

The role of neuropsychological profiles in adult ADHD and ischemic stroke:
Implications for clinical practice

A cumulative dissertation

Doctorate in Psychology (Dr. rer. nat.)

Submitted to the University of Bremen, Faculty 11

by

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Bremen, October 2014

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Acknowledgements

First and foremost, I wish to thank the head of the Centre of Clinical Psychology and Rehabilitation and my advisor Professor Franz Petermann. He gave me the opportunity to accomplish the present thesis and supported this research with his knowledge, painstaking effort in proof reading the drafts, and patience.

I would particularly like to thank Monika Daseking, who mainly supervised the current research. I am thankful for your advice and insight throughout my work, for the non-stop availability for help with questions and thoughts. Thank you for the positive vibes and for creating a relaxed yet productive atmosphere in the weekly PhD College meetings.

I have been blessed with a friendly and cheerful group of fellow PhD students. It has been a great pleasure to work with Franziska Korsch and Walter, Franz Pauls, Angelika Kullik, Lina Werpup, Hanna Weber, Nicole Gust, and Esmahan Belhadj. As this work can sometimes be a bit lonely, I thank you all for the lunch- and coffee breaks, for the in-depth and fruitful discussions on various topical issues, for many laughs and moments together that enrich every day anew.

Special acknowledgements to Philipp Seegers for your continuous love, your patience, understanding, and the unconditional and unselfish support for the billions of things I have wished for. Thank you!

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Abstract

By quantifying behavioural outcomes that are related to cognitive functioning, neuropsychological assessments allow to predict the manifestations of intellectual ability. Thereby, they take an important role in the clinical management of patients with mental illnesses and neurological conditions. The literature increasingly discusses the role of neuropsychological assessments for diagnosis and treatment in adults with ADHD or a stroke. However, inconsistent neuropsychological profiles in both groups lead to a lack of clarity regarding the role of neuropsychological assessments in clinical care. The aim of the present thesis was to assess intellectual functions and cognitive profiles by means of the Wechsler Adult Intelligence Scale-Fourth Edition (WAIS-IV) in adults with ADHD and in patients with a first-ever unilateral, ischemic stroke in inpatient rehabilitation facilities in comparison to a control group. The purpose was to determine whether cognitive impairments vary as a function of clinical characteristics, including comorbidity and medication in adults with ADHD (study I) and language impairment, neglect, hemianopsia, and post-stroke depression in patients with a stroke (study II). In addition, the present thesis evaluated the contribution of self-reports for predicting cognitive functioning in adults with ADHD (study III). The results of the studies on adults with ADHD revealed deficits in intellectual and cognitive functions when compared to a randomly matched control group. Particularly, subtests of verbal working memory and processing speed indicated impairment. Therefore, the General Ability Index provided a more reliable alternative to the Full-Scale IQ. Lower performances compared to the controls appeared to be robust with respect to comorbidity and medication (study I). Moreover, no relationship between self-reported symptoms and cognitive performance could be found (study III). With respect to patients with a stroke, it could be demonstrated that both right hemisphere stroke (RHS) and left hemisphere stroke (LHS) patients are likely to have significant impairments in all domains of cognitive functions compared to a randomly matched control group. RHS patients were particularly deficient in measures requiring visuo-spatial abilities and LHS patients in those with language and processing speed demands. Language impairments following LHS had a negative impact on subtests requiring verbal skills. Neglect and hemianopsia reduced some error variance on working memory, processing speed, and language subtests. A PSD turned out to decrease performance on subtests with visuo-spatial and processing speed requirements (study II). Based on the results, the clinical utility of the WAIS-IV and the general role of neuropsychological assessments in both groups are discussed.

Zusammenfassung

Störungsspezifische Verhaltensauffälligkeiten, die im Zusammenhang mit Beeinträchtigungen bestimmter kognitiver Funktionen auftreten, erlauben es neuropsychologischen Testverfahren intellektuelle Fähigkeiten zu prognostizieren. Dadurch nehmen sie eine wichtige Rolle in der Planung und Umsetzung der Behandlungen von Patienten mit psychischen und neurologischen Erkrankungen ein. Neuropsychologische Verfahren werden in der Literatur vermehrt im Zusammenhang mit Diagnosestellung und Behandlung bei Erwachsenen mit einer ADHS und bei Patienten mit einem Schlaganfall diskutiert. Inkonsistente neuropsychologische Profile in diesen Gruppen führen jedoch zu Unklarheiten bei der klinischen Anwendung von neuropsychologischen Verfahren. Das Ziel der vorliegenden kumulativen Dissertation bestand darin, die intellektuellen Fähigkeiten und kognitiven Leistungsprofile von ADHS Erwachsenen und Rehabilitanden mit einem unilateralen, ischämischen Schlaganfall auf Basis der Wechsler Adult Intelligence Scale - Vierte Auflage (WAIS-IV) mit einer Kontrollgruppe zu vergleichen. Weiterhin bestand die Absicht zu bestimmen, ob ein funktionaler Zusammenhang zwischen kognitiven Defiziten und klinischen Charakteristika, Komorbidität und Medikation bei ADHS Erwachsenen (vgl. Studie I) und kognitiven Defiziten und Sprachdefizite, Neglekt, Gesichtsfeldeinschränkung und Post-Stroke Depressionen bei Patienten mit einem Schlaganfall, besteht (vgl. Studie II). Um die Bedeutung der Selbsteinschätzung bei ADHS Erwachsenen näher zu ermitteln, wurden ferner die Zusammenhänge zwischen der kognitiven Leistungsfähigkeit und dem subjektiv eingeschätzten Schweregrad der ADHS-Symptomatik untersucht (vgl. Studie III). Die Ergebnisse der Studien zu ADHS im Erwachsenenalter zeigten unterdurchschnittliche intellektuelle und kognitive Fähigkeiten im Vergleich zur zufällig gematchten Kontrollgruppe besonders im Arbeitsgedächtnis und in der Verarbeitungsgeschwindigkeit. Daher konnte der Allgemeine Fähigkeitsindex als validere Alternative zum Gesamt-IQ herangezogen werden (vgl. Studie I). Eine weitere Studie zu ADHS im Erwachsenenalter stellte insgesamt nur geringe Zusammenhänge zwischen der Selbstbeurteilung und dem Fähigkeitsprofil in der WAIS-IV fest (vgl. Studie III). In der Studie zur intellektuellen und kognitiven Leistungsfähigkeit bei links- (LHS) und rechts-hemisphärischen (RHS) Schlaganfallpatienten wiesen die Ergebnisse bedeutsame Beeinträchtigungen im Vergleich zu einer zufällig gematchten Kontrollgruppe auf. Hohe Effektstärken in den Untertests ergaben sich bei RHS Patienten mit Anforderungen an räumlich-visuelle Leistungen und bei LHS Patienten mit Sprach- und Gedächtnisanforderungen. Spracheinschränkungen nach einem LHS hatten einen negativen

