be related to criminality in individuals with ADHD.

1.5.4 ADHD and substance use – intergroup variation

Alcohol and drug use is associated with a range of negative consequences for the individual and society (Sales et al., 2018). Substance use disorders are defined as abuse of or dependence on alcohol, illicit drugs or nicotine (American Psychiatric Association, 2013). In line with DSM-V's move to a continuum-based conceptualization of alcohol and drug use problems (American Psychiatric Association, 2013), this study will focus on all substance use, and will not be limited to those with a substance use disorder. This is because substance use in itself causes risk of negative consequences, such as injury during intoxication, and/or developing problematic use over time (Halkjelsvik & Storvoll, 2015). Negative consequences of substance use, such as elevated risk of criminality and neuropsychological impairment, may be further exacerbated combined with a diagnosis of ADHD (Weafer, Fillmore, & Milich, 2009; Compton, Volkow, & Lopez, 2017; Gustavson et al., 2017).

The available literature indicates that children with ADHD have an increased risk of earlier and increased use of tobacco, alcohol and illicit substances in adolescence and adulthood (Franke et al., 2018; Charach, Yeung, Climans, & Lillie, 2011). ADHD is prevalent

among adults seeking treatment for substance use disorders, with prevalence estimated to be 23% (van Emmerik-van Oortmerssen et al., 2012; van Emmerik-van Oortmerssen, Crunelle, & Carpentier, 2013). Furthermore, the combination of SUDs and ADHD increases the risk of other comorbid disorders such as mood, anxiety, and borderline personality disorder, as well as worse treatment outcomes (Lugoboni et al, 2017; van Emmerik-van Oortmerssen et al., 2014).

Some researchers have found that the elevated risk of SUDs among individuals with ADHD is robust to demographic and methodological differences that vary across studies (Lee, Humphreys, Flory, Liu, & Glass, 2011; Groenman et al., 2017). However, others have found that the risk of some substance use outcomes is inconsistent when considering the onset, escalation, and course into adulthood (Molina et al., 2018). Finally, some researchers have found that females with ADHD have an increased risk of substance use (Dalsgaard, Mortensen, Frydenberg, & Thomsen, 2014), while others have found the opposite (Babinski et al., 2011; Hinshaw, 2018). In conclusion, the developmental trajectories from ADHD to substance use are complex (Luo & Levin, 2017).

Studies on ADHD and substance use have been criticized for not providing data on the developmental paths of substance use. They have also been criticized for focusing on substance use diagnoses rather than developmentally sensitive continuous variables such as frequency of substance use (Molina et al., 2013). In the current study, the aim is to provide more research about the developmental risks of substance use.

1.5.5 ADHD and substance use – intragroup variation

1.5.4.1 Neuropsychological impairments

The self-medication hypothesis posits that individuals with ADHD use substances to try to compensate for the core symptoms and impairments associated with the disorder (Szobot & Bukstein, 2008; Young & Sedgwick, 2015). The hypothesis also suggests that individuals with ADHD use stimulant drugs for their short-term attention enhancing effects (Young & Sedgwick, 2015).

The impairment of executive functions such as inhibitory control (i.e. impulsivity) is a critical element in several theories of addiction (Kalivas & Volkow, 2005; Dom, Hulstijn, & Sabbe, 2006). Additionally, poor behavioral inhibition may lead to school difficulties

(Biederman et al., 2004), which in turn increase the risk of substance use problems in adulthood (Moffitt et al., 2011). Poor executive function has been cross-sectionally associated with substance use and SUDs across multiple substances in adults (Gustavson et al., 2017) and adolescents (Iacono, Malone & McGue, 2008). This relationship may partially be explained by the deteriorating effect that substance use has on executive function (Gustavson et al., 2017). However, some research have shown that poorer executive function appears before substance use in individuals with ADHD, which indicate that executive deficits may be a predictor of substance use. Nigg et al. (2006) found that response inhibition in adolescence were predictive of the number of illicit drugs used and comorbid alcohol and drug use, independent of IQ, ADHD, conduct symptoms and age. Likewise, according to Moeller & Dougherty (2002), children and adolescents who later develop substance abuse, show increased impulsivity. Together these findings indicate that poor executive function could be a risk factor for substance use, that substance use and executive function share genetic liability, and/or that substance use affects executive functions negatively. These three explanations are not mutually exclusive (Gustavson et al., 2017).

Some longitudinal studies have failed to link executive deficits in individuals with ADHD to later SUDs (Wilens et al., 2011; Groenman et al., 2015). In addition, a cross-sectional study of adolescents with and without ADHD symptoms, Handley et al. (2011) found that reactive disinhibition (i.e. sensation seeking) was associated with adolescent substance use, whereas executive disinhibition (i.e. impulsivity) was not a unique predictor. The results indicated a reward-driven pathway, rather than an executive dysfunction pathway to adolescents' substance use (Handley et al., 2011), which is consistent with findings from other studies (e.g. Castellanos-Ryan, Rubia, & Conrod, 2011).

In summary, several neuropsychological functions related to ADHD have been linked both cross-sectionally and longitudinally to substance use. However, the findings are somewhat conflicted, and few studies have investigated the neuropsychological functions as predictors of substance use. In the current study, we will investigate the longitudinal association between neuropsychological functions in adolescence and substance use in adulthood.

1.5.4.2 Symptoms

As explained in the previous section, the self-medication hypothesis proposes that substance use may function as self-medication to alleviate the core symptoms of inattention and

hyperactivity/impulsivity. For instance, the use of sedative drugs may attenuate restlessness or hyperactivity (Young & Sedgwick, 2015). Patients with ADHD report an improvement in their ADHD-specific symptoms when using alcohol, cannabis, and cocaine for self-medication purposes (Ohlmeier et al., 2008; Volkow et al., 2003).

Associations have been found between symptoms of inattention in childhood (Molina & Pelham, 2003) and adulthood (Ohlmeier et al., 2008) and substance use in adolescence and adulthood respectively. This could be due to patients with an inattentive presentation of ADHD being more likely to use substances for stimulation (Ohlmeier et al., 2008).

Hyperactivity/impulsivity symptoms have also been associated with substance use in individuals with ADHD (Miranda, Colomer, Berenguer, Roselló, & Roselló, 2016). Ohlmeier et al. (2008) found that individuals whose clinical presentation is dominated by hyperactivity/impulsivity symptoms have a higher use of high-risk drugs, such as heroin, compared to individuals with mainly inattentive symptoms. This was explained by the more experimental and reckless nature of such individuals.

Internalizing symptoms, which are not core symptoms of but common in individuals with ADHD (Danielson et al., 2018), have also been implicated in the risk of substance use. Internalizing symptoms have been proposed to partially develop as a consequence of the difficulties individuals with ADHD experience in various domains of life, which may affect their self-esteem (Barfield, 2018). It has been suggested that substance use may be used to manage such negative affect (Molina & Pelham, 2014). In line with this hypothesis, cross-sectional research has shown an increased risk of addictive illnesses for individuals with ADHD and comorbid disorders such as depression and anxiety (Ohlmeier et al., 2008). Prospective studies have also shown that children with internalizing symptoms are at an increased risk of early initiation of alcohol consumption (Conrod & Nikolaou, 2016). Internalizing traits such as neuroticism, hopelessness, and anxiety sensitivity have been associated with the tendency to report using substances to cope with anxiety and depression (Conrod & Nikolaou, 2016). However, there has been little research on this topic (Molina & Pelham, 2014).

In summary, some have suggested that individuals with ADHD may use substances for the alleviation of symptoms of the disorder. Furthermore, there have been findings of mainly cross-sectional associations between ADHD-specific as well as internalizing symptoms, and substance use in individuals with ADHD. Some research exists that supports the “self-medication” hypothesis, but more longitudinal research is needed to find

associations between early symptoms and the risk of developing SUDs. In the current study, we explore this association longitudinally.

1.5.4.3 Comorbidity

A great deal of literature suggests that the pathway from ADHD to SUDs is explained by the development of disruptive behavior disorders like ODD and CD, and that these co-occurring conditions increase the risk of SUDs (Mannuzza et al., 2004). In accordance with this explanation, behavior genetic studies have shown that externalizing disorders share a common genetic vulnerability to substance use (Conrod & Nikolaou, 2016).

Some researchers argue that the co-occurrence of externalizing problems, ODD or CD entirely explains the relationship between ADHD and SUD (Serra-Pinheiro et al., 2012; August et al., 2006). Rodgers et al. (2015) found indications that ADHD may have an effect on alcohol dependence, but not other SUDs, independently of CD/ODD. Finally, researchers have also argued that ADHD and CD/ODD may interact and cause a higher risk for SUDs than each of the individual causes of the disorders (Flory & Lynam, 2003).

In summary, conditions comorbid to ADHD have been found to be associated with substance use. In the current study, we will contribute to investigating the potential predictive effect of ADHD symptoms, as well as internalizing and externalizing problems in adolescence, on substance use in adulthood in individuals with ADHD.

1.6 Research aims

In conclusion, the longitudinal studies that follow children with ADHD to adulthood indicate a higher risk of both later substance use and abuse as well as criminal offences. In both instances, ODD and CD in childhood or adolescence elevates the risk and, in some studies, seems to account for them entirely. However, although it has been established that the diagnosis of ADHD increases the risk of crime and substance use problems, most children with ADHD avoid both crime and substance problems in later life. Furthermore, there seems to be a complex and not fully understood association between ADHD, substance use, and the emergence of criminality (Knecht et al., 2015). Few longitudinal studies have examined what specific characteristics of children and youth with ADHD may be used to predict later criminality and substance use. Further knowledge of such longitudinal associations enables a

better understanding of what factors determine whether a child or adolescent is more at risk of adverse outcomes. This information would provide a better foundation for early detection and intervention of criminality and substance use problems.

The present study aims to help bridge this gap in knowledge. First, it will investigate the prevalence and course of criminality among individuals with ADHD, as well as the prevalence of substance use in comparison to a healthy control group after 23 years (follow-up). Second, it will explore the potential predictive effects of neuropsychological functions, attention, internalizing, and externalizing problems, and comorbid ODD in adolescents with ADHD at baseline on criminality and substance use reported at follow-up.

1.6.1 Hypotheses

Based on the presented evidence, we have the following hypotheses:

- 1) Higher level of neuropsychological impairments in adolescents with ADHD (baseline), will predict more substance use and criminality 23 years later (follow-up).

- 2) Higher levels of ADHD symptoms (measured with the Child Behavior Checklist-4/18 (CBCL) Attention problems) and global symptoms (measured with the Global Assessment Scale of Symptoms) in adolescence will predict a higher instance of criminality and substance use at follow-up. Higher levels of externalizing problems (measured with the CBCL Externalizing) in adolescence will predict higher use of substances and more criminality at follow-up. Higher rates of internalizing problems (measured with the CBCL Internalizing) in adolescence will predict more alcohol and drug use at follow-up.

- 3) Comorbid oppositional defiant disorder in adolescents with ADHD (baseline) will predict more substance use and criminality at follow-up.

These hypotheses will be investigated with quantitative statistical methods described in Chapter 2. The results will be presented in Chapter 3, and discussed in Chapter 4.

2 Methods

This study is part of a larger research project based on data collected at three timepoints from 1992 to 2017. The original cross-sectional study at time-point 1 (T1) (Øie & Rund, 1999) compared groups of adolescents with early-onset schizophrenia, ADHD, and a healthy control group (HC) on a comprehensive neuropsychological test battery. After 13 years (time-point 2, T2), Øie and colleagues (see Øie, Sundet & Rund, 2010; Øie, Sundet & Ueland, 2011) investigated the development of neuropsychological functions and the associations between neuropsychological functions at T1 and functional outcomes at T2 in the same groups. The T2 investigation was repeated again 23 years after T1 (time-point 3, T3). The current study uses neuropsychological, overall and specific symptom measures, and comorbidity measures from T1 as predictor variables, and criminality measured at T2 and T3 and substance use measured at T3 as outcome measures. The study includes the 19 individuals in the ADHD, and 26 individuals in the Healthy Control group that were available for assessment at T3 ($N=45$).

2.1 Participants

2.1.1 Baseline/T1

A thorough description of the demographic information of the participants from T1 and T2 can be found in earlier publications (Øie & Rund, 1999; Øie et al., 2010; Øie et al., 2011). At baseline the ADHD group consisted of 20 male adolescents diagnosed with ADHD by senior clinicians using a semi-structured interview and the Parents Rating Scale (Wender, Wood & Reimherr, 1985), a standardized measure of hyperactivity, inattention and impulsivity at age 6-10. All participants had to meet the DSM-III-R criteria for ADHD, and had to have symptom-related problems both at school and at home. All participants were between 12 and 18 years old at T1. The ADHD group was significantly younger than the HC group ($F(1.43) = 13.6$ $p < .001$). The ADHD group ($N = 19$) had a mean age of 14 years ($SD = 1.5$ years) and the HC group ($N = 26$) had a mean age of 15.8 years ($SD = 1.7$ years).

The ADHD group was recruited from the National Centre for Child and Adolescent Psychiatry in Oslo, and were all outpatients. Fourteen of the participants were given additional diagnoses: oppositional defiant disorder (ODD) ($n = 9$), developmental reading

disorder (DRD) ($n = 2$), and both of the previous ($n = 3$). These comorbidities reflect previous findings in research on ADHD (Jensen, Martin & Cantwell, 1997), indicating that the sample is representative in this regard. At T1 12 participants used stimulant medication, but this was discontinued for at least 24 hours before the testing (methylphenidate, $n = 11$; dextroamphetamine, $n = 1$). One participant received haloperidol (1 mg/day) because of tics.

The HC group consisted of volunteers recruited from regular schools, 16 males and 14 females. They were screened at baseline with the Child Behavior Checklist 4-18 (CBCL) (Achenbach & Edelbrock, 1991), and all participants with a total raw score higher than 45 on the total measure were excluded. The mean total raw-score on the CBCL was 60.3 ($SD = 17.7$) in the ADHD group and 13.4 ($SD = 10.2$) in the HC group.

At T1, all participants were screened for factors likely to affect central nervous system functions, such as substance abuse, medical disease and previous head injuries with loss of consciousness. Another exclusion criteria was intellectual disability, with a score below 70 on the Wechsler Intelligence Scale for Children-Revised (WISC-R, Wechsler, 1974) serving as the cut-off.

2.1.2 13-year follow-up

At T2 19 participants in the ADHD group were available for retest, one was deceased (Norwegian Cause of Death Registry). The diagnosis was confirmed at T2 based on the DSM-IV in 15 of the participants, and four participants no longer met the diagnostic criteria for any psychiatric disorder. There is support for diagnostic continuity for ADHD between the DSM-III-R and the DSM-IV (Biederman et al., 1997). Four participants in the ADHD group were diagnosed with comorbid antisocial personality disorder and two with bipolar disorder at T2. Four of the ADHD participants had been hospitalized due to substance abuse between T1 and T2 ($M = 62.0$ weeks, $SD = 107.5$), but none of them were inpatients at T2 (Øie et al., 2011). Twenty-seven percent ($n = 5$) of the individuals received pharmacological treatment for ADHD.

2.1.3 23-year follow-up

All of the 19 participants in the ADHD group from T2 were available for retest at T3. The diagnosis was again confirmed based on the DSM-IV and eleven participants still fulfilled the criteria. Among these eleven, four participants fulfilled the criteria for ADHD only, while seven participants also fulfilled criteria for other disorders; five for depression or anxiety, one participant for a bipolar disorder, one fulfilled the criteria for Tourette's syndrome.

From T2 to T3, the number of individuals in the HC group were reduced from 30 to 26. One had an illness that prevented the person from participating, one was dead due to a medical disease (Norwegian Cause of Death Registry), and two declined to participate in the T3-study. The 26 individuals who remained in the study were included in the analyses.

All of the participants originally in the ADHD group, except from the one who was deceased, were included in the analyses ($n = 19$), including the eight whose ADHD had remitted. The goal of the study is to investigate outcomes in adolescents with ADHD, some of which later lose the diagnosis. To exclude those who later lost the diagnosis either due to other diagnoses or remission would remove positive outcomes from the study, create a bias towards negative outcomes and reduce the validity of the study.

2.2 Baseline measures

2.2.1 Neuropsychological measures

A comprehensive neuropsychological test battery was administered at T1 (Øie & Rund, 1999). The scores of tests that measure the same ability/domain were grouped together on the measures where we had several tests in order to increase the reliability of our scores. Using domains instead of individual tests increases statistical power and reduces the risk of type 1 errors that could occur with multiple analyses.

These neuropsychological domains were made from subtests showing satisfactory psychometric properties in the retest sample based on definitions given in the Øie and Rund (1999) study and modified according to Saykin et al. (1991, 1994; Øie et al., 2011). This was done by converting raw scores to standard z-scores on each test based on the means and standard deviations of the HC group (including the five participants who dropped out at T3) at T1, and then averaging each subject's z scores on tests within each domain. Where high

scores indicated impairment, the direction was reversed so that high scores always indicated better neuropsychological function.

We used the same eight aggregate measures as Øie et al. (2011); Executive function, Visual memory, Verbal memory, Visuomotor processing, Motor coordination, Auditory attention, Selective attention and Visual attention.

The Executive function domain consists of the Wisconsin Card Sorting Test (WCST): Perseverative responses (Heaton, 1981). The test measures the subjects ability to show flexibility in adapting to new rules (Nyhus & Barceló, 2009) versus the repeating of behaviors when they are no longer appropriate (Stedron, Sahni, & Munakata, 2005). As such, the number of perseverative responses measures important executive functions such as failing to inhibit inappropriate responses as well as working memory and cognitive flexibility. The WCST is a commonly used measure of executive function deficits in ADHD, and perseverative responses is one of the measures that best measures the differences between ADHD- and healthy control groups (Romine et al., 2004).

The Visual memory domain consists of the Kimura Recurring Figure test: Total correct score (Kimura, 1963), a valid and reliable instrument for the assessment of non-verbal memory function (Rixecker & Hartje, 1980).

The Verbal memory domain consists of the California Verbal Learning Test (CVLT), Total correct words at trial A1-5 (Delis, Kramer, Kaplan, & Ober, 1987). The CVLT is a widely used measure of the ability of verbal learning and memory (Yi, 2011).

The Visuomotor processing domain consists of the mean of Trail Making Test A, Trail Making Test B, measured as seconds to complete (Reitan & Wolfson, 1993), and the Digit Symbol–Coding from WISC–R (Wechsler, 1974) measured by number of symbols correctly coded in 120 seconds. The Trail Making Test (TMT) measures visual search, scanning, speed of processing and mental flexibility (Tombaugh, 2004). The Digit Symbol-Coding test measures psychomotor speed (Le Fever, 1985). The number of correct symbols within the allowed time is measured (Wechsler, 1974).

The Motor coordination domain consists of the Grooved Pegboard Test: Mean time in seconds to complete for dominant and non-dominant hand (Reitan & Wolfson, 1993). The Grooved Pegboard Test measures motor speed and eye-hand coordination performance with the dominant vs the non-dominant hand (Reitan & Wolfson, 1993).

The Auditory attention domain consists of the Seashore Rhythm Test: Mean number of correct answers (Reitan & Wolfson, 1993), Digit Spans maximum span forward and span

backward from WISC-R (Wechsler, 1974), and the Digit Span Distractibility Tests proportion of correctly repeated digits with and without distracter digits read in between targets (Oltmanns & Neale, 1975). The Seashore Rhythm test measure non-verbal auditory attention (Reitan & Wolfson, 1993). The Digit Span forward and backward and the Digit Span distractibility Test measure auditory attention and working memory (Wechsler, 1974).

The Selective attention domain consists of the Dichotic Listening task: Mean number of correct right ear answers from the Forced Right condition, and number of correct left ear answers from the Forced Left condition (Hugdahl & Andersson, 1986). The Dichotic Listening task is commonly used to measure auditory selective attention, which is the ability to attend to some stimuli, while suppressing others (Hugdahl & Andersson, 1986).