Einfluss auf Untertests mit sprachlichen Anforderungen. Neglekt und Gesichtsfeldeinschränkungen reduzierten die Fehlervarianz mit Bezug auf die Untertests des Arbeitsgedächtnisses, der Verarbeitungsgeschwindigkeit und der Sprache (vgl. Studie II). Es zeigte sich, dass eine Post-Stroke Depression die Leistung in den Untertests mit räumlich-visuellen Anforderungen und zu der Verarbeitungsgeschwindigkeit einschränkt. Basierend auf den Ergebnissen wurde die klinische Nutzbarkeit der WAIS-IV und die generelle Rolle von neuropsychologischen Verfahren in der Diagnostik und der Therapieplanung in den beiden Gruppen diskutiert.

Abbreviations

ADHD	Attention Deficit Hyperactivity Disorder
ANOVA	one-way analysis of variance
AR	Arithmetic
BD	Block Design
CA	Cancellation
CHC	Cattell-Horn-Carroll theory
CD	Coding
DS	Digit Span
DSM-V	Diagnostic and Statistical Manual of Mental Disorders - Fifth Edition
DSM-IV-TR	Diagnostic and Statistical Manual of Mental Disorders - Fourth Edition, Text Revision
FSIQ	Full Scale Intelligence Quotient
<i>g</i>	general intelligence
GAI	General Ability Index
Gc	Crystallised intelligence
Gf	Fluid intelligence
Gq	Quantitative Knowledge
Gs	Processing Speed
Gsm	Short term memory
Gv	Visual Processing
ICD-10	International Classification of Diseases, Tenth Edition
IN	Information
LHS	left hemisphere stroke
LNS	Letter-Number Sequencing
MANOVA	two-way analysis of variance
MANCOVA	two-way analysis of covariance
MCA	Middle Cerebral Artery
MR	Matrix Reasoning
PC	Picture Completion
PIQ	Performance Intelligence Quotient
PSD	Post Stroke Depression
RHS	right hemisphere stroke
SI	Similarities
SS	Symbol Search
VC	Vocabulary
VIQ	Verbal Intelligence Quotient
VP	Visual Puzzles
WAIS	Wechsler Adult Intelligence Scale
WAIS-IV	Wechsler Adult Intelligence Scale - Fourth Edition

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Introduction

David Wechsler (1975, p.139)

“What we measure with tests is not what tests measure – not information, not spatial perception, not reasoning ability. These are only a means to an end. What intelligence tests measure is something much more important: the capacity of an individual to understand the world about him and his resourcefulness to cope”.

Clinical neuropsychology, a field that integrates clinical neurosciences and psychology, is concerned with the relationship between brain abnormalities and behavioural manifestations. Neuropsychological assessments have long served the purpose to diagnose such abnormalities. However, their importance in this role has diminished greatly over the past years. Nowadays, they contribute to the clinical management of patients with brain damage and mental illnesses by predicting the manifestations of behavioural outcomes through the quantification of strengths and weaknesses in knowledge and skills (Lezak, Howieson & Loring, 2004). With increasing research on the identification of these abilities, psychological pioneers brought about an elusive, overarching construct to characterize an individual’s overall mental capability: intelligence (e.g. Spearman, 1904). Following them, many techniques to measure intelligence emerged offering a chance to grasp the diversity and nature of human mental abilities in neuropsychology. These measurements form an essential component in neuropsychological assessments, because they evaluate the cognitive consequences of brain damage and mental illness (Lezak et al., 2004). Today, the gold standard of intelligence testing are the Wechsler Adult Intelligence Scales (Hartman, 2009). Already early in the literature, the scales have been described as the “workhorse of neuropsychological assessment” and “the single most utilised component of the neuropsychological repertory” (Lezak, 1988, p. 53). In its newest edition, the Wechsler Adult Intelligence Scale-IV (WAIS-IV; Wechsler, 2008) measures a large variety of cognitive abilities and provides an option to value one important function of clinical neuropsychology: attention. According to the Cattell-Horn-Carroll theory of cognitive abilities (CHC; McGrew & Flanagan, 1998; Schneider & McGrew, 2012), attention is well

grounded in the construct of intelligence. Therefore, the understanding of attention deficits and those of other cognitive abilities can be conceived with the implementation of the WAIS-IV. Such information can be employed for diagnostic purposes, functional potential estimates, and treatment options for patients with brain damage and mental illnesses.

The applications of neuropsychological assessments are especially important in conditions with manifested deficits in attention. These include neurological disorders as a result of brain damage, such as stroke and those caused by neuro-developmental dysfunctions, such as Attention-Deficit/Hyperactivity Disorder (ADHD). Next to central deficits in attention, adults with ADHD or those affected by a stroke suffer from a variety of substantial defects in cognitive aptitudes, which negatively affect their adaptability, daily functioning and, in turn, their quality of life (Barker-Collo, Feigin, Parag, Lawes & Senior, 2010; Kooij et al., 2010; Zinn et al., 2004). Due to the adverse impact on attention, cognitive functions, and consequent functional outcomes, neuropsychological testing may serve specific purposes for these groups. By identifying cognitive deficits and the progress of cognitive functioning, neuropsychological testing has been linked to the diagnostic process, treatment planning, and the prediction of functional potential for adults with ADHD (see Hervey, Epstein & Curry, 2004; Seidman, 2006, for reviews) as well as to the rehabilitation success of patients with a stroke (Barker-Collo, Feigin, Parag, et al., 2010; Heruti et al., 2002; Nys, van Zandvoort, de Kort, van der Worp, et al., 2005; Zinn et al., 2004). In both disorders, neuropsychological functioning deserves special attention.

Until today, ADHD in adulthood is so contentious that many clinicians have a limited understanding about its diagnosis, assessment, and treatment (Ramsay, 2010). In order to improve these processes the importance of identifying neuropsychological dysfunctions has increasingly been stressed. This is a relatively new approach in adult ADHD and of growing interest in scientific research. In clinical practice, however, the value of neuropsychological assessments is not fully recognised (Surman, 2013). A reason for this may be that no specific pattern of cognitive dysfunctions of adult ADHD has been identified yet (see Boonstra, Oosterlaan, Sergeant & Buitelaar, 2005; Bridgett & Walker, 2006; Schoechlin & Engel, 2005; Seidman, 2006; Woods, Lovejoy & Ball, 2002, for reviews). Despite the heterogeneous research findings, neuropsychological assessments might still be beneficial to the diagnostic and treatment process for ADHD in adulthood. The advantage is that it is possible to more narrowly define the behaviour of interest in a controlled setting in order to understand the pathophysiology of the adult better. Since the diagnostic process of adult ADHD is centrally based on information from self-reports, whose reliability

have often been questioned (Kooij et al., 2008; McCann & Roy-Byrne, 2004), this approach may offer more objective corroborative information beyond that of self-evaluation. Therefore, the main purpose of integrating objective psychometric measures into the diagnostic process is to clarify the underlying mechanisms for functional challenges and barriers to quality of life, which can also be tackled during therapy (Surman, 2013).

In patients with a stroke, an anatomical diagnosis is achieved through diverse imaging techniques. These devices, however, cannot determine the nature and severity of behavioural stroke sequelae (Lezak et al., 2004). Therefore, neuropsychological assessments represent an essential determinant for the manifestations of such deficits, particularly at the point of rehabilitation. In rehabilitation, the assessment of the cognitive status is considered indispensable because it evaluates the potential to actively take part in therapy. This method may improve evidence-based-clinical decision making and rehabilitation success may increase. Despite these advantages, comprehensive neuropsychological assessments in rehabilitation settings have not been standardised and are not accomplished routinely (Barker-Collo & Feigin, 2006; Bland et al., 2013; Heruti et al., 2002). On the contrary, the scientific community drew much attention to neuropsychological post-stroke impairment. Until today, however, there is no clear picture of neuropsychological profiles for different stroke types and at different points in the course of recovery, especially because of the heterogeneity of stroke in general and the linkage between neuropsychological performance and stroke characteristics as well as clinical symptoms (Barker-Collo, Feigin, Parag, et al., 2010; Leśniak, Bak, Czepiel, Seniów & Członkowska, 2008; Planton et al., 2012; Wilde, 2010). More consistent research findings concerning neuropsychological functioning are needed for a more standardised application in clinical practice.

For both, adults with ADHD or stroke, this leads us to realisation that the role of neuropsychological assessments is important. While they have certain universal uniformities in both groups to detect deficits and to evaluate the progressing of functioning, they serve different purposes as to contemplate diagnoses for adults with ADHD and to plan rehabilitation intervention for patients with a stroke. Accordingly, they alleviate the symptoms of the conditions. Moreover, the implementation of neuropsychological assessments for these purposes cannot be fully realised yet in clinical practice since the cognitive profiles that characterize both adults with ADHD or stroke are still diffuse. Further research, using psychometrically sound instruments that precisely define the cognitive patterns in each disorder, is indispensable and could offer more information on critical

issues in diagnostic and therapeutic approaches. The current investigation aims to fill this gap with the following methodology:

The objective approach

- The examination of WAIS-IV cognitive profiles in adults with ADHD
- The examination of WAIS-IV cognitive profiles in patients with a stroke
- The influence of confounding variables in WAIS-IV assessment

The subjective approach

- The status of self-evaluation in the assessment of ADHD in adulthood

By providing information on these approaches, the present thesis evaluates to what extent the WAIS-IV is sensitive to cognitive and intellectual impairments in adults with ADHD or stroke. It offers insights on the benefits and drawbacks of the full and in-depth battery of the WAIS-IV in psychiatric and neurological rehabilitation settings, with regards to diagnosis and clinical care. Furthermore, since information from self-reports have often been questioned, the role of self-evaluation in adults with ADHD is addressed and put into context with the value of psychometric assessment for diagnosis. For implementation purposes, the thesis was embedded within a larger research project involving the German standardization and adaption of the WAIS-IV. In the following paragraphs further details on the development of the current thesis and its underlying project are provided.

The originating project of the present thesis

The present thesis, within the fields of clinical psychology and neuropsychology, was accomplished between 2011 and 2014 at the Centre of Clinical Psychology and Rehabilitation, University of Bremen. The research compared the intellectual and cognitive functioning in adults with ADHD and in first-ever unilateral, ischemic stroke patient in inpatient rehabilitation facilities to a control group. The emphasis of the present research was the development of comprehensive cognitive profiles with the WAIS-IV. The empirical studies relevant for the thesis (see Appendix B to D) originated during the German standardization and adaption of the WAIS-IV and its norm development at the Centre of Clinical Psychology and Rehabilitation, University of Bremen. Between February 2012 and August 2012, normative data was obtained from a stratified sample of 1,454 examinees aged between 16:0 and 90:11 years. Within 13 age groups, the basis for stratification included sex, education, and geographical regions to ensure that the normative sample consti-

tuted a representative proportion of individuals living in Germany. A random match of the normative sample served as the control sample in the studies.

The present thesis focused on a cohort of adult patients with a diagnosis of ADHD and on a sample of elderly patients with a first-ever unilateral, ischemic stroke in inpatient rehabilitation facilities. The eligible participants were invited for assessment and informed about the present research. Those who voluntarily agreed to participate gave their written informed consent prior to assessment. In total, 124 adults with ADHD and 112 stroke patients were recruited and assessed between March 2012 and May 2013. Adults with ADHD were recruited from the 'Ameos Dr Heines' outpatient psychiatric clinic in Bremen and via press releases and public advertisements. The assessment took place within the premises of the Centre of Clinical Psychology and Rehabilitation as well as in the outpatient clinic in Bremen. The patients with a stroke were recruited through two inpatient rehabilitation facilities: the 'Neurozentrum Niedersachsen' in Bad Essen and the 'Rehabilitation Zentrum' in Wilhelmshaven and their evaluation took place in their premises. The data served as the basis for three empirical publications, which have been published in peer-reviewed national and international scientific journals outlined below.