The Visual attention domain consists of the Backward Masking task: Mean number of correctly identified digits at the 33 ms and the 49-ms interstimulus intervals (Rund, Øie, & Sundet, 1996). The Backward Masking task assess the earliest stages of visual processing; pre-attentive processes. The mean number of correct responses in each condition were used as the measures of the visual attention domain.

In addition to the eight domains, we estimated a Full Scale IQ-measure (FSIQ) at T1 by combining Similarities and Block design from the WISC-R (Wechsler, 1974). We did not include the other measures from the WISC-R, Digit span and Digit Symbol-Coding, in our FSIQ estimate as they were included in measures of specific domains. We used Sattler & Dumont's (2004) table chart for estimating full-scale IQ (FSIQ) from combinations of subtests. This was to prohibit issues arising from computing general intelligence from only two subtests. Such issues entails the phenomenon of small deviations in sub-tests aggregated to larger deviations when combined, as well as the phenomenon of regression towards the mean.

2.2.2 Symptom, emotional, and behavioral problem measures

The Global Assessment Scale of Symptoms (GAS) was administered at T1. This scale is used by clinicians to rate patients' symptomatology and functioning on a 100-point scale where 1 is severe symptoms/lack of function and 100 is lack of symptoms/excellent functioning. The GAS has been found to have good reliability and validity in several populations (Endicott, Spitzer, Fleiss, & Cohen, 1976).

The Child Behavior Checklist/4-18 (CBCL) (Achenbach & Edelbrock, 1991) was filled out by the participant's mothers at T1. It is a part of the general questionnaire Achenbach System of Empirically Based Assessment (ASEBA). The CBCL is for children between 4-18 years of age, and measures children's emotional, social and behavior problems, as well as competences (Achenbach & Edelbrock, 1991). The scores are reported as T-scores, with high scores reflecting more problems. T-scores above 65 (93rd percentile) are considered to be in the borderline range, and T-scores above 70 (98th percentile) are considered to be in the clinical range (Achenbach & Edelbrock, 1991). The CBCL is one of the most widely used questionnaires for assessing children's behavior problems (Biederman et al., 1995). Decades of research has supported the validity and reliability of the questionnaire (Achenbach & Rescorla, 2001). The CBCL has eight empirically based syndrome scales; Anxious/Depressed, Withdrawn/Depressed, Somatic Complaints, Social Problems, Thought Problems, Attention Problems, Delinquent Behavior, and Aggressive Behavior. Due to the fact that attention problems are a core symptom of ADHD, we used the CBCL Attention problems scale. This scale has been shown to have "excellent convergence" with the diagnosis of ADHD in mixed samples (Biederman, 1993).

In addition, we also used two composite scores of the eight syndrome scales found by Achenbach & Edelbrock (1991) with second-order factor analyses of the syndrome scales; Internalizing problems and Externalizing problems. The CBCL Internalizing consists of the scales Anxious/Depressed, Withdrawn/Depressed, and Somatic Complaint problems. The CBCL Externalizing consists of Aggressive behavior and Delinquent behavior problems. We did not include the CBCL Social problems- or Thought problems scales. The choice of scales was guided by the knowledge of which symptoms are most central to the ADHD diagnosis and the most common comorbid disorders, as presented in the introduction. The composite scores are computed by summing the raw scores on the symptom scales included, and are then converted to and reported as T-scores. The Internalizing and Externalizing scales are not mutually exclusive, although they constitute different problems.

There is evidence for high construct validity of the CBCL (Achenbach & Rescorla, 2001). There is a significant association between the scores on the CBCL and concurrent scales of other instruments and with DSM criteria, including ADHD. The scale Attention problems correlates .53 with the DSM ADHD diagnosis, the scale Externalizing correlates .30 with DSM conduct disorder diagnosis, and the scale Internalizing correlates .45 with DSM depression diagnosis (Achenbach & Rescorla, 2001). The CBCL also have been found to

have a high criterion-related validity, as it significantly discriminates referred- and non-referred children (Achenbach & Rescorla, 2001).

2.2.3 Comorbidity measure

Comorbidity was diagnosed at T1 by a senior clinician using a semi-structured interview, according to DSM-III-R. As the comorbidity measure at T1, we computed a dichotomous measure named “Oppositional Defiant Disorder/No Oppositional Defiant Disorder”. The individuals with a comorbid ADHD and ODD diagnosis at T1 were scored as “1”, while the individuals with an ADHD diagnosis only were scored as “0”. We did not include the HC group in this measure, as none had a psychiatric diagnosis at T1.

2.3 Outcome measures

2.3.1 Criminality measures

Criminality was measured in the ADHD group at T2 and T3 in an interview. The participants responded how many times they had been convicted of a crime and how many days they had been incarcerated from T1 to T2 and later from T2 to T3. The face validity of self-report on criminality might not be the best, as it is not desirable to have been convicted and the participants might underreport. However, a review found that self-report measures of criminality match official records (Hindelang, Hirschi, & Weis, 1979), self-report measures of criminality have been found to be valid and reliable for research purposes (Thornberry & Krohn, 2000). Self-report of criminality is used in research all over the world (Klein, 2012).

We made measures of criminality by combining the data from T2 and T3. We made one measure for the number of convictions in total from T1 to T3, and one for number of total incarcerated days from T1 to T3. Combining the time points gives a more robust measure and increases the statistical power. We also made a dichotomous measure of criminality by recoding the number of convictions T2+T3 so that all convictions above zero were coded as one, while zero remained zero.

These outcome measures give us different kinds of information about the nature of the criminality; continuous and dichotomous. If it is a continuous variable, we can predict not only whether someone will be convicted of a crime, but the severity of this criminality, as

indicated by many short incarcerations or few long ones. This information is provided by our first two measures. If it is predictable as a dichotomous variable, we can predict the likelihood someone will be convicted of a crime or not, information provided by our third measure.

As a result, we ended up with three criminality measures; a measure of whether the subjects were convicted or not, a measure of number of the total number of convictions, and a measure of the total number of days convicted.

2.3.2 Substance use measures at T3

At T3 alcohol consumption was measured with the ten-item Alcohol Use Disorder Identification Test (AUDIT), and drug use was measured with the eleven-item Drug Use Disorder Identification Test (DUDIT). The AUDIT and the DUDIT are self-report screening tools that identify at-risk or problematic use of alcohol and drugs in the past 12 months (Babor, de la Fuente, Saunders & Grant, 1992; Berman, Bergman, Palmstierna, & Schlyter, 2005). The measures are based on the DSM-IV and ICD-10 criteria for substance use disorders, but are not sufficient to place a diagnosis (Babor et al, 1992; Berman et al., 2005). Both instruments use scoring on a continuous interval scale. The DUDIT provides a score from 0 to 44, with higher scores indicative of more severe drug problems. The DUDIT cut-off score for problematic use is recommended to be six for men and two for women (Berman et al., 2005). The AUDIT provides a score from 0 to 40, with higher scores indicative of more hazardous alcohol use patterns. Total scores of eight or more are used as a cut-off point for problematic use of alcohol (Babor et al., 1992). The AUDIT and the DUDIT have satisfactory reliability and validity for use as both as research tools, and as clinical tools (Bergman & Källmén, 2002; Hildebrand, 2015). The AUDIT and the DUDIT are widely used internationally as a screening tool for substance use disorders, and have generated a large body of research (Allen, Litten, Fertig, & Babor, 1997; Berman et al., 2005).

In addition to using the total scores of the AUDIT and the DUDIT, we made a dichotomous measure, based on the cut-off for problematic use on the AUDIT. The measure will be described as problematic/not problematic alcohol use. We were not able to use scores above cut-off for problematic use on the DUDIT because there was not enough variation in this measure to use it in the analyses. Instead, we made a dichotomous drug use/no drug use measure, with scores above zero on the DUDIT coded as 1, and scores of zero coded as 0. We

did not make a dichotomous alcohol/no alcohol-measure, as there was not enough variation in this measure.

2.4 Ethical considerations

The research design for the T1, T2 and T3 was approved by the Regional Committee for Medical Research Ethics in Eastern Norway (REK Øst-Norge REK 1 # 98-05-04113; 2015/180/REK sør-øst C), and the study was conducted in accordance with the Helsinki Declaration of the World Medical Assembly, and the Privacy Ombudsman for Research. Written consent was given by all participants after a complete description of the study, and also by the parents at T1. One potential negative consequence of participating was tiredness from the testing, the risk of this was minimized by repeated breaks during the testing. Feedback from the test results were given to the participants by the neuropsychologist testing them. At T2 and T3 the participants were given 500 NOK as compensation for participating in the study.

2.5 Analyses

2.5.1 Descriptive statistics

First, we looked at the descriptive statistics of the predictors at T1. Then we performed one-way Analyses of Variance (ANOVA) to investigate potential differences between the ADHD- and the HC group concerning the eight neuropsychological domains and the estimated FSIQ-measure, as well as the parent-rated problems as measured with CBCL at T1.

We then looked at the descriptive statistics of the outcome variables. Regarding criminality, we only had information about the ADHD group. On the substance measures we compared the descriptive statistics of the different drug and alcohol measures in both groups and performed one-way ANOVAs between the groups.

Finally, we looked at the descriptive statistics of co-occurring convictions (T2+T3) and problematic alcohol and drug use at T3.

2.5.2 Prediction of criminality

The first hypothesis was that neuropsychological abilities at T1 would predict criminality from T1 to T3. To test this, we performed Spearman rank order analyses. This method correlates the eight neuropsychological domains and the estimated FSIQ-measure at T1 with the rank order of subjects on the two continuous criminality measures; number of convictions and number of days sentenced at T2+T3. Because the continuous criminality variables did not meet the criteria of normality, we could not use parametric statistical methods with these measures. There are no tests of statistical significance for the Spearman correlations, but it is common to treat it as a normal Pearson correlation while being cautious about borderline cases (Howell, 2013), so we went with this approach. We also performed one-way ANOVAs with the eight neuropsychological domains and the estimated FSIQ-measure at T1 as independent variables and the dichotomous criminality variable (T2+T3) as the dependent variable.

The second hypothesis was that ADHD symptoms, global symptoms, and externalizing problems at T1 would predict criminality from T1 to T3 in ADHD populations. To test this, we performed Spearman rank order correlations for the continuous criminality variables and ANOVA for the dichotomous criminality variable. The analyses included scores on the CBCL at T1 and the GAS at T1 and the rank order of the two continuous criminality measures (T2+T3). The CBCL measures of externalizing problems, internalizing problems, attention problems, as well as the total CBCL score, all satisfied criteria for parametric statistics, and were all included in the analysis. One-way ANOVAs were performed, using the CBCL scores and the GAS scores at T1 as independent variables and the dichotomous criminality measure (T2+T3) as the dependent variable.

The third hypothesis was that comorbid ODD at T1 would predict criminality from T1 to T3. To test this hypothesis, Chi-square tests of independence were performed. Separate analyses were run for the association between comorbid ODD at T1 and the dichotomous criminality measure (T2+T3).

2.5.3 Prediction of substance use

We found that the AUDIT-measure met the criteria for normality, and thus we could use parametric statistics. However, the DUDIT-measure did not meet the criteria for normality, and thus we used non-parametric statistics with this measure. Since it is the prediction of

substance use in the ADHD group that is of interest in this study, we performed the analyses with this group only.

The first hypothesis was that neuropsychological functioning at T1 would predict alcohol and drug use at T3 in ADHD populations. To test this hypothesis we performed simple linear regression- and Spearman rank order analyses. In the simple linear regression analyses, we used the eight neuropsychological domains and the estimated FSIQ-measure at T1 as independent variables and the continuous AUDIT-measure at T3 as the dependent variable, and ran separate analyses for each independent variable. We could not control for confounding variables in our analyses, as our sample size is too small to lose additional degrees of freedom. The Spearman rank order analyses had the same independent variables at T1 and the rank order of the DUDIT scores at T3.

In addition to the regression analyses, we performed separate one-way ANOVAs with the same eight neuropsychological domains and estimated IQ as independent variables, and the dichotomous “problematic/not problematic alcohol use”, and the “drug use/no drug use” measure at T3 as the dependent measure.

The second hypothesis was that ADHD symptoms, global symptoms, and externalizing and internalizing problems at T1 would predict substance use at T3. To test this hypothesis we performed simple linear regression- and Spearman rank order analyses. In the simple linear regression analyses, the scores on the CBCL and the GAS at T1 were used as independent variables and the AUDIT measure at T3 as dependent variables. The same independent variables at T1 were correlated with the DUDIT measure at T3 in the Spearman rank order analyses. The CBCL measures at T1 of Externalizing problems, Internalizing problems, Attention problems, as well as the Total CBCL score, were included in the separate analyses. We also performed one-way ANOVAs with the symptom/problem measures at T1 and the dichotomous problematic alcohol-, and drug use/no drug use measure at T3.

To test the third hypothesis of whether comorbid conduct problems at T1 would predict problematic alcohol use at T3, Chi-square tests of independence were performed. Separate analyses were run for comorbid ODD at T1 and the dichotomous problematic alcohol use-, and drug use/no drug use measure at T3.

3 Results

3.1 Descriptive statistics

3.1.1 Baseline characteristics of the ADHD- and the HC group

Table 1.

Descriptive statistics and F-value of the eight neuropsychological domains, estimated FSIQ and symptoms at T1 in the ADHD- and HC group.

<i>Neuropsychological Domains and symptoms measured at T1</i>	<i>ADHD Group</i>		<i>HC Group</i>		<i>F-value</i>
	Mean	Std. Deviation	Mean	Std. Deviation	
Auditory attention	-1.0304	1.00365	-.0468	.73348	$F(1,42) = 14.135^{**}$
Executive function	-.4946	1.11537	-.0500	.99146	$F(1,40) = 1.869$
Motor function	-.7562	1.29828	.0867	.76611	$F(1,43) = 7.451^{**}$
Selective attention	.1215	.98391	-.0520	.89372	$F(1,43) = .380$
Verbal memory	-1.1969	1,09577	-.0386	.97878	$F(1,43) = 13.901^{**}$
Visual memory	-.6691	1.60872	-.0058	.97069	$F(1,43) = 2.962$
Visuomotor processing	-.9733	.99587	.0219	.79795	$F(1,43) = 13.846^{**}$
Visual attention	-.5160	.83506	-.0265	.92789	$F(1,43) = 3.319$
Estimated IQ	106.2105	12.66921	115.8462	15.67085	$F(1,43) = .033^*$
CBCL Attention problems	66.89	7.852	51.77	3.326	$F(1,43) = 77.896^{**}$
CBCL Externalizing problems	67.68	7.319	44.96	7.676	$F(1,43) = 100.008^{**}$
CBCL Internalizing	60.32	12.220	46.31	9.707	$F(1,43) = 18.365^{**}$
CBCL Total	67.74	6.109	44.65	8.494	$F(1,43) = 101.600^{**}$

*. F-value is significant at the 0.05 level

**.. F-value is significant at the 0.01 level

Table 1 list the descriptive statistics of the neuropsychological performance and the amount of emotional and behavioral problems. The groups differed with regard to four of the eight neuropsychological domains, as well as estimated FSIQ. The four domains were Auditory attention, Verbal memory, Visuomotor processing, and Motor function. The groups differed on all measures of emotional and behavioral problems.

3.1.2 Criminality

Nine of the individuals with ADHD diagnosis (47%) were convicted from T1 to T3. The number of convictions per person ranged from one to nine ($M = 1.9$, $SD = 2.6$). The total number of days the nine convicted subjects were sentenced ranged from 20 to 1648 days ($M = 273.5$, $SD = 516.7$). There was a slight decrease in total of crimes committed with age, from mean 21 convictions between T1 and T2, to mean 15 convictions from T2 to T3. Most of the individuals convicted of at least one crime by T2 were convicted of more crimes by T3 ($n = 7$). At T3 there was only one individual convicted of a crime that had not been convicted at least once by T2.

3.1.3 Substance use

The descriptive statistics are shown in Figure 1. Ninety-five percent of the individuals in the ADHD group reported consuming alcohol at T3, with the mean score on the AUDIT being 5.47 ($SD = 4.44$). In comparison, 88.5% of the HC group reported consuming alcohol, with a mean score of 5.19 ($SD = 4.63$). However, only 26.3% of the ADHD group and 11.5% of the HC group reported alcohol use classified as problematic on the AUDIT. In the ADHD group, 42.1% reported drug use ($M = 3.26$, $SD = 7.41$), and a quarter (10.5%) of these are classified as problematic use in the DUDIT. In the HC group 15.4% reported drug use ($M = .46$, $SD = 1.21$), but none were classified as having problematic use. None of the individuals in the ADHD group had both problematic alcohol and drug use at T3.

One-way ANOVAs showed no significant differences in substance use between the ADHD and HC groups, neither for total use, nor problematic use of alcohol or drugs. The differences in the groups on the scores on the AUDIT and the DUDIT are shown in Figure 1.

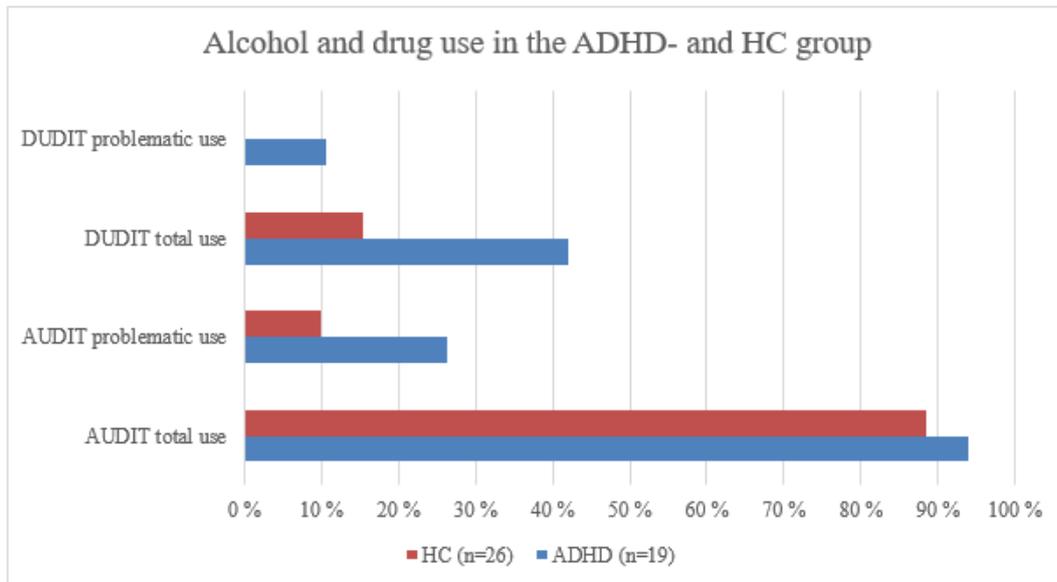


Figure 1. Percent of total and problematic drug use in the ADHD- and HC group as measured with the Alcohol Use Disorder Identification Test and the Drug Use Disorder Identification Test.

3.1.4 Co-occurring criminality and problematic substance use

The descriptive statistics showed that four individuals (21%) in the ADHD group had both problematic substance use (above cut-off on the AUDIT or DUDIT) at T2 and convictions (T2+T3). Of the five individuals with problematic alcohol use at T3, two of them had been convicted from T1 to T3 (40%). Of the two individuals with problematic drug use at T3, both of them had been convicted from T1 to T3.

3.2 Baseline predictors of criminality (T2+T3)

3.2.1 Neuropsychology

Spearman's rank order correlations were run to determine the relationship between the eight neuropsychological domains and the estimated FSIQ measure at T1 and the two continuous criminality measures (number of convictions in total, and number of days sentenced in total). There were no statistically significant correlations between the variables. Also, one-way ANOVAs were run to reveal any significant differences between the groups with and without

convictions (total from T1 to T3) on the neuropsychological domains at T1. No statistically significant differences were found.