Publications

Study I (see Appendix B):

Theiling, J., & Petermann, F. (2014). Neuropsychological Profiles on the WAIS-IV of ADHD adults. *Journal of Attention Disorders*. Retrieved October 1, 2014, from <http://jad.sagepub.com/content/early/2014/01/21/1087054713518241.abstract>

Study II (see Appendix C):

Theiling, J., Petermann, F., & Daseking, M. (2013). WAIS-IV profiles in first-ever unilateral ischemic stroke patients. *Zeitschrift für Neuropsychologie*, *24*, 239-252.

Study III (see Appendix D):

Theiling, J., Petermann, F., & Daseking, M. (2013). Zusammenhang zwischen selbsteingeschätzter ADHS-Symptomatik und der Leistungsfähigkeit in der WAIS-IV. *Gesundheitswesen*, *75*, 768-744.

Thesis outline

The current thesis can be structured into three parts, starting with a theoretical chapter to provide a foundation for the investigation (section 1 and 2). Section 1 covers the diversity of attention and its role in cognitive functions and intelligence. To this end, the constructs of attention,

cognitive function, and intelligence are more thoroughly defined and put into context with the Cattell-Horn-Carroll theory. Section 2 includes an overview of those two disorders which form the basis of this research: ADHD in adulthood and unilateral, ischemic stroke. This section defines each of these conditions based on information of diagnosis, aetiology or pathogenesis, and clinical symptoms. Separately for each disorder, a selective overview of current neuropsychological research is provided and the assessment process is portrayed. Based on the literature review provided in these sections, section 3 presents the present empirical investigation by outlining the rationale and aims of the studies on adults with ADHD or stroke.

The second part moves on to the methods of the current research (section 4 and 5). Section 4 provides an overview of the target populations, sample compositions, and the control group. Subsequently, the assessment tools used, the data management, and the use of statistical analyses are outlined. In section 5, the results of this research are summarised and supplementary statistics are portrayed.

The final part discusses the results of the current research and offers advice to clinical care to consider the advantages and limitations of the WAIS-IV for assessment and treatment of ADHD in adults and stroke (section 6 and 7). Section 6 distils the clinical implications of the results for diagnostic and therapeutic purposes. For both conditions, the general role of neuropsychological assessments to the diagnostic process and therapeutic intervention is addressed and critically discussed. General limitations to the research methods and design are outlined last. Section 7 includes a main conclusion and constitutes the closing part for his work. The appendices comprise supplementary material on the WAIS-IV, print-outs of the published studies, a state of the author's contribution to the publications, and the declaration of originality.

Theoretical Foundation

1 Towards a conceptual framework

Cognition is a component of human behaviour that contributes to the comprehension and acquisition of new knowledge by relating existing information, perception, and action in a meaningful way. In the field of cognitive psychology, cognition is studied by observing how and why humans' think, reason, learn, and remember. Many different mental abilities contribute to the nature of cognition but they cannot be directly observed and only be inferred from behaviour. Since observed performance is the only way to reconfigure mental abilities their clustering depends on the perspective and the techniques used for identification (Lezak et al., 2004). In order to establish a theoretical foundation for the present thesis, the following section provides a classification for three constructs, whose meanings are related to the concept of cognition. These include *attention*, *cognitive functions*, and *intelligence*. A definition for each of these concepts is provided and their relationship to each other is discussed.

1.1 Attention

Attention is one of the oldest controversial topics in psychology and still an incoherent construct today. Many years of research created the consistent view of attention as a multidimensional construct with different facets (Cohen, R. A., 1993). Some taxonomies that evolved are psychological (Mirsky, Anthony, Duncan, Ahearn & Kellam, 1991; Van Zomeren & Brouwer, 1994), others are based on neuroanatomy (Mesulam, 1985; Posner & Petersen, 1990). Therefore, different fields within the profession consider attention from various perspectives: some within behavioural dimensions (e.g. Diagnostic and Statistical Manual of Diseases) and others with respect to neuropsychology. Despite this ambiguity, a working definition of attention needs to be provided. Since most neuropsychological researchers accepted the multifaceted nature of attention its definition is grounded in the field of neuropsychology.

Attention has been described as an “assembly of basic abilities” (p. 247) and is often understood as “the allocation and control of limited processing resources in stimulus-driven and data-driven mental information processing” (Schweizer, 2010, p. 250). Since many researchers have a similar understanding of the controlling and allocating character of attention for more complex cognitive abilities (Schweizer, 2005, 2010), it is mostly depicted as a higher-order mental processing system in the brain, which is divided into several sub-components (see Table 1). The inte-

grated set of correlates in the brain mediates the efficacy of cognitive functions and produces neuropsychological impairment when the relevant brain regions are damaged (Cohen, R. A., 1993; Parasuraman, 1998; Posner & Petersen, 1990). This allows the prediction that cognitive disturbances and behavioural manifestations in conditions with central deficits of attention probably have their underlying causes within one or more of its components.

Table 1. Sub-components of attention

Selective attention (<i>also focused attention</i>)	The process of stimulus discrimination in a temporal-spatial frame of reference. Attention can be continuously shifted to the stimulus in focus, voluntarily or involuntarily, and can be directed by events in the outer environment (Cherry, 1953; Cohen, R. A., 1993).
Sustained attention	Characterised by mental effort and to remain attentive to one or more sources of information continuously (Davies, Jones & Taylor, 1984).
Divided attention	The monitoring of multiple sources of information at once, effortful or automatic. It focuses on the effect of limited capacity and the ability to divide resources in dual-tasks, which involve the presentation of simultaneous information (Posner & Boies, 1971; Treisman, 1969).
Alertness (<i>also vigilance</i>)	A state of wakefulness to detect stimuli that are presented infrequently over a longer period of time, with the ability of orienting or the capability of stable activation of attention (Posner & Petersen, 1990).
Attention switching	The ability to shift the focus of attention between locations, indicated by a cue (Van Zomeren & Brouwer, 1994)
Attentional control	A higher mental process that monitors and regulates responses. It amplifies relevant information and decreases activation of irrelevant/interfering information. Hence, it is responsible for inhibitory functions and complex goal-oriented behaviour (Norman & Shallice, 1986; Posner & Petersen, 1990; Posner & Rothbart, 2007).

Note. All components have two broad dimensions: *intensity* and *selectivity*, of which the former denotes the capability to activate and maintain attention (e.g. alertness, sustained attention) and of which the latter includes the alteration of responsiveness to a stimulus from a set of others (e.g. selective, divided attention).

1.2 Cognitive functions

Cognitive functions are divided into different classes and consist of many different abilities, which (1) select and integrate information, (2) store and retrieve information, (3) (re)organize information, and (4) act upon this information. Thus, they embody multiple domains of mental abilities that are essential for human thinking, reasoning, learning, and remembering. Each of the classes comprises more specific abilities, for example object recognition, concept formation, short term memory or speed of information processing. These abilities can be distinguished conceptually but their classification is not straightforward since the different abilities overlap and interact

with each other (Lezak et al., 2004). Together, they are often described as an intellectual process and the fundamental core of intelligence (Sternberg, 2009). The present thesis refers to all of these as cognitive functions, the umbrella term for specific components of intellectual abilities, which when deficient involve specific functional impairment.

They are to be separated from *executive functions*¹, which represent more complex and global abilities that supervise, control, organize, maintain, and integrate cognitive functions in order to initiate goal-oriented behaviour (Lezak et al., 2004). Until today, there is no formally agreed-upon definition on executive functions and there is little consensus of the components included. However, they have often been used to describe a variety of specific functions that deal with abstract thinking, inhibition, novelty, control, problem-solving, planning, and mental flexibility. Sometimes attention is subordinated to executive functions (e.g. Purdy, 2011) and been considered to underlie and maintain the activity of cognitive functions (Lezak et al., 2004). The relationship between attention and cognitive functions is further specified in the following.

Attention and the conceptualization of cognitive functions

Regarding attention as a multidimensional construct also implies that it has indistinct boundaries with cognitive functions (Schmidt-Atzert, Krumm & Bühner, 2008; Schweizer, Moosbrugger & Goldhammer, 2005). It is difficult to separate them from each other as their functions are interrelated and their definitions are controversial. Therefore, it is widely accepted that attention is an integral component to the conceptualization of cognitive functions. Particularly, the close relationship between attention and working memory as well as processing speed has been emphasised in the literature (Schweizer, 2010; Schweizer & Moosbrugger, 2004; Spikman & van Zomeren, 2010). Different theoretical models have evolved over the past decades to explain their relationship.

One model proposes the role of attentional control (Schneider & Shiffrin, 1977), also referred to as the supervisory attentional system (Norman & Shallice, 1986), in relation to working memory (e.g. Baddeley & Hitch, 1974; Cowan, 1988). The construct of attention is here understood as a function of higher mental processing and as considerably intertwined with working memory functions (Engle, 2002; Engle, Tuholski, Laughlin & Conway, 1999; Kane, Bleckley, Conway & Engle, 2001). Their relationship becomes evident in many neuropsychological tests, for example, in simple memory tasks, such as ‘Digit Span Forward’. During this task, attentional con-

¹ There is no intention of the present thesis to elaborate thoroughly on executive functions. However, its term will still play a role throughout these lines. Therefore, it is important to understand its meaning.

control can enhance performance by engaging to maintain information in short-term memory. In more complex span tasks, for example ‘Digit Span Sequencing’, the efficiency of attentional control can be measured more directly as great demands are placed on these mechanisms through information that needs to be encoded, processed, and successfully transferred and retrieved from long-term memory (Schneider & McGrew, 2012). Additional credence for the idea of their relatedness originates from neuroimaging studies that indicate similarities in neuronal activation and overlaps of active brain structures during attentional and working memory performance (e.g. Pessoa & Ungerleider, 2004).

The role of attention in information processing serves mainly as a source of efficiency for quick availability of information, simultaneous processing or coordination. In other words, speed and accuracy during which information is processed relates to changes in attention. As such, individuals with intact cognitive functions, such as language comprehension and visuo-spatial abilities may still perform poorly on processing speed tasks due to an inappropriate allocation of attentional resources (Cohen, R. A., 1993). Sometimes authors categorize attention as a special variant of processing speed (Schmidt-Atzert et al., 2008) or emphasize their relationship in theoretical models, such as the Cattell-Horn-Carroll theory of cognitive abilities (CHC; Schneider & McGrew, 2012). According to the CHC perspective, great credence is being placed to the aspect of attention during processing tasks in Wechsler Intelligence Scales. Subtests measuring processing speed are suggested to be strongly influenced by sustained attention, shifting of attention, and adaptive allocation of attention. In addition to processing speed, other cognitive abilities in the CHC model also interact with attention. In the literature, it is discussed to what extent attention explains the concept of intelligence (Schweizer, 2010). In order to understand their association, the following section defines intelligence and addresses their relationship by taking into account the CHC perspective.

1.3 Intelligence

Intelligence is a controversial topic in psychology and today there is a lack of agreement on a theory and its structure. Looking back at a long theoretical history², David Wechsler continues to be one of the most accepted professionals in the field of intelligence. With his conception that intelligence is “the aggregate or global capacity of the individual to act purposefully, to think rationally, and to deal effectively with his environment” (Wechsler, 1939, p. 3), he established a

² For a comprehensive review on theories of intelligence and its assessment please refer to Flanagan, Genshaft and Harrison (2012).