3.2.2 Symptomatology, emotional and behavioral problems

3.2.2.1 The Child Behavior Checklist/4-18

Spearman's rank order correlation analyses were run to determine the associations between the CBCL Total, Attention problems, Externalizing and Internalizing scores at T1, and number of convictions (total from T1 to T3) and days sentenced (total from T1 to T3) in the ADHD group. The results, presented in Table 2, showed a statistically significant correlation between the scores on the CBCL Externalizing at T1 and number of convictions (two-tailed, $r_s=.527, p=.021$). There was also a statistically significant correlation between the scores on the CBCL Externalizing at T1 and number of days sentenced ($r_s=.584, p=.011$). The other correlations were not statistically significant.

One-way ANOVAs with the five CBCL scores at T1 as independent variables and the dichotomous criminality measure as dependent variable showed that the group with convictions from T1 to T3 had significantly higher scores on the CBCL Externalizing at T1, $F(1,18) = 5.690, p<.05$ (Convictions, $M = 71.44, SD = 6.97$, no convictions, $M = 64.30, SD = 6.09$). There were no significant differences between the group with and without convictions on the CBCL Total, Internalizing, or Attention problems score at T1.

3.2.2.2 The Global Assessment Scale of Symptoms (GAS)

The Spearman rank order correlations between the GAS score at T1 and number of days sentenced (T2+T3) and number of convictions (T2+T3), showed a statistically significant correlation between the GAS and number of days sentenced (two-tailed, $r_s=-.496, p=.037$). Higher symptom load at T1 was associated with higher number of days sentenced. There was no significant correlation between the GAS and number of convictions.

The GAS did not predict whether the subjects were convicted or not when performing a one-way ANOVA. The results of the ANOVA is presented in Table 3.

Table 2.

Spearman rank order correlations between symptom measures at T1 and criminality at T2+T3 in the ADHD-group (N=19).

<i>Variable at T1</i>	<i>Number of days sentenced (T2+T3)</i>	<i>Sig (two-tailed)</i>
CBCL		
Externalizing	.584	.011*
Internalizing	.043	.867
Attention problems	.197	.433
Total	.368	.133
Global Assessment Scale; Symptoms	-.496	.037*
<i>Variable at T1</i>	<i>Number of convictions (T2+T3)</i>	<i>Sig (two-tailed)</i>
CBCL		
Externalizing	.527	.021*
Internalizing	.036	.885
Attention problems	.244	.313
Total	.362	.128
Global Assessment Scale; Symptoms	-.447	.055

*. Correlation is significant at the 0.05 level (two-tailed)

Table 3.

One-way Analyses of Variance: Group differences on symptom measures at T1 between individuals with and without convictions at T3 in the ADHD-group (N=19).

Symptoms T1	<i>Convictions</i>		<i>No convictions</i>		<i>F(1,17)</i>	<i>p</i>
	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>		
CBCL/6-18						
CBCL Externalizing	71.44	6.10	64.30	6.10	5.690	.029*
CBCL Internalizing	61.33	11.46	59.40	13.42	.113	.741
CBCL Attention problems	67.56	8.70	66.30	7.44	.115	.739
CBCL Total	69.67	6.49	66.00	5.50	1.781	.200
Global Assessment Scale; Symptoms	48.78	6.22	53.90	7.13	2.757	.115

*. Group difference is significant at the 0.05 level (two-tailed)

3.2.3 Comorbid Oppositional Defiant Disorder

The chi square analysis between ODD/no ODD at T1 and convicted/not convicted at T3 showed no significant relation between the variables, $X^2(1, N = 19) = 0.9, p = .343$. Neither of these analyses show a significant relationship between the variables.

3.3 Baseline predictors of substance use

3.3.1 Neuropsychology

The simple linear regression- and the Spearman rank order analyses with the eight neuropsychological domains and the estimated FSIQ measure at T1 as independent variables did not predict the total use of alcohol or drugs measured by the AUDIT or the DUDIT at T3. We found no significant differences in the neuropsychological domains between the group with and without problematic alcohol use at T3 in the one-way ANOVAs.

3.3.2 Symptomatology, emotional and behavioral problems

3.3.2.1 The Child Behavior Checklist/4-18

The simple linear regression- and Spearman rank order analyses with scores on the CBCL at T1 as independent variables showed that scores on the CBCL did not predict total alcohol or drug use at T3 in participants with ADHD. The one-way ANOVAs with the scores on the CBCL at T1 as independent variables showed no group differences on the CBCL between the groups with problematic and not problematic alcohol use or between the groups with drug use/no drug use at T3.

3.3.2.2 The Global Assessment Scale of Symptoms (GAS)

The simple linear regression- and Spearman rank order analyses showed that scores on the GAS at T1 were not statistically significantly associated with drug or alcohol use at 23-year follow-up in the ADHD-group. The one-way ANOVAs showed no significant group differences on the GAS-scores between the group with problematic alcohol use and the group with no problematic alcohol use, when it comes to symptom load. Neither were there any differences on the GAS measure between the group with and without drug use.

3.3.3 Comorbid Oppositional Defiant Disorder

The chi square analysis between ODD/no ODD at T1 and problematic/non-problematic alcohol use at T3 showed no statistically significant relation between the variables, $X^2(1, N = 19) = .130, p = .719$. The association between ODD/no ODD at T1 and drug use/no drug use at T3 was not significant, $X^2(1, N = 19) = 1.659, p = .198$.

4 Discussion

4.1 The prevalence and course of criminality

One of the aims of this study was to investigate the prevalence and course of both criminality and substance use in individuals with ADHD. The results showed that about half of the 19 participants with ADHD had been convicted of a crime in the follow-up period from adolescence (T1) to adulthood (T3). This result is consistent with the prevalence of criminality found in another Scandinavian longitudinal study conducted by Dalsgaard et al. (2013). In the present study, the results revealed a slight decrease in crimes committed in adulthood, compared to adolescence and early adulthood. These findings are in line with research that have reported that criminality peaks at the ages of 18-20 years old and decreases steadily thereafter (Statistics Norway, 2018).

4.2 The prevalence of substance use

There were no significant differences in either alcohol or drug use between the ADHD- and the HC group at follow-up (T3). This does not replicate the results of other studies reporting an increased use of drugs and alcohol among adults with ADHD, compared to adults in the general population (Franke et al., 2018; Groenman et al., 2017). The present study shows that abstention from alcohol is a marginal phenomenon in both groups. Although there is no statistically significant difference between the groups when it comes to problematic alcohol use, there is a numerical difference. Three out of ten in the ADHD group had scores indicating problematic alcohol use, compared to only one out of ten in the HC group. This difference is comparable to that reported in Groenman et al.'s (2017) meta-analysis. This indicates that the lack of a statistical difference in problematic alcohol use might be due to the small sample size in the present study not providing adequate statistical power to detect any differences. A prospective longitudinal study by Molina et al. (2018) also found numerical, but not statistically significant, differences in alcohol use and misuse in early adulthood between an ADHD- and a healthy control group. They argued that further longitudinal research is needed before concluding that the risk for alcohol abuse is inconsequential for ADHD (Molina et al., 2018).

Although many of the individuals in the ADHD group do not necessarily have an alcohol problem, they could still be at risk for negative consequences. Adults with ADHD have been shown to have increased sensitivity to the negative effect of alcohol on inhibitory control compared to healthy controls (Weafer, Fillmore & Milich, 2009). The risks of alcohol, such as injury during intoxication (Halkjelsvik & Storvoll, 2015), may be exacerbated by the impulsive symptoms which puts individuals at risk for injuries (Merrill, Lyon, Baker & Gren, 2009). Furthermore, alcohol use may lead to the development of problematic use over time (Halkjelsvik & Storvoll, 2015).

Of the participants with ADHD, about 40% reported to have used drugs in the past year, compared to about 15 percent in the HC group. Although there is no statistically significant difference between the groups regarding drug use, the numerical difference is comparable to that of other studies (Groenman et al., 2017). Furthermore, none of the participants in the HC group reported problematic drug use, while around one out of ten reported problematic drug use in the ADHD group. This indicates that having ADHD diagnosed in childhood is a risk factor for problematic drug use in adulthood, even though there is no statistically significant difference between the two groups.

Of the individuals with problematic alcohol- and drug use at T3, 2 out of 5 and 2 out of 2 respectively, had committed crimes from T2 to T3. Although these are small groups of individuals, the fact that substantial proportions within the groups had committed crimes may reflect the findings from other studies that problematic substance use and criminality frequently co-occur in individuals with ADHD (Mannuzza et al., 2008; Gudjonsson et al., 2012; Knecht et al., 2015).

Consuming alcohol and using drugs when having the symptoms and impairments associated with ADHD, may cause more harm compared to others without such symptoms. Substance use may have a short-lived effect in ameliorating symptoms and neuropsychological impairment in individuals with ADHD, and thus can be used for self-medication purposes (Gould, 2010; Conrod & Nikolaou). On the other hand, in the long term such use can lead to greater problems, such as increased difficulties with the structuring of daily life (Kronenberg, Slager-Visscher, Goossens, van den Brink, & van Achterberg, 2014). This is unfortunate, as individuals with ADHD report that structure helps them function better, and the absence of structure contributes to the continuation of substance use (Kronenberg et al., 2014). The use of drugs or alcohol may also lead to impairment in neuropsychological functions, deteriorating physical and mental health, and disturb the

normal sleep-wake balance (Faraone et al., 2015; Stavro, Pelletier, & Potvin, 2013; Compton, Volkow, & Lopez, 2017; Lugoboni et al., 2017; van Emmerik-van Oortmerssen et al., 2014; Gould, 2010; Hser et al., 2017).

In summary, considering the small sample size in the present study, the findings indicate that the ADHD- and HC group are representative regarding substance use. Furthermore, the combination of symptoms and impairments associated with ADHD in combination with substance use may cause a range of negative consequences. As such, it is important to know more about early risk factors for substance use, which will be discussed further in the upcoming sections.

4.3 Neuropsychological function as a predictor

Our first hypothesis was that neuropsychological measures at baseline (T1) in the ADHD group would be associated with criminality and substance use at follow-up (T3). The hypothesis was based on research linking executive function impairments to substance use (Nigg et al., 2006) and criminality (Knecht et al., 2015).

4.3.1 Criminality

The results in the current study did not show support for our hypothesis that neuropsychological impairments at baseline would be associated with more criminality from T1 to T3 in the ADHD group. This may indicate that it is not the neuropsychological deficits in ADHD that predicts the risk for criminality in these individuals, or that our sample is too small to detect this association. As such, the current study did not find support for the hypothesis that impairment in prefrontal cortex, which is assumed to reflect impaired executive functions/impulsivity play an important role in predicting crime (Knecht et al., 2015; Hanoch et al., 2012). Further, we did not replicate findings of cross-sectional associations between executive deficits and criminality, an association found both in ADHD- and non-ADHD populations (Ginsberg et al., 2010; Morgan & Lilienfeld, 2000; Ogilvie et al., 2011). However, the ADHD group were impaired on only four of the eight neuropsychological domains. Even the EF measure, thought to measure a possible core impairment in ADHD, did not differ between the ADHD- and HC group. It is less probable to find a significant within-group prediction as long as the mean score and the standard deviation

did not deviate from normal performance. However, even in the neuropsychological domains that were significantly impaired with within-group variability of the same sizes as among the controls, there were no significant predictors for criminality. For instance, the estimated FSIQ-score was significantly lower in the ADHD group, compared to the HC group. This is not in line with evidence from a meta-analysis of longitudinal studies finding associations between IQ-deficits and later criminality in ADHD populations (Mohr-Jensen & Steinhausen, 2016).

Nevertheless, there still is a lack of studies that explore the longitudinal predictions of neuropsychological impairments on later criminality in individuals with ADHD.

4.3.2 Substance use

Neuropsychological functioning at T1 did not predict substance use at follow-up in the ADHD group. This is in accordance with evidence from other prospective longitudinal studies that have found no associations between neuropsychological measures in childhood and substance use in adolescence (four years of follow-up, Groenman et al., 2015), and executive function in adolescence and substance use in adulthood (five years of follow-up, Wilens et al., 2011). However, the same issue regarding significant differences between the ADHD- and HC group on only four of the neuropsychological measures, and estimated IQ, is also applicable to the current findings.

It is possible that by using an aggregate measure of neuropsychological functioning, we could have failed to detect more fine-grained effects (Groenman et al., 2015). For instance, Nigg et al. (2006) found that a measure of response inhibition as a measure of executive inhibition, predicted later substance use. It is possible that response inhibition is an executive function that is specifically sensitive to the effect of alcohol, and thus could partly explain which children with ADHD have increased SUD risk (Molina & Pelham, 2014), and that other specific measures could have equally specific associations.

As one such specific association, Molina & Pelham (2014) propose that expansion of neuropsychological test batteries with tests that measures motivation-reward processes and the interplay between these and higher-order executive functions, could better uncover neuropsychological vulnerabilities, especially for substance use disorders. A study on non-ADHD students conducted by Bø and colleagues (2017) included a measure of risky decision making in addition to tests measuring executive function. They found that risky decision

making, but not executive function, was associated with severity of binge drinking 18 months later (Bø, Billieux, Gjerde, Eilertsen, & Landrø, 2017). Other studies also have implicated a reward-pathway driven by sensation seeking in the risk of substance use in individuals with and without ADHD symptoms (Castellanos-Ryan et al., 2011, Handley et al., 2011).

The suggestion made by Molina & Pelham (2014) is in accordance with Conrod & Nikolaou's (2016) explanation of a dual-process model of control, which involves the cortico-basal-ganglia/limbic networks. The basal-ganglia/limbic circuitry drives behavior by a sensitivity towards rewards and positive affect. The prefrontal control circuits are implicated in adequate control of such responses. When these prefrontal functions are not fully developed, as is the case in adolescence, the vulnerability towards risky decisions such as experimentations with substances, is increased. The fact that adolescents with ADHD show increased impairment in prefrontal functions, makes them more vulnerable toward such risk-taking behavior (Martel, Nikolas, & Nigg, 2007). Further, this neurodevelopmental imbalance in the circuitry has been shown to be prolonged or aggravated by heavy substance use during adolescence, resulting in less controlled substance use, and such increasing the risk for SUDs (Conrod & Nikolaou, 2016). The suggestion made by Molina & Pelham (2014) is also supported by Adisetiyo & Gray (2017) who found that aberrant reward-processing underlying impulsivity could be the neural correlate of heightened SUD risk in ADHD populations.

Nevertheless, it seems that neuropsychological impairments in adolescence do not distinguish those individuals with ADHD that have a risk for substance use 23 years later. However, again, our sample may be too small to detect such individual differences in vulnerability.

4.4 Symptoms and emotional and behavioral problems as predictors

Our second hypothesis was that ADHD symptoms, global symptoms, and externalizing and internalizing problems in adolescence would be associated with criminality and substance use at follow-up (T3). We specified that externalizing problems would be related to criminality and substance use, while both externalizing and internalizing symptoms would be related to substance use.

4.4.1 Criminality

The current study found longitudinal associations between higher level of global symptoms (Global Assessment Scale) at T1 and the higher incidence of criminality in the ADHD group at follow-up. This finding is in line with other research reporting that the severity of ADHD symptoms in childhood, especially the hyperactive/impulsive symptoms, are associated with criminality in adulthood (Mohr-Jensen & Steinhausen, 2016; Barkley et al., 2004). According to Moffit & Scott (2008), children with ADHD have much greater genetic, neuropsychological and psychosocial burden than do healthy children, which are risk factors for later criminal behavior. Further, since individuals with ADHD are at risk of developing substance use disorders, this may be a possible trajectory from ADHD symptoms in adolescence to criminality in adulthood (Von Polier et al., 2012). According to Fletcher & Wolfe (2009), the finding that there is an increased risk of crimes among individuals with ADHD, fits with economic models of crime: that is, individuals with ADHD have lower expectations for job opportunities, and thus they are more likely to commit a crime than other otherwise similar adolescents and young adults. However, it is uncertain if this model is generalizable to the Norwegian society, which has a welfare system providing economic support for unemployed individuals.

Further, we found that parents reports of higher prevalence of externalizing problems (the CBCL Externalizing) in the children with ADHD at T1 were associated with higher incidence of criminality at follow-up (measured at T2 and T3). The CBCL Externalizing consists of aggressive and rule-breaking behavior, which we interpret to reflect the comorbid behavior problems often present in ADHD, as well as perhaps the impulsive symptoms of ADHD. Thus, our finding is consistent with prospective longitudinal studies that have shown a significant association between childhood impulsivity, conduct problems and future criminality (Erskine et al, 2016; Babinski, Hartsough & Lambert, 1999). In light of studies finding that impulsivity is a core feature in both ADHD and behavior/conduct problems (Martel, Levinson, Lee, & Smith, 2017), it is interesting that there were no significant association between the neuropsychological tests measuring impulsivity in adolescence and criminality in adulthood. One possible reason for this discrepancy, could be that neuropsychological tests are good at measuring so called modular functions, but not as good at measuring overarching regulatory functions. This is due to the fact that such overarching functions are not shown in the highly controlled test situation. This is where report forms such

as the CBCL-6/18 have their strengths, as they measure such functions in the context of everyday life-situations.

There could be several explanations as to why externalizing problems in adolescence are associated with later criminality. For one, the overabundance of externalizing problems in children and adolescents with ADHD, can result in fearlessness and the likelihood of breaking social norms (Eisenberg, Hofer, & Vaughan, 2007). Another explanation is that antisocial behavior in children with ADHD is highly heritable (Von Polier et al., 2012). Further, a possible pathway proposed by Molina & Pelham (2014) is that the presence of a difficult temperament in children with ADHD could interact with environmental factors such as parenting, and escalate behavior problems. This interaction could eventually result in deviant behavior such as criminality. Moreover, parents of children with ADHD and antisocial behavior more often suffer from psychiatric disorders (Pfiffner, McBurnett, Rathouz & Judice, 2005), which may cause further difficulties with parenting. Johnston & Jassy (2007) have proposed a model of child-parent interaction for children with ADHD where the child's symptoms of inattention or impulsivity increase the likelihood of parents responding in non-optimal manners towards the child. Examples of such parental behaviors were giving in to the child, having angry outbursts or withdrawing from the child. This interaction may result in the child developing oppositional/conduct problems (Johnston & Jassy, 2007).

Another hypothesis that may explain the association between externalizing behavior in adolescence and later criminality, could be that children with ADHD and behavior problems tend to affiliate with deviant peers, which increases the risk of developing antisocial behavior such as criminal offences (Pratt, Cullen, Blevins, Daigle & Unnever, 2002). Lee (2011) found that a variant of the MAOA-gene in interaction with affiliation with deviant peers increased the risk of antisocial behavior in a youth sample. This highlights the impact of the interplay and transactions of genes with the environment. Not only problematic peer affiliation, but also problems with peers - such as problematic peer interaction, social skills, peer victimization and externalizing behavior have been found to be prevalent among children with ADHD (Kok, Groen, Fuermaier, & Tucha, 2016). Furthermore, a longitudinal study from the MTA group, (see Murray-Close et al., 2010) followed children with ADHD, and found the development of vicious cycles among peer problems over time. Specifically, peer rejection lead to poorer social skills, which in turn resulted in further peer rejection. Such problems also affected other areas of functioning, leading to cascading effects over time.