“gold standard” for our contemporary understanding. That is, intelligence as a multidimensional construct composed of different abilities and a global index for cognitive functions. Therefore, it is not regarded as a narrow and stable construct but a rather dynamic and integrative one (Flanagan et al., 2012; Schneider & McGrew, 2012), which predicts many life outcomes such as achievement, educational and occupational success, as well as adult poverty and pathology (Gottfredson, 1997). Based on this theory, many factor-analytic studies examined the amount of variance explained by other cognitive functions. To illustrate, attention has often been included to define individual intellectual differences (Schweizer et al., 2005) and a large proportion of variance in intelligence has been found to be explained by attention (Schweizer, 2010). With the idea in mind that attention is involved in intelligence and perhaps a reasonable mediator of other cognitive functions, it is important to consider their relationship within the confines of modern psychometric theories of intelligence, such as the CHC theory.

The Cattell-Horn-Carroll theory of intelligence

For the present thesis, the first comprehensive and empirically validated model of cognitive abilities is outlined. The CHC theory is an hierarchical model of intelligence proposed by McGrew and Flanagan (1998) and integrates Carroll’s three-stratum (Carroll, 1993) and the Cattell-Horn model of fluid (Gf) and crystallised (Gc) intelligence (Horn & Noll, 1997). Concisely, the three-stratum theory proposes a three-tier model of cognitive abilities that includes the general intelligence factor (g) at the broadest level (stratum III), broad abilities which represent stable characteristics of humans at stratum II (e.g. Gf and Gc), and a number of narrow abilities at stratum I (see Carroll, 1993, for detailed information). The Cattell-Horn model originally involves Cattell’s idea that g was best explained by Gf and Gc. A refinement by Horn extended the theory into a model of cognitive abilities with the inclusion of an array of broad abilities beyond that of Gf and Gc (Horn & Cattell, 1966). With the integration of both models (Flanagan, Genshaft & Harrison, 1997; Flanagan et al., 2012), the contemporary conceptualization of the CHC model serves as an umbrella model for the theory of intelligence and has formed the foundation for a large number of tests for intelligence (Schneider & McGrew, 2012). For example, the Wechsler Adult Intelligence Scale-IV (WAIS-IV; Wechsler, 2008) represents six broad abilities, including Gf, Gc, Visual Processing (Gv), Short-Term Memory (Gsm), Processing Speed (Gs), and Quantitative Knowledge (Gq). The extent to which the CHC theory is grounded in the WAIS-IV is depicted in Figure 1. For more information on CHC abilities and the WAIS-IV please refer to the Table A1.

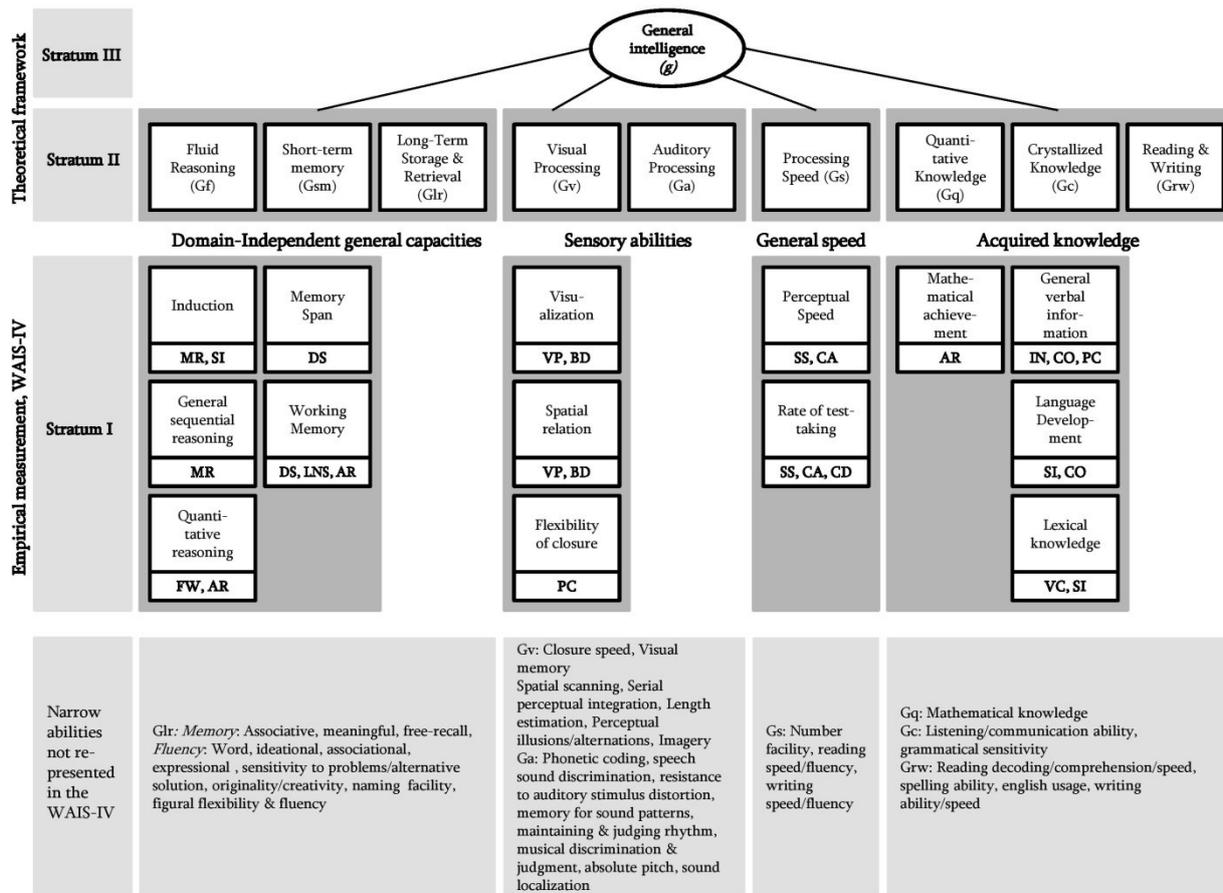


Figure 1. CHC theory partly grounded in the WAIS-IV (adapted from Schneider & McGrew, 2012).

AR, Arithmetic; BD, Block Design; CD, Coding; DS, Digit Span; FW, Figure Weights; Ga, Auditory processing; Gc, Crystallised intelligence; Gf, Fluid reasoning; Glr, Long-term storage and retrieval; Gq, Quantitative knowledge; Gs, Processing speed; Grw, reading and writing; Gsm, Short-term memory; Gv, Visual processing; IN, Information; LNS, Letter-Number Sequencing; MR, Matrix Reasoning; PC, Picture Completion; SI, Similarities; SS, Symbol Search; VC, Vocabulary; VP, Visual Puzzles; WAIS-IV, Wechsler Adult Intelligence Scale - Fourth Edition.

Attention and the conceptualization of the CHC theory

Attention is not represented as an independent ability within the CHC theory although it has often been empirically linked to *g* (e.g. Burns, Nettelbeck & McPherson, 2009; Schweizer et al., 2005). Even pioneers of intelligence, such as Charles Spearman and John Bissell Carroll proposed early that attention is an essential source of *g* (Stankov, 1983) and that “attention is involved [...] in all cognitive performances and, thus, in all performances that are regarded as indicating cognitive abilities” (Carroll, 1993, p. 547). In this regard, research supports earlier views on this relationship with the observation that tasks in intelligence tests, which have a high loading on *g*, also demand allocation of processing resources (Schweizer, Zimmermann & Koch, 2000). However, studies considering the role of attention in intelligence vary in their findings. This may be due

to how differently intelligence is conceptualised. Accumulative research investigating the relationship between attention and the CHC perspective of intelligence using factor-analytic analyses particularly emphasize an association between different components of attention and *g* as well as an overlap between measures of *Gf* and *Gs* with those of attention (Schweizer et al., 2000). Most importantly, sustained attention was found to predict intelligence (Schweizer & Moosbrugger, 2004) and was closest associated to *Gf* (Crawford, 1991). Other constructs of attention, of which some had great similarities to *Gs*, have been identified to load high on general intelligence (Schweizer et al., 2005). Further credence to the relationship between attention and intelligence was added from a factor analytic study suggesting that the performance in tasks that approximate Carroll's taxonomy of general intelligence can be predicted by a battery of attention tests (Burns et al., 2009). Together, these findings assume that (a) attention in general is an essential aspect that is integrated in the concept of intelligence according to the CHC theory, that (b) sustained attention is especially related to *Gf* because it reflects important properties of information-processing and in turn is most closely related to general intelligence, and (c) that sustained attention is related to *Gs*, because of the great overlap between measures of sustained attention and processing speed. Considering this, it becomes clear that attention is narrowly intertwined with intelligence and cognitive functions. Therefore, many pathological conditions with deficits of attention experience subtle or severe dysfunctions in general intellectual and cognitive functioning. In order to understand the nature of attention deficits and their consequences, the next section introduces two pathological conditions with central deficits of attention.

2 Disorders related to attention deficits

Attention deficits of neurological conditions and mental illnesses serve as the underlying basis for inappropriate operation of cognitive functions; some of which are more severely affected than others, yet, none of them can operate at normal levels (Gitelman, 2003). Generally, the nature of attention deficits varies as a function of the type of condition which is considered. Therefore, psychiatric developmental and neurological conditions can affect attention and cognitive domains differently. In turn, these deficits result in diverse symptomatic manifestations. Some disorders produce large disturbances (e.g. hemispatial neglect), whereas others result in wide ranging cognitive deficits (Cohen, R. A., 1993). To understand the clinical aspects of both, psychiatric and neurological disorders and their consequences, the following sections introduce two distinguishable conditions with central deficits of attention. One is drawn from the field of psychology, a psychiatric neuro-developmental disorder of attention, Attention Deficit Hyperactivity Disorder (ADHD), and another from the field of neurology, which is a medically acquired neurological disorder, stroke. The following part introduces these disorders separately in terms of their aetiology, pathogenesis, and clinical picture.

2.1 Psychiatric neuro-developmental disorder of attention (ADHD)

ADHD has long been thought to be a disorder only occurring in children. In 1970, ADHD was first mentioned in adults and a sizeable foundation of research supports the notion that it persists, as a reliable and valid disorder, into adulthood (Barkley, Fischer, Smallish & Fletcher, 2002; Biederman, Petty, Evans, Small & Faraone, 2010; Kessler et al., 2010). The extent to which symptoms of ADHD continue into adulthood is difficult to estimate because of methodological heterogeneities across studies. Thus, rates of children with ADHD still demonstrating symptoms as adults range between 29% and 76% (Barbarese et al., 2013; Biederman, Petty, Clarke, Lomedico & Faraone, 2011; Biederman, Petty, Evans, et al., 2010; Biederman, Petty, Monuteaux, et al., 2010; Kessler et al., 2006; Lara et al., 2009). Despite this very high persistence, the acceptance of ADHD as a lifespan disorder is relatively recent (Schmidt & Petermann, 2009b). Currently, the prevalence of adults who meet the criteria for ADHD has been estimated³ at 4.4% in the United States (Kessler et al., 2006) and 4.7% in Germany (Zwaan et al., 2012). Regarding its economic burden,

³ Estimates vary due to the application of different diagnostic criteria and sample selection (see Simon, Czobor, Bálint, Mészáros & Bitter, 2009, for a review).

recent research found substantial costs of adult ADHD in the United States (see Doshi et al., 2012, for a review). Overall, the national increment costs (= societal costs) for adult ADHD or to the adults' family members were estimated up to \$194 billion. These costs were substantially higher than those for children and adolescents. The largest costs indicated for adults were those for productivity and income loss (\$87 to \$137 billion). This suggests that ADHD in adulthood affects not only the adults themselves but the whole society in many areas of life.

Since it was accepted that ADHD affects adults, a large amount of scientific studies has emerged, but the literature is often contradictory in discussing its aetiology, clinical symptom presentation, assessment, diagnosis, and treatment. Together, the growing evidence and opinions about the disorder bring confusion into clinical practice and there is an urgent need to develop some uniformity into the discussion of adult ADHD. The purpose of this section is to offer an overview of ADHD in adulthood. More particularly, it defines ADHD in adults by providing a brief outline on the diagnostic criteria and recent acknowledgements, aetiology and neurobiology, symptoms, and comorbid conditions. A more thorough part addresses current research findings on neuropsychological deficits. Subsequently, an overview of the assessment process in clinical practice is provided.