A different explanation of the association between externalizing in adolescence and criminality in adulthood in the ADHD group, could be that the unfortunate psychosocial outcomes associated with ADHD and externalizing behaviors may lead to criminality (Erskine et al., 2016). For instance, even while receiving medication, children with ADHD have worse educational outcomes than their peers (Fleming et al., 2017). Research consistently show that poor academic achievement are risk factors for criminal behavior (Hinshaw, Carte, Sami, Treuting & Zupan, 2002). The interplay between psychosocial outcomes are complex, and they often interact and reinforce each other. Erskine et al. (2016) found that childhood ADHD and CD were associated with high-school drop-out, and they suggested that this may result in unemployment and financial stress later in life. This increases the likelihood of the development of SUDs, which again increase the risk of criminality (Mannuzza et al., 2008; Erskine et al., 2016).

To summarize, the finding that externalizing problems at T1 are associated with criminality at T2+T3 is consistent with other research, and there are several possible explanations for the association. Although the current study found an association between global symptoms and externalizing problems at baseline and criminality at follow-up, it is uncertain whether it is the diagnosis of ADHD or comorbid conditions that accounts for the associations discussed in this section. This question will be further discussed in the section about comorbid ODD as a predictor for criminality.

4.4.2 Substance use

In the current study there were no associations between the symptom and emotional and behavioral measures in adolescence and substance use 23 years later. This is likely due to the small sample size in the study. Other studies have found longitudinal and cross-sectional associations between inattentive and impulsive symptoms in children and adults with ADHD and substance use in adulthood (Molina & Pelham, 2003; Miranda et al., 2016).

As explained in the introduction, research indicate that individuals with ADHD may use substances as a way of coping with the symptoms of the disorder (Ohlmeier et al., 2008). Ohlmeier et al. (2008) argue that substances stimulate the release of dopamine, and therefore reduces symptoms of ADHD. Likewise, Conrod & Nikolaou (2016) argue that sensation seeking traits which are common in externalizing disorders increase both behavioral sensitivity, but also pharmacological sensitivity to the incentive-rewarding properties of

substances. In addition, impulsivity have been shown to cause increased sensitivity to stimulant substances, and this is proposed to be because of paradoxical effects on impulsivity in individuals with higher baseline levels of impulsivity (Conrod & Nikolaou, 2016). Individuals with the trait of impulsivity, often described as behavioral inhibition, have also been proposed to have an inherited common vulnerability for SUDs (Iacono et al., 2008). Other genetic and environmental factors contribute to influence the expression of the SUDs. Moreover, the individual also shapes his or her environment. This is thought to result in individual differences in which of the disorders an individual ends up developing (Iacono et al., 2008).

Further findings from the current study was the lack of an association between externalizing problems (CBCL Externalizing) at baseline and substance use at follow-up. This is conflicting with the many studies finding that comorbid ODD or CD increases the risk of substance use in individuals with ADHD (e.g. Serra-Pinheiro et al., 2012; August et al., 2006). The fact that many other studies have found this association may indicate that our lack of finding may be due to lack of statistical power.

In addition to the lack of associations between inattentive and externalizing problems in adolescence and substance use at follow-up, we did not find an association between internalizing problems in adolescence and substance use at follow-up. This is not in line with the findings that internalizing traits such as neuroticism, hopelessness and anxiety sensitivity are associated with the tendency to use substances as a coping mechanism (Conrod & Nikolaou, 2016), and that adults with comorbid ADHD and internalizing disorders have an increased risk of addiction (Ohlmeier et al., 2008). However, there have been little research on this topic in the literature about ADHD (Molina & Pelham, 2014). Molina & Pelham (2014) argue that the link between internalizing symptoms and substance use might be more relevant when it comes to adults with ADHD, because rates of depression increases, and more negative affect is caused by the withdrawal from substances.

4.5 Comorbid Oppositional Defiant Disorder as a predictor

The third hypothesis in the study was that the comorbid diagnoses of Oppositional Defiant Disorder in the ADHD group at baseline (T1) would be predictive of substance use and criminality at follow-up (T3). This hypothesis was based on the research that describes

comorbid behavior problems as a key predictor for substance use and criminality (e.g. Miranda et al., 2016; Mohr-Jensen, Bisgaard, Boldsen, & Steinhausen, 2019).

4.5.1 Criminality

The current study failed to provide support for the third hypothesis. Co-occurring ODD and ADHD at T1 were not associated with criminality from T1 to T3. This is conflicting with the findings from studies that have found prospective links between comorbid ODD in childhood and adulthood criminality in individuals with ADHD (Sibley et al., 2011). Moreover, it is conflicting with the findings from the study in the current study, which show that externalizing problems at baseline, which are the core symptoms of ODD, are associated with criminality in adulthood. As such, the lack of an association in the present study could be due to a power problem, because of our small sample size of 19 participants. In addition, the CBCL Externalizing measure a broader range of externalizing behavior than the ODD diagnosis, which may explain some of the discrepancy. However, others have also failed to find any difference in the risk of criminality between children diagnosed with ADHD and ODD, and children with ADHD only (Ayaz, Ayaz, & Kayan, 2015; Sibley et al., 2011). A recent meta-analysis by Mohr-Jensen & Steinhausen (2016) reported that few studies have investigated the effect of ODD on the risk of later convictions, and that many of these studies found no effect of the disorder. As such, it is possible that only more severe externalizing symptoms as indicated by conduct disorder is predictive of criminality in individuals with ADHD.

4.5.2 Substance use

There was no statistically significant association between comorbid ODD at T1 and substance use at follow-up (T3). This is conflicting with longitudinal studies that found an association between childhood ODD and ADHD and SUDs (Harpold et al., 2007; August et al., 2006). However, ODD is considered as less severe than CD, and thus the link between comorbid CD have been found to be stronger than that of ODD (Rodgers et al., 2015). Others have also failed to find any difference in the risk of substance use between children diagnosed with ADHD and ODD, and children with ADHD only (Ayaz et al., 2015; Groenman et al., 2013). As such the question of whether comorbid ODD in individuals with ADHD accounts for the risk of using substances is a relevant question that is still not resolved in the literature (Serra-

Pinheiro et al., 2012). Research suggests that a general externalizing spectrum model that indicate that the shared variance among externalizing disorders could be explained by common genetic and environmental influences, as well as premorbid personality traits and neuropsychological deficits (Castellanos-Ryan et al., 2011). In the research on ODD/CD-comorbidity, it could be useful to not only study the effect of the co-occurrence with ADHD, but of its development and interaction with ADHD and substance use and criminality over time. In addition, it could also be helpful to study the relation of ODD/CD to other mediators and moderators of SUD risk in ADHD (Molina & Pelham, 2014).

4.6 Implications for prevention and treatment

One of the main findings of the present study was that about half of the ADHD group had been convicted by follow-up (T3). Behavior problems measured by the CBCL Externalizing and symptom load measured by the GAS at baseline predicted criminality measures at T2 and T3. There was a substantial amount of alcohol and drug use in the ADHD group, but no significant differences between the ADHD- and control group. None of our other baseline variables could predict criminality or substance use at follow-up.

Providing individuals with ADHD the appropriate treatment that address all of their problems, including comorbid disorders and other secondary difficulties is an important public health challenge. Knowing which of the adolescents with ADHD that have an especially heightened risk of negative outcomes can be used to guide treatment, and to show which factors to target. This is important in order to use societal resources as effectively as possible, to reduce both personal and societal costs. Furthermore, it is important to focus on interventions in adolescence, as this is a particular crucial developmental period because of shifts from parental supervision to more independent interactions with peers and experimentations with alcohol and illicit drugs (Conrod & Nikolaou, 2016). Also, the early maturation of the fronto-basal-ganglia/limbic circuitry in adolescence, combined with the impairment of executive functions in individuals with ADHD, may increase the vulnerability towards substance use (Conrod & Nikolaou, 2016).

Early intervention is vital to avoid a cascade of the negative health and psychosocial problems found to be associated with ADHD (Erskine et al., 2016). The findings of the current study imply a need for long-term treatment planning from adolescence and into adulthood, especially in cases with high symptom load and externalizing symptoms. The

continued treatment and focus on possible challenges in adulthood, could potentially prevent these individuals from having unfortunate outcomes. In the Norwegian mental health care system, the transition from children's care to the adult health care system might entail changes in treatment and follow-up (Ørstadvik et al., 2016), which might be a critical period for the development of problems in adulthood. The 13-year follow-up study conducted by Øie et al. (2011) showed that most of the individuals with ADHD stopped receiving treatment when they turned 18. Twenty-seven percent received medication, while the other 73% did not receive any treatment despite the fact that 15 individuals still fulfilled the criteria for ADHD.

4.7 Strengths and limitations

There are several strengths of the study in the current study. These include thorough clinical assessment of the participants early in the illness process and a long follow-up time. The inclusion of a full neuropsychological test-battery is also a strength, as it allows for investigation of the variation within the ADHD group, and its association with later outcomes. The same is true for the symptom measures, where both ADHD-specific and more general psychiatric symptoms were included. This ensures a more complete overview of the total load of symptoms and behavior problems our participant's experience. The study also had a high retention rate. To our knowledge, this is the first study that have investigated the prediction of criminality and substance use longitudinally from neuropsychological functioning-, ADHD symptoms, as well as more general emotional and behavioral problems and ODD in adolescence.

Neuropsychology and symptoms can be seen as two core components of ADHD. Neuropsychological deficits can be conceptualized as the underlying deficits associated with ADHD, whereas symptoms reflect how these deficits translate into problems with functioning. As they are most commonly measured today, they have opposing strengths and weaknesses. Neuropsychological tests have high construct validity and reliability, but moderate validity when predicting everyday cognitive functioning (Chaytor & Schmitter-Edgecombe, 2003). Symptom and behavioral measures have great content validity and ecological validity, but also have an element of evaluation, which threatens reliability (Gomez, Burns, Walsh & De Moura, 2003). Their mutual inclusion strengthens this study.

The inclusion of measures such as The CBCL and GAS are a strength of the study as the instruments are frequently used in the children's mental health services in Norway

(Norwegian Society of Child and Adolescent Psychiatry, 2016; Norwegian Directorate of Health, 2008).

However, the study does have some limitations. As all clinical samples in Norway, the participants in the ADHD group fulfilled the ICD diagnosis of HKD as well. The criteria for HKD are stricter than those for ADHD, which would indicate that our ADHD sample is not representative for the whole ADHD diagnosis in the DSM, and as such our findings might not generalize to the whole of this group. This is important for all research done on ADHD in countries using the ICD in health care, especially when participants are recruited via the health care system. Santosh et al (2005) found that HKD was a moderator of treatment response when re-analysing data from an American treatment efficacy study and checking which participants also fit the requirements for HKD. The subgroup with HKD showed a greater effect of medication compared to behavioral interventions. Another limitation regarding generalizability of the findings, is that the ADHD sample only consisted of males.

The small sample size has low statistical power, meaning that some real associations might not come out as significant in our analyses. The small sample size would also limit the possibility of controlling for confounding variables that could account for the associations that were found in the analyses. As mentioned in the introduction, several researchers have noted the possibility of a referral bias in ADHD clinical populations (e.g. Biederman et al, 2002), which might also be true in this case. When it comes to symptom measures, the ADHD group had an inclusion criteria of minimum 45 on the CBCL total, which may have resulted in a restriction of range on this measure. While it is not likely that individuals that fulfill the criteria for ADHD have scores below the clinical cut-off on the CBCL, this is not guaranteed.

Another limitation is the lack of specificity regarding the measures of criminality and substance use, both over time and the nature of the phenomenon. A potential weakness is that we asked about convictions, rather than criminal behavior, so it is possible that we missed information if the participants committed criminal acts but were not caught. The nature of the crimes was also not recorded. Whether these crimes were impulsive or planned, aggressive or non-violent, carried out alone or in groups, might give important insights to the link between ADHD and criminality. The same is true for details about substance use. For instance, there were no diagnostics of SUDs at T3, only self-report of symptoms.

Neither criminality nor substance use was measured at T1. Moreover, substance abuse was an exclusion criteria in the study. Studies have found that 15-19% of Norwegian 13-15 year olds have consumed alcohol (Bakken, 2017; Kraus and Nociar, 2016). Age of debut with

substances has been shown to be a risk factor for progression to heavier substance use several negative outcomes (Richmond-Rakerd, Slutske, & Wood, 2017). In Norway, youth and young adults are the age groups that commit the most crime, and the most common age in the crime statistics is 19 years old (Bakken, 2017). This shows that our study might have missed information by not including a measure of alcohol and substance use, criminality or rule breaking at T1. These are possible confounding variables that may have had an effect on our analyses.

Remission and treatment was not controlled for in the analyses. Information about treatment other than medications received was not registered, but treatment history may serve as a confounding variable. The associations between persistence of ADHD into adulthood and other outcomes have been investigated, but with few longitudinal studies and mixed results (Breyer, Lee, Winters, August, & Realmuto, 2014). Where one study found that remittance reduced the risk of substance use to normal levels (Huntley & Young, 2012), another found no association between substance use and remittance (Breyer et al., 2014). It is thus possible that the rate of remission in the individuals in the study may have affected the results. However, this issue is difficult to control for in a study with this small of a sample.

The current study did not measure whether the participants had experienced trauma. De Sanctis, Newcorn and Halperin (2014), found that the experience of maltreatment accounted for the risk of delinquency in individuals with ADHD.

4.8 Recommendations for future research

More prospective longitudinal studies are still needed to understand the complex interaction between ADHD and negative outcomes such as criminality and substance use (Franke et al, 2018). The studies could benefit from including multiple risk factors to maximize prediction (Molina & Pelham, 2014). While there is strong support for ADHD populations having increased risk compared to the general population, there are fewer studies on the intragroup variation of risk in the ADHD population, especially longitudinally.

With regard to intragroup variation in the ADHD population, future research should focus on detail when it comes to the individual presentation. As studies focusing on the different subtypes of ADHD or on HKD, which is similar to the combined subtype, show that these subgroups have differences such as different levels of risk of negative outcomes (Murphy, Barkley & Bush, 2002) and different response to treatment such as medication

(Santosh et al., 2005). This is especially important when considering the increased likelihood of participants fulfilling HKD criteria in clinical samples from countries using the ICD in health care.

In-depth information about the nature of the crimes can help researcher to identify specific risks of different kinds of criminality. Such information might inspire new hypotheses about the nature of the relationship between the risk-factors and the outcome, which would enable better preventative measures. This would reduce both societal and personal costs related to criminality.

Further, as previously discussed, common negative psychosocial factors associated with ADHD should be further examined in relation to criminality and substance use. Such difficulties are among others poor academic achievement, impulsive choices, impairments in social skills, peer rejection and deviant peer affiliation. Such factors may serve as mediators or moderators of the ADHD symptoms' prediction of criminality and substance use. It would be especially useful to look at these factors in a population based study, such as that of Rowland et al. (2015) in order to get the complete picture and bypass potential referral biases, such as the gender bias suggested by Bruchmüller et al. (2012). Investigation of the transactions over time between these outcomes themselves, between the outcomes and the symptoms of ADHD and comorbid disorders would give important information. For instance, the issue of whether ADHD increases the risk of substance use and criminality beyond that of ODD/CD is unresolved, and more studies are needed to conclude on this topic. Longitudinal studies that use trajectory-based methods to distinguish individuals with specific patterns of functioning over time would be more useful instead of single point-in-time measurement (Molina & Pelham, 2014), like the current study has done.

As previously discussed, future longitudinal studies that examine the role of neuropsychological impairment on the risk of criminality and substance use, could benefit from including tests that measure reward-processing and decision making, such as the Iowa Gambling Task, to their neuropsychological test batteries (Bechara, Damasio, Damasio, & Anderson, 1994; Groenman et al., 2015; Molina & Pelham, 2014; Conrod & Nicolaou, 2016). It may also be relevant to measure specific components of these abilities, such as risky decision making (Bø et al., 2017).

The purpose of identifying risk factors for poor outcome is to guide interventions. Thus, it is important to adjust the results for the effect of ADHD treatment, and have a large enough sample size (Szobot & Bukstein, 2008). Moreover, one should analyze how

differences in treatment affect prognosis. One important question is whether break in treatment continuity from childhood to adult psychiatric services represents a risk factor. Another important issue that is in need of further research is the association between the persistence or remittance of the ADHD diagnosis and criminality and substance use (Breyer et al., 2014).

Finally, it could be interesting to know more about the symptoms, impairments and problems, as well as resources, from the children and adolescents affected of the disorder's point of view. Parental and teachers reports are influenced by their attitudes, beliefs, and health literacy (Barfield, 2018), and items are interpreted from their point of view (Gadow et al., 2004). In adults with ADHD, self-report measures have been found to have higher validity than informant reports (Sandra Kooij et al., 2008). Childhood self-report measures have been found to have acceptable validity and reliability, and provide useful information (Klimkeit et al., 2006).

4.9 Conclusion

In summary, this study found that a substantial proportion of individuals with ADHD in childhood have problematic substance use and commit criminal offences in adulthood. More global symptoms as measured with the Global Assessment Scale and CBCL, as well as externalizing problems in adolescence, were significantly associated with criminality at follow-up (T3). Neuropsychological impairments or comorbid Oppositional Defiant Disorder in adolescence were not significantly associated with criminality at follow-up. In addition, neither neuropsychological impairments nor comorbid conduct problems in adolescence predicted substance use 23 years later. These findings may have implications for prevention and treatment for adolescents with ADHD, such as the targeting of impulsive symptoms and behavior problems, and better long-term treatment for individuals with ADHD to prevent problematic substance use and criminality in adulthood.

5 Literature

- Abikoff, H. B., Jensen, P. S., Arnold, L. L. E., Hoza, B., Hechtman, L., Pollack, S., . . . Wigal, T. (2002). Observed Classroom Behavior of Children with ADHD: Relationship to Gender and Comorbidity. *Journal of Abnormal Child Psychology*, *30*(4), 349-359. Doi: 10.1023/A:1015713807297
- Achenbach, T. M., & Edelbrock, C. J. B. (1991). *Child Behavior Checklist*. Burlington (Vt), 7, 371-392.
- Achenbach, T. M., & Rescorla, L. (2001). *ASEBA School-Age Forms & Profiles*. Burlington, VT.
- Adisetiyo, V., & Gray, K. M. (2017). Neuroimaging the Neural Correlates of Increased Risk for Substance Use Disorders in Attention-Deficit/Hyperactivity Disorder—A Systematic Review. *The American Journal on Addictions*, *26*(2), 99-111. Doi: 10.1111/ajad.12500
- Agnew-Blais, J. C., Polanczyk, G. V., Danese, A., Wertz, J., Moffitt, T. E., & Arseneault, L. (2016). Evaluation of the Persistence, Remission, and Emergence of Attention Deficit/Hyperactivity Disorder in Young Adulthood. *JAMA Psychiatry*, *73*(7), 713-720. Doi:10.1001/jamapsychiatry.2016.0465
- Agnew-Blais, J. C., Polanczyk, G. V., Danese, A., Wertz, J., Moffitt, T. E., & Arseneault, L. (2018). Young Adult Mental Health and Functional Outcomes among Individuals with Remitted, Persistent and Late-Onset ADHD. *The British Journal of Psychiatry*, *213*(3): 526-534. Doi: 10.1192/bjp.2018.97
- Al Ghriwati, N., Langberg, J. M., Gardner, W., Peugh, J., Kelleher, K. J., Baum, R., . . . Epstein, J. N. (2017). Impact of Mental Health Comorbidities on the Community Based Pediatric Treatment and Outcomes of Children with Attention Deficit Hyperactivity Disorder. *Journal of developmental and behavioral pediatrics: JDBP*, *38*(1), 20–28. Doi: 10.1097/DBP.0000000000000359
- Allen, J. P., Litten, R. Z., Fertig, J. B., & Babor, T. (1997). A Review of Research on the Alcohol Use Disorders Identification Test (AUDIT). *Alcoholism: Clinical and Experimental Research*, *21*(4), 613-619. Doi: 10.1111/j.1530-0277.1997.tb03811.x
- American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.). Arlington, VA: American Psychiatric Publishing.
- Andersen, S. L., & Teicher, M. H. (2000). Sex Differences in Dopamine Receptors and Their

- Relevance to ADHD. *Neuroscience & Biobehavioral Reviews*, 24(1), 137-141.
Doi: 10.1016/S0149-7634(99)00044-5
- Andersson, H. W., Ådnanes, M., & Hatling, T. (2004). *Nasjonal kartlegging av tilbud om diagnostisering og helhetlig behandling av barn og ungdom med hyperkinetiske forstyrrelser/ADHD. [National Assessment of Diagnostic and Coherent Treatment of Children and Adolescents with Hyperkinetic Disorder/ADHD.]* Trondheim, Norway: SINTEF.
- Retrieved from: https://www.sintef.no/globalassets/upload/helse/psykisk_helse/pdf-filer/stf78_a045012---nasjonal-kartlegging-adhd.pdf [Norwegian]
- Armstrong, D., Lycett, K., Hiscock, H., Care, E., & Sciberras, E. (2015). Longitudinal Associations between Internalizing and Externalizing Comorbidities and Functional Outcomes for Children with ADHD. *Child Psychiatry & Human Development*, 46(5), 736-748. Doi: 10.1007/s10578-014-0515-x
- August, G. J., Winters, K. C., Realmuto, G. M., Fahnhorst, T., Botzet, A., & Lee, S. (2006). Prospective Study of Adolescent Drug Use among Community Samples of ADHD and Non-ADHD Participants. *Journal of the American Academy of Child & Adolescent Psychiatry*, 45(7), 824-832. Doi: 10.1097/01.chi.0000219831.16226.f8
- Ayaz, A. B., Ayaz, M., & Kayan, E. (2015). Negative Outcomes in Attention Deficit/Hyperactivity Disorder Comorbid with Oppositional Defiant Disorder. *Irish Journal of Psychological Medicine*, 32(4), 307-312. Doi: 10.1017/ipm.2014.91
- Babinski, L. M., Hartsough, C. S., & Lambert, N. M. (1999). Childhood Conduct Problems, Hyperactivity-Impulsivity, and Inattention as Predictors of Adult Criminal Activity. *The Journal of Child Psychology and Psychiatry and Allied Disciplines*, 40(3), 347-355. Retrieved from: <https://onlinelibrary.wiley.com/doi/abs/10.1111/14697610.00452>
- Babinski, D. E., Pelham, W. E., Jr., Molina, B. S. G., Gnagy, E. M., Waschbusch, D. A., Yu, J., . . . Karch, K. M. (2011). Late Adolescent and Young Adult Outcomes of Girls Diagnosed with ADHD in Childhood: An Exploratory Investigation. *Journal of Attention Disorders*, 15(3), 204-214. Doi:10.1177/1087054710361586
- Babor, T. F., de la Fuente, J. R., Saunders, J., & Grant, M. (1992). *AUDIT The Alcohol Use Disorders Identification Test: Guidelines for use in Primary Health Care*. Geneva: World Health Organization
- Bakken, A. (2017). Ungdata 2017. [Young Data 2017] *Rapport*, 10, 17. Oslo: NOVA.
Retrieved 07.04.2019 from

<http://www.hioa.no/Om-OsloMet/Senter-for-velferds-og-arbeidslivsforskning/NOVA/Publikasjoner/Rapporter/2017/Ungdata-2017>
[Norwegian]

- Barbarese, W. J., Colligan, R. C., Weaver, A. L., Voigt, R. G., Killian, J. M., & Katusic, S. K. (2013). Mortality, ADHD, and Psychosocial Adversity in Adults with Childhood ADHD: A Prospective Study. *Pediatrics, 131*(4), 637-644.
Doi: 10.1542/peds.20122354
- Barfield, P. A. (2018). Life Satisfaction in Children with Attention Deficit Hyperactivity Disorder: Looking Beyond Proxy Reports. *Journal of Child and Adolescent Psychiatric Nursing, 31*(4), 102-108. Doi:10.1111/jcap.12218
- Barkley, R. A. (1997). Behavioral Inhibition, Sustained attention, and Executive Functions: Constructing a Unifying Theory of ADHD. *Psychological Bulletin, 121*(1), 65-94.
Doi: 10.1037/0033-2909.121.1.65
- Barkley, R. A. (Ed.). (2014). *Attention-Deficit Hyperactivity Disorder: A Handbook for Diagnosis and Treatment*. New York, Guilford Publications.
- Barkley, R. A. (2016). Recent Longitudinal Studies of Childhood Attention Deficit/Hyperactivity Disorder: Important Themes and Questions for Further Research. *Journal of Abnormal Psychology, 125*(2), 248-255.
Doi: 10.1037/abn0000125
- Barkley, R. A., Fischer, M., Smallish, L., & Fletcher, K. (2004). Young Adult Follow-Up of Hyperactive Children: Antisocial Activities and Drug Use. *Journal of Child Psychology and Psychiatry, 45*(2), 195-211. Doi:10.1111/j.1469-7610.2004.00214.x
- Batty, M. J., Liddle, E. B., Pitiot, A., Toro, R., Groom, M. J., Scerif, G., . . . Hollis, C. (2010). Cortical Gray Matter in Attention-Deficit/Hyperactivity Disorder: A Structural Magnetic Resonance Imaging Study. *Journal of the American Academy of Child & Adolescent Psychiatry, 49*(3), 229-238. Doi: 10.1016/j.jaac.2009.11.008
- Bechara, A., Damasio, A. R., Damasio, H., & Anderson, S. W. (1994). Insensitivity to Future Consequences Following Damage to Human Prefrontal Cortex. *Cognition, 50*(1), 7-15. Doi: 10.1016/0010-0277(94)90018-3
- Bergman, H., & Källmén, H. (2002). Alcohol Use among Swedes and a Psychometric Evaluation of the Alcohol Use Disorders Identification Test. *Alcohol and Alcoholism, 37*(3), 245-251. Doi:10.1093/alcalc/37.3.245
- Berman, A. H., Bergman, H., Palmstierna, T., & Schlyter, F. (2005). Evaluation of the Drug

- Use Disorders Identification Test (DUDIT) in Criminal Justice and Detoxification Settings and in a Swedish Population Sample. *European Addiction Research*, 11(1), 22-31. Doi:10.1159/000081413
- Biederman, J., Faraone, S. V., Doyle, A., Lehman, B. K., Kraus, I., Perrin, J., & Tsuang, M. T. (1993). Convergence of the Child Behavior Checklist with Structured Interview Based Psychiatric Diagnoses of ADHD Children With and Without Comorbidity. *Journal of Child Psychology and Psychiatry*, 34(7), 1241-1251. Doi: 10.1111/j.1469-7610.1993.tb01785.x
- Biederman, J., Faraone, S., Milberger, S., Curtis, S., Chen, L., Marris, A., . . . Spencer, T. (1996). Predictors of Persistence and Remission of ADHD into Adolescence: Results From a Four-Year Prospective Follow-up Study. *Journal of the American Academy of Child & Adolescent Psychiatry*, 35(3), 343-351. Doi: 10.1097/00004583-199603000-00016
- Biederman, J., Faraone, S. V., Weber, W., Russell, R. L., Rater, M., & Park, K. S. (1997). Correspondence between DSM-III-R and DSM-IV Attention-Deficit/Hyperactivity Disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 36(12), 1682-1687. Doi: 10.1097/00004583-199712000-00016
- Biederman, J., Monuteaux, M. C., Doyle, A. E., Seidman, L. J., Wilens, T. E., Ferrero, F., . . . Faraone, S. V. (2004). Impact of Executive Function Deficits and Attention Deficit/Hyperactivity Disorder (ADHD) on Academic Outcomes in Children. *Journal of Consulting and Clinical Psychology*, 72(5), 757-766. Doi:10.1037/0022-006X.72.5.757
- Biederman, J., Mick, E., Faraone, S. V., Braaten, E., Doyle, A., Spencer, T., . . . Johnson, M. A. (2002). Influence of Gender on Attention Deficit Hyperactivity Disorder in Children Referred to a Psychiatric Clinic. *American Journal of Psychiatry*, 159(1), 36-42. Doi: 10.1176/appi.ajp.159.1.36
- Biederman, J., Newcorn, J., & Sprich, S. (1991). Comorbidity of Attention Deficit Hyperactivity Disorder. *American Journal of Psychiatry*, 148(5), 564-577. Doi: 10.1176/ajp.148.5.564
- Biederman, J., Petty, C. R., Monuteaux, M. C., Fried, R., Byrne, D., Mirto, T., . . . Faraone, S. V. (2010). Adult Psychiatric Outcomes of Girls with Attention Deficit Hyperactivity Disorder: 11-Year Follow-Up in a Longitudinal Case-Control Study. *American Journal of Psychiatry*, 167(4), 409-417. Doi: 10.1176/appi.ajp.2009.09050736

- Biederman, J., Wozniak, J., Kiely, K., Ablon, S., Faraone, S., Mick, E., . . . Kraus, I. (1995). CBCL Clinical Scales Discriminate Prepubertal Children with Structured Interview Derived Diagnosis of Mania from Those with ADHD. *Journal of the American Academy of Child & Adolescent Psychiatry*, 34(4), 464-471.
Doi: 10.1097/00004583-199504000-00013
- Breyer, J. L., Lee, S., Winters, K. C., August, G. J., & Realmuto, G. M. (2014). A Longitudinal Study of Childhood ADHD and Substance Dependence Disorders in Early Adulthood. *Psychology of Addictive Behaviors*, 28(1), 238-246.
Doi: 10.1037/a0035664
- Bridgett, D. J., & Walker, M. E. (2006). Intellectual Functioning in Adults with ADHD: A Meta-Analytic Examination of Full Scale IQ Differences between Adults with and Without ADHD. *Psychological Assessment*, 18(1), 1-14.
Doi: 10.1037/1040-3590.18.1.1
- Bruchmüller, K., Margraf, J., & Schneider, S. (2012). Is ADHD Diagnosed in Accord with Diagnostic Criteria? Overdiagnosis and Influence of Client Gender on Diagnosis. *Journal of Consulting and Clinical Psychology*, 80(1), 128. Doi: 10.1037/a0026582
- Burgess, P. W., Alderman, N., Forbes, C., Costello, A., Laure, M. C., Dawson, D. R., ... & Channon, S. (2006). The Case for the Development and Use of “Ecologically Valid” Measures of Executive Function in Experimental and Clinical Neuropsychology. *Journal of the International Neuropsychological Society*, 12(2), 194-209.
Doi: 10.1017/S1355617706060310
- Bø, R., Billieux, J., Gjerde, L. C., Eilertsen, E. M., & Landrø, N. I. (2017). Do Executive Functions Predict Binge-Drinking Patterns? Evidence from a Longitudinal Study in Young Adulthood. *Frontiers in Psychology*, 8. Doi:10.3389/fpsyg.2017.00489
- Cahill, B., Coolidge, F., Segal, D., Klebe, K., Marle, P., & Overmann, K. A. (2012). Prevalence of ADHD and its Subtypes in Male and Female Adult Prison Inmates. *Behavioral Sciences & the Law* 30(2). 154-166. Doi: 10.1002/bsl.2004
- Cantwell, D. P. (1996). Attention Deficit Disorder: A Review of the Past 10 Years. *Journal of the American Academy of Child & Adolescent Psychiatry*, 35(8), 978-987.
Doi: 10.1097/00004583-199608000-00008
- Carlson, C. L., Tamm, L., & Gaub, M. (1997). Gender Differences in Children with ADHD, ODD, and Co-Occurring ADHD/ODD Identified in a School Population. *Journal of the American Academy of Child & Adolescent Psychiatry*, 36(12), 1706-1714.

Doi: 10.1097/00004583-199712000-00019

- Castellanos-Ryan, N., Rubia, K., & Conrod, P. J. (2011). Response Inhibition and Reward Response Bias Mediate the Predictive Relationships between Impulsivity and Sensation Seeking and Common and Unique Variance in Conduct Disorder and Substance Misuse. *Alcoholism: Clinical and Experimental Research*, 35(1), 140-155. Doi:10.1111/j.1530-0277.2010.01331.x
- Caye, A., Rocha, T. B., Anselmi, L., Murray, J., Menezes, A. M., & Barros, F. C. (2016a). Attention-Deficit/Hyperactivity Disorder Trajectories from Childhood to Young Adulthood: Evidence from a Birth Cohort Supporting a Late-Onset Syndrome. *JAMA Psychiatry*, 73(7), 705-712. Doi: 10.1001/jamapsychiatry.2016.0383
- Caye, A., Swanson, J., Thapar, A., Sibley, M., Arseneault, L., Hechtman, L., . . . Rohde, L. A. (2016c). Life Span Studies of ADHD—Conceptual Challenges and Predictors of Persistence and Outcome. *Current Psychiatry Reports*, 18(12), 111. Doi: 10.1007/s11920-016-0750-x
- Chang, Z., Lichtenstein, P., Asherson, P. J., & Larsson, H. (2013). Developmental Twin Study of Attention Problems: High Heritabilities throughout Development. Developmental Twin Study of Attention Problems. *JAMA Psychiatry*, 70(3), 311-318. Doi: 10.1001/jamapsychiatry.2013.28
- Charach, A., Yeung, E., Climans, T., & Lillie, E. (2011). Childhood Attention Deficit/Hyperactivity Disorder and Future Substance Use Disorders: Comparative Meta-analyses. *Journal of the American Academy of Child & Adolescent Psychiatry*, 50(1), 9-21. Doi:10.1016/j.jaac.2010.09.019
- Chaytor, N., & Schmitter-Edgecombe, M. (2003). The Ecological Validity of Neuropsychological Tests: A Review of the Literature on Everyday Cognitive Skills. *Neuropsychology Review*, 13(4), 181-197. Doi:10.1023/B:NERV.0000009483.91468.fb
- Coghill, D., Toplak, M., Rhodes, S., & Adamo, N. (2018). Cognitive functioning in ADHD. In Banaschewski, T., Coghill, D., & Zuddas, A. (Ed), *Oxford Textbook of Attention Deficit Hyperactivity Disorder*. Oxford United Kingdom, Oxford University Press.
- Coghill, D. R., Seth, S., & Matthews, K. (2014). A Comprehensive Assessment of Memory, Delay Aversion, Timing, Inhibition, Decision Making and Variability in Attention Deficit Hyperactivity Disorder: Advancing Beyond the Three-Pathway Models. *Psychological Medicine*, 44(9), 1989-2001. Doi: 10.1017/S0033291713002547

- Compton, W. M., Volkow, N. D., & Lopez, M. F. (2017). Medical Marijuana Laws and Cannabis Use: Intersections of Health and Policy Medical Marijuana Laws and Cannabis Use Editorial. *JAMA Psychiatry*, *74*(6), 559-560.
Doi:10.1001/jamapsychiatry.2017.0723
- Connor, D., Steeber, J. & McBurnett, K. (2010). A Review of Attention Deficit/Hyperactivity Disorder Complicated by Symptoms of Oppositional Defiant Disorder or Conduct Disorder. *Journal of Developmental & Behavioral Pediatrics*, *31*(5), 427-440. Doi: 10.1097/DBP.0b013e3181e121bd
- Conrod, P. J., & Nikolaou, K. (2016). Annual Research Review: On the Developmental Neuropsychology of Substance Use Disorders. *Journal of Child Psychology and Psychiatry*, *57*(3), 371-394. Doi:10.1111/jcpp.12516
- Cortese, S. (2012). The Neurobiology and Genetics of Attention-Deficit/Hyperactivity Disorder (ADHD): What Every Clinician Should Know. *European Journal of Paediatric Neurology*, *16*(5), 422-433. Doi: 10.1016/j.ejpn.2012.01.009
- Cortese, S., Ferrin, M., Brandeis, D., Buitelaar, J., Daley, D., Dittmann, R. W., . . . Sonuga Barke, E. J. S. (2015). Cognitive Training for Attention-Deficit/Hyperactivity Disorder: Meta-Analysis of Clinical and Neuropsychological Outcomes From Randomized Controlled Trials. *Journal of the American Academy of Child & Adolescent Psychiatry*, *54*(3), 164-174. Doi:10.1016/j.jaac.2014.12.010
- Copeland, W. E., Shanahan, L., Costello, E., & Angold, A. (2009). Childhood and Adolescent Psychiatric Disorders as Predictors of Young Adult Disorders. *Archives of General Psychiatry*, *66*(7), 764-772. Doi: 10.1001/archgenpsychiatry.2009.85
- Counts, C. A., Nigg, J. T., Stawicki, J. A., Rappley, M. D., & Von Eye, A. (2005). Family Adversity in DSM-IV ADHD Combined and Inattentive Subtypes and Associated Disruptive Behavior Problems. *Journal of the American Academy of Child & Adolescent Psychiatry*, *44*(7), 690-698. Doi: 10.1097/01.chi.0000162582.87710.66
- Cuffe, S. P., Visser, S. N., Holbrook, J. R., Danielson, M. L., Geryk, L. L., Wolraich, M. L., & McKeown, R. E. (2015). ADHD and Psychiatric Comorbidity: Functional Outcomes in a School-Based Sample of Children. *Journal of Attention Disorders*, *1087054715613437*. Doi: 10.1177/1087054715613437
- Dalsgaard, S., Brøbech, P., Frydenberg, M., & Thomsen, P. (2013). Long-Term Criminal Outcome of Children with Attention Deficit Hyperactivity Disorder. *Criminal Behaviour and Mental health* *23*(2), 86-98. Doi: 10.1002/cbm.1860

- Dalsgaard, S., Mortensen, P. B., Frydenberg, M., & Thomsen, P. H. (2014). ADHD, Stimulant Treatment in Childhood and Subsequent Substance Abuse in Adulthood — A Naturalistic Long-Term Follow-Up Study. *Addictive Behaviors*, *39*(1), 325-328. Doi: 10.1016/j.addbeh.2013.09.002
- Dalsgaard, S., Østergaard, S. D., Leckman, J. F., Mortensen, P. B., & Pedersen, M. G. (2015). Mortality in Children, Adolescents, and Adults with Attention Deficit Hyperactivity Disorder: A Nationwide Cohort Study. *The Lancet*, *385*. Doi: 10.1016/s0140-6736(14)61684-6
- Damiano, D., & Forssberg, H. (2019). Poor Data Produce Poor Models: Children with Developmental Disabilities Deserve Better. *The Lancet Global Health*, *7*(2), e188. Doi: 10.1016/S2214-109X(18)30498-4
- De Sanctis, V. A., Newcorn, J. H., & Halperin, J. M. (2014). A prospective look at substance Use and Criminal Behavior in Urban ADHD Youth: What is the Role of Maltreatment History on Outcome? *ADHD Attention Deficit and Hyperactivity Disorders*, *6*(2), 79-86. Doi: 10.1007/s12402-013-0124-8
- Delis, D. C., Kramer, J. H., Kaplan, E., & Ober, B. A. (1987). *The California Verbal Learning Test Research Edition Manual*. San Antonio: Psychological Corporation.
- Dom, G., Hulstijn, W., & Sabbe, B. (2006). Differences in Impulsivity and Sensation Seeking between Early- and Late-Onset Alcoholics. *Addictive Behaviors*, *31*(2), 298-308. Doi:10.1016/j.addbeh.2005.05.009
- Einarsson, E., Sigurdsson, J. F., Gudjonsson, G. H., Newton, A. K., & Bragason, O. O. (2009). Screening for Attention-Deficit Hyperactivity Disorder and Co-morbid Mental Disorders among Prison Inmates. *Nordic Journal of Psychiatry*, *63*(5), 361-367. Doi:10.1080/08039480902759184
- Eisenberg, N., Hofer, C., & Vaughan, J. (2007). Effortful Control and Its Socioemotional Consequences. In J. J. Gross (Ed.), *Handbook of Emotion Regulation* (pp. 287-306). New York, NY, US: The Guilford Press.
- Endicott, J., Spitzer, R. L., Fleiss, J. L., & Cohen, J. (1976). The Global Assessment Scale: A Procedure for Measuring Overall Severity of Psychiatric Disturbance. *Archives of General Psychiatry*, *33*(6), 766-771. Doi:10.1001/archpsyc.1976.01770060086012
- Erskine, H. E., Ferrari, A. J., Nelson, P., Polanczyk, G. V., Flaxman, A. D., Vos, T., . . . Scott, J. G. (2013). Research Review: Epidemiological Modelling of Attention Deficit/Hyperactivity Disorder and Conduct Disorder for the Global Burden of

- Disease Study 2010. *Journal of Child Psychology and Psychiatry*, 54(12), 1263-1274.
Doi: 10.1111/jcpp.12144
- Erskine, H. E., Norman, R. E., Ferrari, A. J., Chan, G. C. K., Copeland, W. E., Whiteford, H. A., & Scott, J. G. (2016). Long-Term Outcomes of Attention-Deficit/Hyperactivity Disorder and Conduct Disorder: A Systematic Review and Meta-Analysis. *Journal of the American Academy of Child & Adolescent Psychiatry*, 55(10), 841-850.
Doi: 10.1016/j.jaac.2016.06.016
- Faraone, S. V., Asherson, P., Banaschewski, T., Biederman, J., Buitelaar, J. K., Ramos Quiroga, J. A., . . . Franke, B. (2015). Attention-Deficit/Hyperactivity Disorder. *Nature Reviews Disease Primers*, 1, 15020. Doi: 10.1038/nrdp.2015.20
- Faraone, S. V., Biederman, J., & Mick, E. (2006). The Age-Dependent Decline of Attention Deficit Hyperactivity Disorder: A Meta-Analysis of Follow-Up Studies. *Psychological Medicine*, 36(2), 159-165. Doi: 10.1017/s003329170500471x
- Faraone, S. V., Biederman, J., Spencer, T., Wilens, T., Seidman, L. J., Mick, E., & Doyle, A. E. (2000). Attention-Deficit/Hyperactivity Disorder in Adults: An Overview. *Biological Psychiatry*, 48(1), 9-20. Doi: 10.1016/S0006-3223(00)00889-1
- Faraone, S. V., & Larsson, H. (2018). Genetics of Attention Deficit Hyperactivity Disorder. *Molecular Psychiatry*. Doi: 10.1038/s41380-018-0070-0
- Faraone, S. V., Perlis, R. H., Doyle, A. E., Smoller, J. W., Goralnick, J. J., Holmgren, M. A., & Sklar, P. (2005). Molecular Genetics of Attention-Deficit/Hyperactivity Disorder. *Biological Psychiatry*, 57(11), 1313-1323. Doi: 10.1016/j.biopsych.2004.11.024
- Faraone, S. V., Sergeant, J., Gillberg, C., & Biederman, J. (2003). The Worldwide Prevalence of ADHD: Is It an American Condition?. *World psychiatry*, 2(2), 104-113. Retrieved from: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1525089/>
- Fayyad, J., Sampson, N. A., Hwang, I., Adamowski, T., Aguilar-Gaxiola, S., Al-Hamzawi, A., ... & Gureje, O. (2017). The Descriptive Epidemiology of DSM-IV Adult ADHD in the World Health Organization World Mental Health Surveys. *ADHD Attention Deficit and Hyperactivity Disorders*, 9(1), 47-65. Doi: 10.1007/s12402-016-0208-3
- Fitzgerald, M., Bellgrove, M. & Gill, M. (Ed.) (2007). *Handbook of Attention Deficit Hyperactivity Disorder*. Chichester: John Wiley & Sons, Ltd.
- Fleming, M., Fitton, C. A., Steiner, M. F. C., McLay, J. S., Clark, D., King, A., . . . Pell, J. P. (2017). Educational and Health Outcomes of Children Treated for Attention Deficit/Hyperactivity Disorder Educational and Health Outcomes of Children Treated

- for ADHD. *JAMA Pediatrics*, 171(7), e170691-e170691.
Doi: 10.1001/jamapediatrics.2017.0691
- Fletcher, J., & Wolfe, B. (2009). Long-Term Consequences of Childhood ADHD on Criminal Activities. *The Journal of Mental Health Policy and Economics*, 12(3), 119.
Doi: 10.2139/ssrn.1489147
- Flory, K., & Lynam, D. (2003). The Relation between Attention Deficit Hyperactivity Disorder and Substance abuse: What Role Does Conduct Disorder Play? *Clinical Child and Family Psychology Review*, 6, 1-16. Retrieved from:
<https://link.springer.com/article/10.1023/A%3A1022260221570>
- Franke, B., Michelini, G., Asherson, P., Banaschewski, T., Bilbow, A., Buitelaar, J. K., . . . Reif, A. (2018). Live fast, Die Young? A Review on the Developmental Trajectories of ADHD Across the Lifespan. *European Neuropsychopharmacology* 10, 1059-1088.
Doi: 10.1016/j.euroneuro.2018.08.001
- Frazier, T. W., Demaree, H. A., & Youngstrom, E. A. (2004). Meta-Analysis of Intellectual and Neuropsychological Test Performance in Attention-Deficit/Hyperactivity Disorder. *Neuropsychology* 18(3), 543-555. Doi: 10.1037/0894-4105.18.3.543
- Friedman, N. P., & Miyake, A. (2017). Unity and Diversity of Executive Functions: Individual Differences as a Window on Cognitive Structure. *Cortex*, 86, 186-204.
Doi: 10.1016/j.cortex.2016.04.023
- Froehlich, T. E., Lanphear, B. P., Epstein, J. N., Barbaresi, W. J., Katusic, S. K., & Kahn, R. S. (2007). Prevalence, Recognition, and Treatment of Attention-Deficit/Hyperactivity Disorder in a National Sample of U.S. Children. *Archives of Pediatrics and Adolescent Medicine*, 161, 857–864. Doi: 10.1001/archpedi.161.9.857
- Gadow, K. D., Drabick, D. A., Loney, J., Sprafkin, J., Salisbury, H., Azizian, A., & Schwartz, J. (2004). Comparison of ADHD Symptom Subtypes as Source-Specific Syndromes. *Journal of Child Psychology and Psychiatry*, 45(6), 1135-1149.
Doi: 10.1111/j.1469-7610.2004.00306.x
- Ginsberg, Y., Hirvikoski, T., & Lindefors, N. (2010). Attention Deficit Hyperactivity Disorder (ADHD) Among Longer-Term Prison Inmates is a Prevalent, Persistent and Disabling Disorder. *BMC Psychiatry*, 10(1), 112. Doi: 10.1186/1471-244X-10-112
- Gioia, G. A., Isquith, P. K., Guy, S. C., & Kenworthy, L. (2000). *BRIEF: Behavior Rating Inventory of Executive Function, Professional Manual*. Lutz, Florida: Psychological Assessment.

- Goldman, L. S., Genel, M., Bezman, R. J., Slanetz, P. J., for the Council on Scientific, A., & American Medical, A. (1998). Diagnosis and Treatment of Attention-Deficit/Hyperactivity Disorder in Children and Adolescents. *Journal of the American Medical Association*, 279(14), 1100-1107. Doi: 10.1001/jama.279.14.1100
- Gomez, R., Burns, G. L., Walsh, J. A., & De Moura, M. A. (2003). Multitrait-Multisource Confirmatory Factor Analytic Approach to the Construct Validity of ADHD Rating Scales. *Psychological Assessment*, 15(1), 3. Doi: 10.1037/1040-3590.15.1.3
- Gould, T. J. (2010). Addiction and Cognition. *Addiction science & clinical practice*, 5(2), 4-14. Retrieved from: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3120118/>
- Gray, C., Korczak, D., Andrews, D., & Bélanger, S. A. (2018). ADHD in Children and Youth: Part 1—Etiology, Diagnosis, and Comorbidity. *Paediatrics & Child Health*, 23(7), 447-453. Doi: 10.1093/pch/pxy109
- Groenman, A. P., Janssen, T. W. P., & Oosterlaan, J. (2017). Childhood Psychiatric Disorders as Risk Factor for Subsequent Substance Abuse: A meta-analysis. *Journal of the American Academy of Child & Adolescent Psychiatry*, 56(7), 556-569. Doi: 10.1016/j.jaac.2017.05.004
- Groenman, A. P., Oosterlaan, J., Greven, C. U., Vuijk, P. J., Rommelse, N., Franke, B., . . . Buitelaar, J. (2015). Neurocognitive Predictors of Substance Use Disorders and Nicotine Dependence in ADHD Probands, Their Unaffected Siblings, and Controls: A 4-year Prospective Follow-Up. *Journal of Child Psychology and Psychiatry*, 56(5), 521-529. Doi:10.1111/jcpp.12315
- Groenman, A. P., Oosterlaan, J., Rommelse, N., Franke, B., Roeyers, H., Oades, R. D., . . . Faraone, S. V. (2013). Substance Use Disorders in Adolescents with Attention Deficit Hyperactivity Disorder: A 4-Year Follow-Up Study. *Addiction*, 108(8), 1503-1511. Doi:10.1111/add.12188
- Gudjonsson, G. H., Sigurdsson, J. F., Sigfusdottir, I. D., & Young, S. (2012). A National Epidemiological Study of Offending and its Relationship with ADHD Symptoms and Associated Risk Factors. *Journal of Attention Disorders*, 18(1), 3-13. Doi: 10.1177/1087054712437584
- Gudjonsson, G. H., Sigurdsson, J. F., Young, S., Newton, A. K., & Peersen, M. (2009). Attention Deficit Hyperactivity Disorder (ADHD). How Do ADHD Symptoms Relate to Personality among Prisoners? *Personality and Individual Differences*, 47(1), 64-68. Doi: 10.1016/j.paid.2009.01.048

- Gustavson, D. E., Stallings, M. C., Corley, R. P., Miyake, A., Hewitt, J. K., & Friedman, N. P. (2017). Executive Functions and Substance Use: Relations in Late Adolescence and Early Adulthood. *Journal of abnormal psychology, 126*(2), 257.
Doi: 10.1037/abn0000250
- Halkjelsvik, T., & Storvoll, E. E. (2015). Andel av befolkningen i Norge med et risikofyllt alkoholkonsum målt gjennom Alcohol Use Disorders Identification Test (AUDIT) [Proportion of the Population in Norway with a Hazardous Alcohol Consume Measured with the Alcohol Use Disorders Identification Test (AUDIT)]. *Nordic Studies on Alcohol and Drugs, 32*(1), 61-72. Doi: 10.1515/nsad-2015-0008
[Norwegian]
- Handley, E. D., Chassin, L., Haller, M. M., Bountress, K. E., Dandreaux, D., & Beltran, I. (2011). Do Executive and Reactive Disinhibition Mediate the Effects of Familial Substance Use Disorders on Adolescent Externalizing Outcomes? *Journal of Abnormal Psychology, 120*(3), 528-542. Doi: 10.1037/a0024162
- Hanoch, Y., Gummerum, M., & Rolison, J. (2012). Second-to-Fourth Digit Ratio and Impulsivity: A Comparison between Offenders and Nonoffenders. *PLoS One, 7*(10), e47140. Doi:10.1371/journal.pone.0047140
- Harpold, T., Biederman, J., Gignac, M., Hammerness, P., Surman, C., Potter, A., & Mick, E. (2007). Is Oppositional Defiant Disorder a Meaningful Diagnosis in Adults?: Results from a Large Sample of Adults with ADHD. *The Journal of Nervous and Mental Disease 195*, 601–605. Doi: 10.1097/NMD.0b013e318093f448
- Hart, E. L., Lahey, B. B., Loeber, R., Applegate, B., & Frick, P. J. (1995). Developmental Change in Attention-Deficit Hyperactivity Disorder in Boys: A Four-Year Longitudinal Study. *Journal of Abnormal Child Psychology, 23*(6), 729-749.
Doi: 10.1007/BF01447474
- Heaton, R. K. (1981). *Wisconsin Card Sorting Test, Manual*. Odessa, FL: Psychological Assessment Resources.
- Hechtman, L., Swanson, J. M., Sibley, M. H., Stehli, A., Owens, E. B., Mitchell, J. T., . . . Group, M. T. A. C. (2016). Functional Adult Outcomes 16 Years after Childhood Diagnosis of Attention-Deficit/Hyperactivity Disorder: MTA Results. *Journal of the American Academy of Child & Adolescent Psychiatry, 55*(11), 945-952 e942.
Doi: 10.1016/j.jaac.2016.07.774
- Helsedirektoratet. (2017). *ADHD/Hyperkinetisk Forstyrrelse – Nasjonal faglig retningslinje*

- for utredning, behandling og oppfølging.* [Norwegian Directorate of Health. (2017) ADHD/Hyperkinetic Disorder -- National Professional Guidelines for Assessment, Treatment and Follow-up.] Retrieved 29.01.2019 from:
<https://helsedirektoratet.no/retningslinjer/adhd> [Norwegian]
- Helsedirektoratet (2008). *Veileder for poliklinikker i psykisk helsevern for barn og unge.* [Norwegian Directorate of Health (2008) Guidelines for Outpatient Clinics in Mental Health Care for Children and Adolescents]. Retrieved 07.04.2019 from
<https://helsedirektoratet.no/folkehelse/psykisk-helse-og-rus/psykisk-helsevern/psykisk-helsevern-for-barn-og-unge> [Norwegian]
- Hildebrand, M. (2015). The Psychometric Properties of the Drug Use Disorders Identification Test (DUDIT): A Review of Recent Research. *Journal of Substance Abuse Treatment*, 53, 52-59. Doi:10.1016/j.jsat.2015.01.008
- Hindelang, M. J., Hirschi, T., & Weis, J. G. (1979). Correlates of Delinquency: The Illusion of Discrepancy between Self-Report and Official Measures. *American Sociological Review*, 995-1014. Doi: 10.1177/1477370815578198
- Hinshaw, S. P. (2018). Attention Deficit Hyperactivity Disorder (ADHD): Controversy, Developmental Mechanisms, and Multiple Levels of Analysis. *Annual Review of Clinical Psychology*, 14(1), 291-316. Doi: 10.1146/annurev-clinpsy-050817-084917
- Hinshaw, S. P., & Arnold, L. E. (2015). ADHD, Multimodal Treatment, and Longitudinal Outcome: Evidence, Paradox, and Challenge. *Wiley Interdisciplinary Reviews. Cognitive science*, 6(1), 39-52. Doi: 10.1002/wcs.1324
- Hinshaw, S. P., Carte, E. T., Sami, N., Treuting, J. J., & Zupan, B. A. (2002). Preadolescent Girls with Attention-Deficit/Hyperactivity Disorder: II. Neuropsychological Performance in Relation to Subtypes and Individual Classification. *Journal of Consulting and Clinical Psychology*, 70(5), 1099–1111.
 Doi: 10.1037/0022-006X.70.5.1099
- Howell, D. C. (2013). *Statistical Methods for Psychology* (8. ed.): Belmont, CA: Wadsworth Publishing.
- Hser, Y.-I., Mooney, L. J., Huang, D., Zhu, Y., Tomko, R. L., McClure, E., . . . Gray, K. M. (2017). Reductions in Cannabis Use are Associated with Improvements in Anxiety, Depression, and Sleep Quality, but not Quality of Life. *Journal of Substance Abuse Treatment*, 81, 53-58. Doi: 10.1016/j.jsat.2017.07.012
- Hugdahl, K., & Andersson, L. (1986). The “Forced-Attention” Paradigm in Dichotic

- Listening to CV-Syllables: A Comparison between Adults and Children. *Cortex*, 22, 417–432. Doi: 10.1016/S0010-9452(86)80005-3
- Humphreys, K. L., Eng, T., & Lee, S. S. (2013). Stimulant Medication and Substance Use Outcomes: A Meta-Analysis Stimulant Medication and Substance Use Stimulant Medication and Substance Use. *JAMA Psychiatry*, 70(7), 740-749. Doi: 10.1001/jamapsychiatry.2013.1273
- Huntley, Z. and S. Young (2012). Alcohol and Substance Use History among ADHD Adults: The Relationship with Persistent and Remitting Symptoms, Personality, Employment, and History of Service Use. *Journal of Attention Disorders* 18(1): 82-90. Doi: 10.1177/1087054712446171
- Iacono, W. G., Malone, S. M., & McGue, M. (2008). Behavioral Disinhibition and the Development of Early-Onset Addiction: Common and Specific Influences. *Annual Review of Clinical Psychology*, 4(1), 325-348. Doi:10.1146/annurev.clinpsy.4.022007.141157
- Jensen, P. S., Arnold, L. E., Swanson, J. M., Vitiello, B., Abikoff, H. B., Greenhill, L. L., . . . Hur, K. (2007). 3-Year Follow-up of the NIMH MTA Study. *Journal of the American Academy of Child & Adolescent Psychiatry*, 46(8), 989-1002. Doi: 10.1097/CHI.0b013e3180686d48
- Jensen, P. S., Martin, D., & Cantwell, D. P. (1997). Comorbidity in ADHD: Implications for Research, Practice, and DSM-V. *Journal of the American Academy of Child & Adolescent Psychiatry*, 36(8), 1065-1079. Doi: 10.1097/00004583-199708000-00014
- Jensen, P. S., Hinshaw, S. P., Kraemer, H. C., Lenora, N., Newcorn, J. H., Abikoff, H. B., . . . Vitiello, B. (2001). ADHD Comorbidity Findings from the MTA Study: Comparing Comorbid Subgroups. *Journal of the American Academy of Child & Adolescent Psychiatry*, 40(2), 147-158. Doi: 10.1097/00004583-200102000-00009
- Johnston, C., & Jassy, J. S. (2007). Attention-Deficit/Hyperactivity Disorder and Oppositional/Conduct Problems: Links to Parent-Child Interactions. *Journal of the Canadian Academy of Child and Adolescent Psychiatry = Journal de l'Academie canadienne de psychiatrie de l'enfant et de l'adolescent*, 16(2), 74-79. Retrieved from: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2242643/>
- Kalivas, P. W., & Volkow, N. D. (2005). The Neural Basis of Addiction: A Pathology of Motivation and Choice. *American Journal of Psychiatry*, 162(8), 1403-1413. Doi: 10.1176/appi.ajp.162.8.1403

- Khalife, N., Kantomaa, M., Glover, V., Tammelin, T., Laitinen, J., Ebeling, H., . . .
Rodriguez, A. (2014). Childhood Attention-Deficit/Hyperactivity Disorder Symptoms Are Risk Factors for Obesity and Physical Inactivity in Adolescence. *Journal of the American Academy of Child & Adolescent Psychiatry*, *53*(4), 425-436.
Doi: 10.1016/j.jaac.2014.01.009
- Kimura, D. (1963). Right Temporal-Lobe Damage. Perception of Unfamiliar Stimuli after Damage. *Archives of Neurology*, *8*, 264–271.
Doi: 10.1001/archneur.1963.00460030048004
- Klein, M. (Ed.). (2012). *Cross-National Research in Self-Reported Crime and Delinquency* (Vol. 50). Springer Science & Business Media.
- Klimkeit, E., Graham, C., Lee, P., Morling, M., Russo, D., & Tonge, B. (2006). Children Should be Seen and Heard: Self-Report of Feelings and Behaviors in Primary-School Age Children with ADHD. *Journal of Attention Disorders*, *10*(2), 181-191.
Doi: 10.1177/1087054706289926
- Knecht, C., de Alvaro, R., Martinez-Raga, J., & Balanza-Martinez, V. (2015). Attention Deficit Hyperactivity Disorder (ADHD), Substance Use Disorders, and Criminality: A Difficult Problem with Complex Solutions. *International Journal of Adolescent Medicine and Health*, *27*(2), 163-175. Doi: 10.1515/ijamh-2015-5007
- Kok, F. M., Groen, Y., Fuermaier, A. B. M., & Tucha, O. (2016). Problematic Peer Functioning in Girls with ADHD: A Systematic Literature Review. *PLoS One*, *11*(11), e0165119. Doi: 10.1371/journal.pone.0165119
- Kolla, N. J., van der Maas, M., Erickson, P. G., Mann, R. E., Seeley, J., & Vingilis, E. (2018). Attention Deficit Hyperactivity Disorder and Arrest History: Differential Association of Clinical Characteristics by Sex. *International Journal of Law and Psychiatry*, *58*, 150-156. Doi: 10.1016/j.ijlp.2018.04.006
- Kraus, L., & Nociar, A. (2016). *ESPAD Report 2015: Results from the European School Survey Project on Alcohol and Other Drugs*. European Monitoring Centre for Drugs and Drug Addiction.
- Kronenberg, L. M., Slager-Visscher, K., Goossens, P. J. J., van den Brink, W., & van Achterberg, T. (2014). Everyday Life Consequences of Substance Use in Adult Patients with a Substance Use Disorder (SUD) and Co-Occurring Attention Deficit/Hyperactivity Disorder (ADHD) or Autism Spectrum Disorder (ASD): A Patient's Perspective. *BMC Psychiatry*, *14*(1), 264. Doi: 10.1186/s12888-014-0264-1

- Lara, C., Fayyad, J., De Graaf, R., Kessler, R. C., Aguilar-Gaxiola, S., Angermeyer, M., ... & Karam, E. G. (2009). Childhood Predictors of Adult Attention-Deficit/Hyperactivity Disorder: Results from the World Health Organization World Mental Health Survey Initiative. *Biological psychiatry*, *65*(1), 46-54. Doi: 10.1016/j.biopsych.2008.10.005
- Lee, S. S., Humphreys, K. L., Flory, K., Liu, R., & Glass, K. (2011). Prospective Association of Childhood Attention-Deficit/Hyperactivity Disorder (ADHD) and Substance Use and Abuse/Dependence: A Meta-Analytic Review. *Clinical Psychology Review* *31*(3): 328-341. Doi: 10.1016/j.cpr.2011.01.006
- Lee, S. S. (2011). Deviant Peer Affiliation and Antisocial Behavior: Interaction with Monoamine Oxidase A (MAOA) Genotype. *Journal of Abnormal Child Psychology*, *39*(3), 321-332. Doi: 10.1007/s10802-010-9474-2
- Levy, F. (1991). The Dopamine Theory of Attention Deficit Hyperactivity Disorder (ADHD). *Australian and New Zealand Journal of Psychiatry*, *25*, 277–283. Doi: 10.3109/00048679109077746
- Le Fever, F. F. (1985). A Noncoding Motoric Equivalent Measures Most of What the Digit Symbol Does, Including Age Changes. *Perceptual and Motor Skills*, *61*(2), 371–377. Doi: 10.2466/pms.1985.61.2.371
- Luman, M., Oosterlaan, J., & Sergeant, J. A. (2008). Modulation of Response Timing in ADHD, Effects of Reinforcement Valence and Magnitude. *Journal of Abnormal Child Psychology*, *36*(3), 445-456. Doi: 10.1007/s10802-007-9190-8
- Luo, S. X. and Levin, F. R. (2017). Towards Precision Addiction Treatment: New Findings in Co-morbid Substance Use and Attention-Deficit Hyperactivity Disorders. *Current Psychiatry Reports* *19*(3): 14. Doi: 10.1007/s11920-017-0769-7
- Lugoboni, F., Levin, F. R., Pieri, M. C., Manfredini, M., Zamboni, L., Somaini, L., & Gerra, G. (2017). Co-occurring Attention Deficit Hyperactivity Disorder Symptoms in Adults Affected by Heroin Dependence: Patients Characteristics and Treatment Needs. *Psychiatry Research*, *250*, 210-216. Doi: 10.1016/j.psychres.2017.01.052
- Mannuzza, S., Klein, R. G., Abikoff, H., & Moulton Iii, J. L. (2004). Significance of Childhood Conduct Problems to Later Development of Conduct Disorder among Children with ADHD: A Prospective Follow-Up Study. *Journal of Abnormal Child Psychology*, *32*(5), 565-573. Doi: 10.1023/B:JACP.0000037784.80885.1a
- Mannuzza, S., Klein, R. G., & Moulton, J. L. (2003). Does Stimulant Treatment Place Children at Risk for Adult Substance Abuse? A Controlled, Prospective Follow-Up

- Study. *Journal of Child and Adolescent Psychopharmacology*, 13(3), 273-282.
Doi: 10.1089/104454603322572606
- Mannuzza, S., Klein, R. G., & Moulton, J. L., 3rd. (2008). Lifetime Criminality among Boys with Attention Deficit Hyperactivity Disorder: A Prospective Follow-Up Study into Adulthood Using Official Arrest Records. *Psychiatry Research*, 160(3), 237-246.
Doi: 10.1016/j.psychres.2007.11.003
- Martel, M. M., Levinson, C. A., Lee, C. A., & Smith, T. E. (2017). Impulsivity Symptoms as Core to the Developmental Externalizing Spectrum. *Journal of Abnormal Child Psychology*, 45(1), 83–90. Doi: 10.1007/s10802-016-0148-6
- Martel, M., Nikolas, M., & Nigg, J. T. (2007). Executive Function in Adolescents with ADHD. *Journal of the American Academy of Child & Adolescent Psychiatry*, 46(11), 1437-1444. Doi:10.1097/chi.0b013e31814cf953
- Maruta, J., Spielman, L. A., Tseretopoulos, I. D., Hezghia, A., & Ghajar, J. (2014). Possible Medication-Resistant Deficits in Adult ADHD. *Journal of Attention Disorders*, 21(14), 1169-1179. Doi: 10.1177/1087054714538659
- McConaughy, S. H., Ivanova, M. Y., Antshel, K., & Eiraldi, R. B. (2009). Standardized Observational Assessment of Attention Deficit Hyperactivity Disorder Combined and Predominantly Inattentive Subtypes. I. Test Session Observations. *School Psychology Review*, 38(1), 45. Retrieved from:
<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2929017/>
- McLennan, J. D. (2016). Understanding Attention Deficit Hyperactivity Disorder as a Continuum. *Canadian Family Physician*, 62(12), 979. Retrieved from:
<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5154646/>
- Danielson, M. L., R. H., Ghandour, R. M., Holbrook, J. R., Kogan, M. D., & Blumberg, S. J. (2018) Prevalence of Parent-Reported ADHD Diagnosis and Associated Treatment among U.S. Children and Adolescents, 2016, *Journal of Clinical Child & Adolescent Psychology*, 47:2, 199-212, Doi: 10.1080/15374416.2017.1417860
- Merrill, R., Lyon, J., Baker, R., & Gren, L. (2009). Attention Deficit Hyperactivity Disorder and Increased Risk of Injury. *Advances in Medical Sciences*, 54(1), 20.
Doi: 10.2478/v10039-009-0022-7
- Millstein, R. B., Wilens, T. E., Biederman, J., & Spencer, T. J. (1997). Presenting ADHD Symptoms and Subtypes in Clinically Referred Adults with ADHD. *Journal of Attention Disorders*, 2(3), 159–166. Doi: 10.1177/108705479700200302

- Miranda, A., Colomer, C., Berenguer, C., Roselló, R., & Roselló, B. (2016). Substance Use in Young Adults with ADHD: Comorbidity and Symptoms of Inattention and Hyperactivity/Impulsivity. *International Journal of Clinical and Health Psychology, 16*(2), 157-165. Doi:10.1016/j.ijchp.2015.09.001
- Miyake A., Friedman N. P. (2012). The Nature and Organization of Individual Differences in Executive Functions: Four General Conclusions. *Current Directions in Psychological Science, 21*, 8-14. Doi: 10.1177/0963721411429458
- Moeller, F. G. & Dougherty, D. M. (2002). Impulsivity and Substance Abuse: What is the Connection? *Addictive Disorders & Their Treatment, 1*(1), 3-10. Retrieved from: <https://oce.ovid.com/article/00132576-200205000-00002/HTML>
- Moffitt, T. E., Arseneault, L., Belsky, D., Dickson, N., Hancox, R. J., Harrington, H., . . . Caspi, A. (2011). A Gradient of Childhood Self-Control Predicts Health, Wealth, and Public Safety. *Proceedings of the National Academy of Sciences of the United States of America, 108*(7), 2693-2698. Doi:10.1073/pnas.1010076108
- Moffitt, T. E., Houts, R., Asherson, P., Belsky, D. W., Corcoran, D. L., Hammerle, M., . . . Caspi, A. (2015). Is Adult ADHD a Childhood-Onset Neurodevelopmental Disorder? Evidence from a Four-Decade Longitudinal Cohort Study. *American Journal of Psychiatry, 172*(10), 967-977. Doi:10.1176/appi.ajp.2015.14101266
- Moffitt, T. E., & Scott, S. (2008). Conduct Disorders of Childhood and Adolescence. *Rutter's Child and Adolescent Psychiatry, 5*, 543564. Doi: 10.1002/9781444300895.ch35
- Mohr-Jensen, C., Bisgaard, C. M., Boldsen, S. K., & Steinhausen, H.-C. (2019). Attention Deficit/Hyperactivity Disorder in Childhood and Adolescence and the Risk of Crime in Young Adulthood in a Danish Nationwide Study. *Journal of the American Academy of Child & Adolescent Psychiatry*. Doi: 10.1016/j.jaac.2018.11.016
- Mohr-Jensen, C., & Steinhausen, H.C. (2016). A Meta-Analysis and Systematic Review of The Risks Associated with Childhood Attention-Deficit Hyperactivity Disorder on Long-Term Outcome of Arrests, Convictions, and Incarcerations. *Clinical Psychology Review, 48*, 32-42. Doi:10.1016/j.cpr.2016.05.002
- Molina, B. S. G., Hinshaw, S. P., Arnold, L. E., Swanson, J. M., Pelham, W. E., Hechtman, L., . . . Marcus, S. (Producer). (2013). Adolescent Substance Use in the Multimodal Treatment Study of Attention-Deficit/Hyperactivity Disorder (ADHD) (MTA) as a Function of Childhood ADHD, Random Assignment to Childhood Treatments, and Subsequent Medication. Doi: 10.1016/j.jaac.2012.12.014

- Molina, B. S. G., Howard, A. L., Swanson, J. M., Stehli, A., Mitchell, J. T., Kennedy, T. M., ...Hoza, B. (2018). Substance Use through Adolescence into Early Adulthood After Childhood-Diagnosed ADHD: Findings from the MTA Longitudinal Study. *Journal of Child Psychology and Psychiatry*, *59*(6), 692-702. Doi: 10.1111/jcpp.12855
- Molina, B. S. G., & Pelham, W. E. (2014). Attention-Deficit/Hyperactivity Disorder and Risk of Substance Use Disorder: Developmental Considerations, Potential Pathways, and Opportunities for Research. *Annual Review of Clinical Psychology*, *10*(1), 607-639. Doi: 10.1146/annurev-clinpsy-032813-153722
- Molina, B. S. G., & Pelham Jr, W. E. (2003). Childhood Predictors of Adolescent Substance Use in a Longitudinal Study of Children with ADHD. *Journal of Abnormal Psychology*, *112*(3), 497-507. Doi:10.1037/0021-843X.112.3.497
- Mordre, M., Groholt, B., Kjelsberg, E., Sandstad, B., & Myhre, A. M. (2011). The Impact of ADHD and Conduct Disorder in Childhood on Adult Delinquency: A 30 Years Follow-Up Study Using Official Crime Records. *BMC Psychiatry*, *11*(1), 57. Doi: 10.1186/1471-244X-11-57
- Moreno-Alcázar, A., Ramos-Quiroga, J. A., Radua, J., Salavert, J., Palomar, G., Bosch, R., . . . Pomarol-Clotet, E. (2016). Brain Abnormalities in Adults with Attention Deficit Hyperactivity Disorder Revealed by Voxel-Based Morphometry. *Psychiatry Research: Neuroimaging*, *254*, 41-47. Doi: 10.1016/j.pscychresns.2016.06.002
- Morgan, A. B., & Lilienfeld, S. O. (2000). A Meta-Analytic Review of the Relation between Antisocial Behavior and Neuropsychological Measures of Executive Function. *Clinical Psychology Review*, *20*(1), 113-136. Doi: 10.1016/S0272-7358(98)00096-8
- Murphy, K. R., Barkley, R. A., & Bush, T. (2002). Young Adults with Attention Deficit Hyperactivity Disorder: Subtype Differences in Comorbidity, Educational, and Clinical History. *The Journal of nervous and mental disease*, *190*(3), 147-157. Retrieved from: <https://oce.ovid.com/article/00005053-200203000-00003/HTML>
- Murray-Close, D., Hoza, B., Hinshaw, S., Arnold, L., Swanson, J., Jensen, P., . . . Wells, K. (2010). Developmental Processes in Peer Problems of Children with Attention Deficit/Hyperactivity Disorder in the Multimodal Treatment Study of Children With ADHD: Developmental Cascades and Vicious Cycles. *Development and Psychopathology*, *22*(4), 785-802. Doi: 10.1017/S0954579410000465
- Nagel, B. J., Bathula, D., Herting, M., Schmitt, C., Kroenke, C. D., Fair, D., & Nigg, J. T. (2011). Altered White Matter Microstructure in Children with Attention

- Deficit/Hyperactivity Disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 50(3), 283-292. Doi: 10.1016/j.jaac.2010.12.003
- Nigg, J. T. (2005). Neuropsychologic Theory and Findings in Attention-Deficit/Hyperactivity Disorder: The State of the Field and Salient Challenges for the Coming Decade. *Biological Psychiatry*, 57(11), 1424-1435. Doi: 10.1016/j.biopsych.2004.11.011
- Nigg, J. T., Tannock, R., & Rohde, L. A. (2010). What Is to Be the Fate of ADHD Subtypes? An Introduction to the Special Section on Research on the ADHD Subtypes and Implications for the DSM–V. *Journal of Clinical Child & Adolescent Psychology*, 39(6), 723-725. Doi: 10.1080/15374416.2010.517171
- Nigg, J. T., Wong, M. M., Martel, M. M., Jester, J. M., Puttler, L. I., Glass, J. M., . . . Zucker, R. A. (2006). Poor Response Inhibition as a Predictor of Problem Drinking and Illicit Drug Use in Adolescents at Risk for Alcoholism and Other Substance Use Disorders. *Journal of the American Academy of Child & Adolescent Psychiatry*, 45(4), 468-475. Doi: 10.1097/01.chi.0000199028.76452.a9
- Norsk Barne- og Ungdoms-psykiatriske Forening (2016) Veileder i BUP. [Norwegian Society of Child and Adolescent Psychiatry, Guidelines for Child and Adolescent Psychiatry] Retrieved 07.04.2019 from: <https://legeforeningen.no/Fagmed/Norsk-barne--og-ungdomspsykiatrisk-forening/Faglig-veileder-for-barne-og-ungdomspsykiatri/> [Norwegian]
- Nyhus, E., & Barceló, F. (2009). The Wisconsin Card Sorting Test and the Cognitive Assessment of Prefrontal Executive Functions: A Critical Update. *Brain and Cognition*, 71(3), 437-451. Doi: 10.1016/j.bandc.2009.03.005
- Ogilvie, J. M., Stewart, A. L., Chan, R. C. K., & Shum, D. H. K. (2011). Neuropsychological Measures of Executive Function and Antisocial Behavior: a Meta-Analysis. *Criminology*, 49(4), 1063-1107. Doi:10.1111/j.1745-9125.2011.00252.x
- Ohlmeier, M. D., Peters, K., Wildt, B. T. T., Zedler, M., Ziegenbein, M., Wiese, B., . . . Schneider, U. (2008). Comorbidity of Alcohol and Substance Dependence with Attention-Deficit/Hyperactivity Disorder (ADHD). *Alcohol and Alcoholism*, 43(3), 300-304. Doi:10.1093/alcalc/agn014
- Oltmanns, T. F., & Neale, J. M. (1975). Schizophrenic Performance when Distractors are Present: Attention Deficit or Differential Task Difficulty? *Journal of Abnormal Psychology*, 84, 205–209. Doi: 10.1037/h0076721
- Pardini, D. A., & Fite, P. J. (2010). Symptoms of Conduct Disorder, Oppositional Defiant

- Disorder, Attention-Deficit/Hyperactivity Disorder, and Callous-Unemotional Traits as Unique Predictors of Psychosocial Maladjustment in Boys: Advancing an Evidence Base for DSM-V. *Journal of the American Academy of Child & Adolescent Psychiatry*, 49(11), 1134-1144. Doi: 10.1016/j.jaac.2010.07.010
- Pfiffner, L. J., McBurnett, K., Rathouz, P. J., & Judice, S. (2005). Family Correlates of Oppositional and Conduct Disorders in Children with Attention Deficit/Hyperactivity Disorder. *Journal of Abnormal Child Psychology*, 33(5), 551–563. Doi: 10.1007/s10802-005-6737-4
- Philipp-Wiegmann, F., Rösler, M., Clasen, O., Zinnow, T., Retz-Junginger, P., & Retz, W. (2018). ADHD Modulates the Course of Delinquency: A 15-year Follow-Up Study of Young Incarcerated Men. *European Archives of Psychiatry and Clinical Neuroscience*, 268(4), 391-399. Doi: 10.1007/s00406-017-0816-8
- Polanczyk, G., de Lima, M. S., Horta, B. L., Biederman, J., & Rohde, L. A. (2007). The Worldwide Prevalence of ADHD: A Systematic Review and Meta-regression Analysis. *American Journal of Psychiatry*, 164(6), 942-948. Doi: 10.1176/ajp.2007.164.6.942
- Polanczyk, G. V., Salum, G. A., Sugaya, L. S., Caye, A., & Rohde, L. A. (2015). Annual Research Review: A meta-analysis of the worldwide Prevalence of Mental Disorders in Children and Adolescents. *Journal of Child Psychology and Psychiatry*, 56(3), 345-365. Doi: 10.1111/jcpp.12381
- Polanczyk, G. V., Willcutt, E. G., Salum, G. A., Kieling, C., & Rohde, L. A. (2014). ADHD Prevalence Estimates Across Three Decades: An Updated Systematic Review and Meta-Regression Analysis. *International Journal of Epidemiology*, 43(2), 434-442. Doi: 10.1093/ije/dyt261
- Pratt, T. C., Cullen, F. T., Blevins, K. R., Daigle, L., & Unnever, J. D. (2002). The Relationship of Attention Deficit Hyperactivity Disorder to Crime and Delinquency: A Meta-Analysis. *International Journal of Police Science & Management*, 4(4), 344–360. Doi:10.1350/ijps.4.4.344.10873
- Rasmussen, P., & Gillberg, C. (2000). Natural Outcome of ADHD with Developmental Coordination Disorder at age 22 Years: A Controlled, Longitudinal, Community-Based Study. *Journal of the American Academy of Child & Adolescent Psychiatry*, 39(11), 1424-1431. Doi: 10.1097/00004583-200011000-00017
- Reitan, R. M., & Wolfson, D. (1993). *The Halstead-Reitan Neuropsychological Test Battery: Theory and clinical interpretation* (2nd ed.). Tucson, AZ: Neuropsychology Press.

- Remschmidt, H. (2005). Global Consensus on ADHD/HKD. *European Child & Adolescent Psychiatry, 14*(3), 127-137. Doi: 10.1007/s00787-005-0439-x
- Reseptregisteret (2019) Antall brukere av ADHD-midler, 0-19 år. Oslo: Folkehelseinstituttet, 2017. [The Norwegian Prescription Database (2019) Number of Users of ADHD Medication, 0-19 Years of Age] Oslo, Norge: Folkehelseinstituttet/The Department of Public Health, 2017. Retrieved 31.03.2019, from: <http://www.reseptregisteret.no/>, 31.03.2019. [Norwegian]
- Richmond-Rakerd, L. S., Slutske, W. S., & Wood, P. K. (2017). Age of Initiation and Substance Use Progression: A Multivariate Latent Growth Analysis. *Psychology of Addictive Behaviors: Journal of the Society of Psychologists in Addictive Behaviors, 31*(6), 664–675. Doi: 10.1037/adb0000304
- Rixecker, H., & Hartje, W. (1980). Kimura's Recurring-Figures-Test: A Normative Study. *Journal of Clinical Psychology, 36*(2), 465–467. Doi: 10.1002/jclp.6120360213
- Rodgers, S., Müller, M., Rössler, W., Castela, E., Preisig, M., & Ajdacic-Gross, V. (2015). Externalizing Disorders and Substance Use: Empirically Derived Subtypes in a Population-Based Sample of Adults. *Social Psychiatry and Psychiatric Epidemiology, 50*(1), 7-17. Doi: 10.1007/s00127-014-0898-9
- Romine, C. B., Lee, D., Wolfe, M. E., Homack, S., George, C., & Riccio, C. A. (2004). Wisconsin Card Sorting Test with Children: A Meta-Analytic Study of Sensitivity and Specificity. *Archives of Clinical Neuropsychology, 19*(8), 1027-1041. Doi: 10.1016/j.acn.2003.12.009
- Rowland, A. S., Skipper, B. J., Umbach, D. M., Rabiner, D. L., Campbell, R. A., Naftel, A. J., & Sandler, D. P. (2015). The Prevalence of ADHD in a Population-Based Sample. *Journal of Attention Disorders, 19*(9), 741-754. Doi: 10.1177/1087054713513799
- Rund, B. R., Øie, M., & Sundet, K. (1996). Backward-Masking Deficit in Adolescents with Schizophrenic Disorders or Attention Deficit Hyperactivity Disorder. *The American Journal of Psychiatry, 153*(9), 1154-1157. Doi: 10.1176/ajp.153.9.1154.
- Rutter, M., Kim-Cohen, J., & Maughan, B. (2006). Continuities and Discontinuities in Psychopathology between Childhood and Adult Life. *Journal of Child Psychology and Psychiatry, 47*(3-4), 276-295. Doi: 10.1111/j.1469-7610.2006.01614.x
- Rutter, M., Moffitt, T. E., & Caspi, A. (2006). Gene-Environment Interplay and Psychopathology: Multiple Varieties but Real Effects. *Journal of Child Psychology and Psychiatry, 47*(3-4), 226-261. Doi: 10.1111/j.1469-7610.2005.01557.x

- Santosh, P. J., Taylor, E., Swanson, J., Wigal, T., Chuang, S., Davies, M., . . . Posner, M. (2005). Refining the Diagnoses of Inattention and Overactivity Syndromes: A Reanalysis of the Multimodal Treatment Study of Attention Deficit Hyperactivity Disorder (ADHD) Based on ICD-10 Criteria for Hyperkinetic Disorder. *Clinical Neuroscience Research*, 5(5), 307-314. Doi: 10.1016/j.cnr.2005.09.010
- Sales, J. M., Wasserman, G., Elkington, K. S., Lehman, W., Gardner, S., McReynolds, L., . . . Knudsen, H. (2018). Perceived Importance of Substance Use Prevention in Juvenile Justice: A Multi-Level Analysis. *Health & Justice*, 6(1), 12. Doi: 10.1186/s40352-018-0070-9
- Sandra Kooij, J. J., Marije Boonstra, A., Swinkels, S. H. N., Bekker, E. M., de Noord, I., & Buitelaar, J. K. (2008). Reliability, Validity, and Utility of Instruments for Self-Report and Informant Report Concerning Symptoms of ADHD in Adult Patients. *Journal of Attention Disorders*, 11(4), 445-458. Doi: 10.1177/1087054707299367
- Satterfield, J. H., Faller, K. J., Crinella, F. M., Schell, A. M., Swanson, J. M., & Homer, L. D. (2007). A 30-Year Prospective Follow-up Study of Hyperactive Boys with Conduct Problems: Adult Criminality. *Journal of the American Academy of Child & Adolescent Psychiatry*, 46(5), 601-610. Doi:10.1097/chi.0b013e318033ff59
- Sattler, J. M. & Dumont, R. (2004). *Assessment of Children: WISC-IV and WPPSI-III Supplement*. San Diego: Sattler Publisher.
- Saykin, A. J., Gur, R. C., Gur, R. E., Mozley, P. D., Mozley, L. H., Resnick, S. M., Kester, D. B., *et al.* (1991). Neuropsychological Function in Schizophrenia: Selective Impairment in Memory and Learning. *Archives of General Psychiatry*, 48, 618–624. Doi: 10.1001/archpsyc.1991.01810310036007
- Saykin, A. J., Shtasel, D. L., Gur, R. E., Kester, D. B., Mozley, L. H., Stafiniak, P., & Gur, R. C. (1994). Neuropsychological Deficits in Neuroleptic Naive Patients with First Episode Schizophrenia. *Archives of General Psychiatry*, 51, 124–131. Doi: 10.1001/archpsyc.1994.03950020048005
- Serra-Pinheiro, M. A., Coutinho, E. S. F., Souza, I. S., Pinna, C., Fortes, D., Araújo, C., . . . Mattos, P. (2012). Is ADHD a Risk Factor Independent of Conduct Disorder for Illicit Substance Use? A Meta-Analysis and Metaregression Investigation. *Journal of Attention Disorders*, 17(6), 459-469. Doi: 10.1177/1087054711435362
- Sibley, M. H., Pelham, W. E., Molina, B. S., Gnagy, E. M., Waschbusch, D. A., Biswas, A., . . . & Karch, K. M. (2011). The Delinquency Outcomes of Boys with ADHD with and

- without Comorbidity. *Journal of abnormal child psychology*, 39(1), 21-32.
Doi: 10.1007/s10802-010-9443-9
- Simon, V., Czobor, P., Bálint, S., Mészáros, A., & Bitter, I. (2009). Prevalence and Correlates of Adult Attention-Deficit Hyperactivity Disorder: Meta-Analysis. *British Journal of Psychiatry*, 194. Doi: 10.1192/bjp.bp.107.048827
- Sonuga-Barke, E., Bitsakou, P., & Thompson, M. (2010). Beyond the Dual Pathway Model: Evidence for the Dissociation of Timing, Inhibitory, and Delay-Related Impairments in Attention-Deficit/Hyperactivity Disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 49(4), 345-355. Doi: 10.1016/j.jaac.2009.12.018
- Sonuga-Barke, E. J. S., Taylor, E., Sembi, S., & Smith, J. (1992). Hyperactivity and Delay Aversion—I. The Effect of Delay on Choice. *Journal of Child Psychology and Psychiatry*, 33(2), 387-398. Doi: 10.1111/j.1469-7610.1992.tb00874.x
- Statistisk Sentralbyrå. (2018). Dette er Norge 2018. [Statistics Norway. (2018). This is Norway 2018]. Retrieved from: <https://www.ssb.no/befolkning/artikler-og-publikasjoner/dette-er-norge-2018>. [Norwegian]
- Stavro, K., Pelletier, J., & Potvin, S. (2013). Widespread and Sustained Cognitive Deficits in Alcoholism: A Meta-Analysis. *Addiction Biology*, 18(2), 203-213.
Doi: 10.1111/j.1369-1600.2011.00418.x
- Stedron, J. M., Sahni, S. D., & Munakata, Y. (2005). Common Mechanisms for Working Memory and Attention: The Case of Perseveration with Visible Solutions. *Journal of Cognitive Neuroscience*, 17(4), 623-631. Doi: 10.1162/0898929053467622
- Surén, P., Thorstensen, A. G., Tørstad, M., Emhjellen, P.E., Furu, K., Biele, G., ... Reichborn-Kjennerud, T. (2018) Diagnostikk av hyperkinetisk forstyrrelse hos barn i Norge. [Diagnostics of Hyperkinetic Disorder in Children in Norway.] *Tidsskrift for den Norske Legerforening* 2018. Doi: 10.4045/tidsskr.18.0418 [Norwegian]
- Swanson, J. M., Arnold, L. E., Molina, B. S. G., Sibley, M. H., Hechtman, L. T., Hinshaw, S. P., . . . the, M. T. A. C. G. (2017). Young Adult Outcomes in the Follow-Up of the Multimodal Treatment Study of Attention-Deficit/Hyperactivity Disorder: Symptom Persistence, Source Discrepancy, and Height Suppression. *Journal of Child Psychology and Psychiatry*, 58(6), 663-678. Doi: 10.1111/jcpp.12684
- Szobot, C. M., & Bukstein, O. (2008). Attention Deficit Hyperactivity Disorder and Substance Use Disorders. *Child and Adolescent Psychiatric Clinics of North America*, 17(2), 309-323. Doi:10.1016/j.chc.2007.11.003

- Tarver, J., Daley, D., & Sayal, K. (2014). Attention-Deficit Hyperactivity Disorder (ADHD): An Updated Review of the Essential Facts. *Child: Care, Health and Development*, 40(6), 762-774. Doi: 10.1111/cch.12139
- Taylor, E., Döpfner, M., Sergeant, J., Asherson, P., Banaschewski, T., Buitelaar, J., ... & Steinhausen, H. C. (2004). European Clinical Guidelines for Hyperkinetic Disorder First Upgrade. *European Child & Adolescent Psychiatry*, 13(1), i7-i30. Doi: 10.1007/s00787-004-1002-x
- Thaler, N. S., Bello, D. T., & Etcoff, L. M. (2013). WISC-IV Profiles are Associated with Differences in Symptomatology and Outcome in Children with ADHD. *Journal of Attention Disorders*, 17(4), 291-301. Doi: 10.1177/1087054711428806
- Thapar, A., Cooper, M., Eyre, O., & Langley, K. (2013). Practitioner Review: What Have We Learnt About The Causes of ADHD?. *Journal of Child Psychology and Psychiatry*, 54(1), 3-16. Doi: 10.1111/j.1469-7610.2012.02611.x
- Thapar, A., Cooper, M., & Rutter, M. (2017). Neurodevelopmental Disorders. *The Lancet Psychiatry*, 4(4), 339-346. Doi: 10.1016/S2215-0366(16)30376-5
- Thornberry, T. P., & Krohn, M. D. (2000). The Self-Report Method for Measuring Delinquency and Crime. *Criminal justice*, 4(1), 33-83. Retrieved from: https://www.ncjrs.gov/criminal_justice2000/vol_4/04b.pdf
- Tombaugh, T. N. (2004). Trail Making Test A and B: Normative Data Stratified by Age and Education. *Archives of Clinical Neuropsychology*, 19(2), 203-214. Doi: 10.1016/S0887-6177(03)00039-8
- Uchida, M., Spencer, T. J., Faraone, S. V., & Biederman, J. (2018). Adult Outcome of ADHD: An Overview of Results from the MGH Longitudinal Family Studies of Pediatrically and Psychiatrically Referred Youth With and Without ADHD of Both Sexes. *Journal of Attention Disorders*, 22(6), 523-534. Doi: 10.1177/1087054715604360
- van Emmerik-van Oortmerssen, K., Crunelle, CL., Carpentier, PJ. (2013). Substance Use Disorders and ADHD: An Overview of Recent Dutch Research. *Tijdschrift voor Psychiatrie* 2013;55:861–6.
- van Emmerik-van Oortmerssen, K., van de Glind, G., Koeter, M. W. J., Allsop, S., Auriacombe, M., Barta, C., . . . Schoevers, R. A. (2014). Psychiatric Comorbidity in Treatment-Seeking Substance Use Disorder Patients with and without Attention Deficit Hyperactivity Disorder: Results of the IASP Study. *Addiction*, 109(2),

- 262-272. Doi:10.1111/add.12370
- van Emmerik-van Oortmerssen, K., van de Glind, G., van den Brink, W., Smit, F., Crunelle, C. L., Swets, M., & Schoevers, R. A. (2012). Prevalence of Attention-Deficit Hyperactivity Disorder in Substance Use Disorder Patients: A Meta-Analysis and Meta-Regression Analysis. *Drug and Alcohol Dependence*, 122(1), 11-19.
Doi: 10.1016/j.drugalcdep.2011.12.007
- van Lieshout, M., Luman, M., Buitelaar, J., Rommelse, N. N. J., & Oosterlaan, J. (2013). Does Neurocognitive Functioning Predict Future or Persistence of ADHD? A Systematic Review. *Clinical Psychology Review*, 33(4), 539-560.
Doi: 10.1016/j.cpr.2013.02.003
- Villodas, M.T., Pfiffner, L. J., & McBurnett, K. (2012). Prevention of Serious Conduct Problems in Youth with Attention Deficit/Hyperactivity Disorder. *Expert Review of Neurotherapeutics*, 12(10), 1253-1263. Doi: 10.1586/ern.12.119
- Vingilis, E., Erickson, P. G., Toplak, M. E., Kolla, N. J., Mann, R. E., Seeley, J., . . . Daigle, D. S. (2015). Attention Deficit Hyperactivity Disorder Symptoms, Comorbidities, Substance Use, and Social Outcomes among Men and Women in a Canadian Sample. *Journal of BioMed Research International*. 2015(9), 1-8. Doi: 10.1155/2015/982072
- Vogt, H., & Lunde, C. (2018). AD/HD-medisinering – svakt vitenskapelig grunnlag. [AD/HD-Medication - Weak Scientific Foundation]. *Tidsskrift for den Norske Legeforening 2018*. Doi: 10.4045/tidsskr.17.0917 [Norwegian]
- Volkow, N. D., Wang, G. J., Ma, Y., Fowler, J. S., Zhu, W., Maynard, L., . . . Swanson, J. M. (2003). Expectation Enhances the Regional Brain Metabolic and the Reinforcing Effects of Stimulants in Cocaine Abusers. *Journal of Neuroscience*, 23(36), 11461-11468. Retrieved from: <http://www.jneurosci.org/content/23/36/11461>
- von Polier, G. G., Vloet, T. D., & Herpertz-Dahlmann, B. (2012). ADHD and Delinquency – a Developmental Perspective. *Behavioral Sciences & the Law*, 30(2), 121-139.
Doi:10.1002/bsl.2005
- Weafer, J., Fillmore, M. T., & Milich, R. (2009). Increased Sensitivity to the Disinhibiting Effects of Alcohol in Adults with ADHD. *Experimental and Clinical Psychopharmacology*, 17(2), 113-121. Doi: 10.1037/a0015418
- Wechsler, D. (1974). *WISC-R Manual: Wechsler Intelligence Scale for Children - Revised*. New York: Psychological Corporation.
- Weissenberger, S., Ptacek, R., Klicperova-Baker, M., Erman, A., Schonova, K., Raboch, J., &

- Goetz, M. (2017). ADHD, Lifestyles and Comorbidities: A Call for a Holistic Perspective – from Medical to Societal Intervening Factors. *Frontiers in Psychology*, 8, 454. Doi: 10.3389/fpsyg.2017.00454
- Wender, P.H., Wood, D.R., Reimherr, F.W. (1985). Pharmacological Treatment of Attention Deficit Disorder, Residual Type (ADD, RT, “Minimal Brain Dysfunction”, “Hyperactivity”) in Adults. *Psychopharmacol Bull.* 1985;21: 222–231. Retrieved from: <https://www.ncbi.nlm.nih.gov/pubmed/3923527>
- Wilens, T. E., Martelon, M., Fried, R., Petty, C., Bateman, C., & Biederman, J. (2011). Do Executive Function Deficits Predict Later Substance Use Disorders among Adolescents and Young Adults? *Journal of the American Academy of Child and Adolescent Psychiatry*, 50(2), 141-149. Doi:10.1016/j.jaac.2010.11.010
- Willcutt, E. G., Doyle, A. E., Nigg, J. T., Faraone, S. V., & Pennington, B. F. (2005). Validity of the Executive Function Theory of Attention-Deficit/Hyperactivity Disorder: A Meta-Analytic Review. *Biological Psychiatry*, 57(11), 1336-1346. Doi: 10.1016/j.biopsych.2005.02.006
- Willcutt, E. G., Nigg, J. T., Pennington, B. F., Solanto, M. V., Rohde, L. A., Tannock, R., . . . Lahey, B. B. (2012). Validity of DSM-IV Attention Deficit/Hyperactivity Disorder Symptom Dimensions and Subtypes. *Journal of Abnormal Psychology*, 121(4), 991-1010. Doi: 10.1037/a0027347
- Willcutt, E. G., Betjemann, R. S., McGrath, L. M., Chhabildas, N. A., Olson, R. K., DeFries, J. C., & Pennington, B. F. (2010). Etiology and Neuropsychology of Comorbidity between RD and ADHD: The Case for Multiple-Deficit Models. *Cortex* 46(10): 1345-1361. Doi: 10.1016/j.cortex.2010.06.009
- World Health Organization. (1992). *The ICD-10 Classification of Mental and Behavioural Disorders: Clinical Descriptions and Diagnostic Guidelines*. Geneva: World Health Organization.
- Wåhlstedt, C., Thorell, L. B., & Bohlin, G. (2009). Heterogeneity in ADHD: Neuropsychological Pathways, Comorbidity and Symptom Domains. *Journal of Abnormal Child Psychology*, 37(4), 551-564. Doi: 10.1007/s10802-008-9286-9.
- Yi, A. (2011). California Verbal Learning Test (California Verbal Learning Test-II). In J. S. Kreutzer, J. DeLuca, & B. Caplan (Eds.), *Encyclopedia of Clinical Neuropsychology* (pp. 475-476). New York, NY: Springer New York. Doi: 10.1007/978-0-387-79948-3_1112

- Young, S. E., Friedman, N. P., Miyake, A., Willcutt, E. G., Corley, R. P., Haberstick, B. C., & Hewitt, J. K. (2009). Behavioral Disinhibition: Liability for Externalizing Spectrum Disorders and Its Genetic and Environmental Relation to Response Inhibition across Adolescence. *Journal of Abnormal Psychology, 118*(1), 117-130.
Doi: 10.1037/a0014657
- Young, S., & Sedgwick, O. (2015). Attention Deficit Hyperactivity Disorder and Substance Misuse: An Evaluation of Causal Hypotheses and Treatment Considerations. *Expert Review of Neurotherapeutics, 15*(9), 1005-1014.
Doi:10.1586/14737175.2015.1059756
- Øie, M.; Sundet, K. S., & Rund, B. R. (2010). Neurocognitive Decline in Early-Onset Schizophrenia Compared With ADHD and Normal Controls: Evidence from a 13 Year Follow-up Study. *Schizophrenia Bulletin*. ISSN 0586-7614. 36(3), s 557- 565 .
Doi: 10.1093/schbul/sbn127
- Øie, M., & Rund, B.R. (1999) Neuropsychological Deficits in Adolescent-Onset Schizophrenia Compared with Attention Deficit Hyperactivity Disorder. *American Journal of Psychiatry, 156*, 1216-1222. Doi: 10.1176/ajp.156.8.1216
- Øie, M., Sundet, K., & Ueland, T. (2011) Neurocognition and Functional Outcome in Early Onset Schizophrenia and Attention-Deficit/Hyperactivity Disorder: A 13-Year Follow-Up. *Neuropsychology, 25*(1), 25-35. Doi: 10.1037/a0020855
- Ørstadvik, R., Gustavson, K., Rohrer-Baumgartner, N., Biele, G., Furu, K., Karlstad, Ø.,... & Aase, H., (2016) *ADHD i Norge. En statusrapport* (Folkehelse rapport 2016:4) [ADHD in Norway. A status report.] Oslo, Folkehelseinstituttet/The Department Of Public Health. Retrieved from:
https://www.fhi.no/globalassets/dokumenterfiler/rapporter/2017/adhd_i_norge.pdf
[Norwegian]