2.1.1 Definition of ADHD in adults

ADHD is a dimensional disorder, which indicates that it is not possible to determine a diagnosis based on the simple presence of abnormal behaviour or clinically significant test results. Instead, ADHD related symptoms represent the end of a dimension of cognitive processes that generate deficits in self-regulation, impulse-control, attention, and organization. Given that these problems are not considered features of ADHD alone, the distinct developmental and symptom profile needs to be recognised through comprehensive assessment methods. Only then, may an adequate diagnosis of ADHD be established (Ramsay, 2010). Sufficiently diagnosing ADHD in adulthood remains challenging and accordingly up to 90% of the adults remain undiagnosed and untreated (Kessler et al., 2006). This suggests that ADHD in adults is poorly understood. The barriers brought along include (a) the unspecified nature and the underlying reasons of ADHD, (b) the nonspecific symptoms and high levels of comorbidity, (c) the lack of diagnostic guidelines, and (d) unspecific assessment methods (Parker et al., 2012). These obstacles contribute to uncertainties in clinical practice. As such, 72% of clinicians find it more difficult to diagnose ADHD in adults than in children, 34% are not very knowledgeable about diagnosing ADHD in adults, and almost half are not confident enough to give a diagnosis (Adler, Shaw, Sitt, Maya & Ippolito, 2009). Consider-

ing this, it seems not surprising that many mental health professionals fail to recognize ADHD in adults. Very problematic are diagnostic classification systems because they stipulate the criteria in adults the same way they are applied in children. A concise overview on the diagnostic criteria of adult ADHD follows, with reference to recent acknowledgements and changes in diagnostic guidelines.

Diagnostic criteria

A diagnosis of ADHD in adults is based on developmentally inappropriate symptoms of inattention, hyperactivity, and impulsivity, which are outlined in diagnostic classification systems, including the Diagnostic and Statistical Manual of Mental Disorders (4th ed.; DSM-IV-TR; American Psychiatric Association, 2000), International Classification of Diseases (10th ed.; *ICD-10*; World Health Organisation, 1992), and the Wender-Utha criteria (Retz-Junginger et al., 2002; Ward, Wender & Reimherr, 1993). Compared to the ICD-10 and the DSM-IV-TR, the Utah criteria have high levels to infer an ADHD diagnosis (Rösler, Retz, et al., 2008). The DSM-IV-TR and ICD-10 almost equally classify ADHD symptoms but they differentiate in the combination of criteria to achieve a diagnosis: the ICD-10, most often used in Europe, more conservatively emphasizes the presence of ADHD symptoms. Persistent symptom patterns of inattention and hyperactivity/impulsivity must be present to achieve a diagnosis of ADHD (F90.0). The DSM-IV-TR differentiates between subtypes of inattention and hyperactivity/impulsivity and classifies three subtypes: predominately hyperactive, predominately inattentive, and a combined type of the former forms. Since the criteria of both classification systems were designed to diagnose children with ADHD a strict usage of them leads to under-diagnoses in adults (Kooij et al., 2010). Emerging from these results are claims that propose inadequate thresholds of the DSM for adults with ADHD (Barkley, Murphy & Fischer, 2008). By siding with the developmental view of ADHD, it has been suggested that using a threshold of four symptoms best classifies adults with ADHD. Many similar assertions evolved from longitudinal empirical research (e.g. UMass and Milwaukee Study; Barkley et al., 2008) and provoked recent advantages in diagnostic development. Today, the DSM in its fifth edition (5th ed.; *DSM-V*; American Psychiatric Association, 2013) acknowledges ADHD as a disorder occurring in adults by including more emphasised diagnostic criteria and more accurately characterizing symptoms. With the DSM-V, adults who previously met partial remission criteria or who did not receive a diagnosis can now meet the diagnosis of persistent ADHD. That means that diagnoses of ADHD in adulthood may increase in the future (see Faraone, Biederman

& Mick, 2006, for a review). The most important changes to diagnosing ADHD in adulthood are outlined below and the diagnostic criteria are depicted in Table 2.

- **Onset:** the age of onset criterion has been broadened and includes ADHD symptomatology by the age ≥ 12 years.
- **Reduction of symptom threshold:** with the recognition of age dependent changes in the course of ADHD, only ≥ 5 instead of six required symptoms of inattentive/hyperactive-impulsive are required (≥ 17 years).
- **Increase of number of symptoms:** more impulse control symptoms have been added. Thus, the list of hyperactive-impulsive symptoms has been increased to 13.
- **Elaboration of symptoms:** specific descriptions of behaviour have been added to the criterion list to facilitate the symptom application throughout development.
- **Exclusion criteria:** Autism Spectrum Disorder is no longer a criterion of exclusion for a diagnosis of ADHD.
- **Chapter category:** ADHD is now considered as a neuro-developmental disorder in order to emphasize disturbances in brain developmental aspects, thereby eliminating the sole focus of ADHD in infancy and childhood.

Table 2. Diagnostic criteria of the DSM-V for adult ADHD (≥ 17 years)

		Hyperactivity/ Impulsivity	Inattentive	Combined
I	Inattention	not required	≥ 5 (for 6 months)	≥ 5 (for 6 months)
	Hyperactivity/ Impulsivity	≥ 5 (for 6 months)	not required	≥ 5 (for 6 months)
II	Age of onset	≤ 12 years	≤ 12 years	≤ 12 years
III	Settings	≥ 2	≥ 2	≥ 2
IV	Functional impairment	There is clear evidence that the symptoms interfere with, or reduce, the quality of social, academic, or occupational functioning		
V	Exclusion criteria	The symptoms are not present during the course of schizophrenia or other psychotic disorder and they are not better explained by other conditions such as mood or anxiety disorder, dissociative or personality disorder or substance intoxication or withdrawal		

Note. Further specification can be made in (1) severity: mild, moderate, and severe and in (2) partial remission: previously met full diagnostic criteria, fewer than the full criteria met during the past six months, and ADHD-related symptoms lead to impairment in functional outcomes (American Psychiatric Association, 2013).

Aetiology and Neurobiology

ADHD is a heterogeneous disorder with multiple and complex aetiologies, which are not yet clearly determined. Generally, it is agreed that both inherited (genetic) and no-inherited (environmental) risk factors interdependently contribute to the development of ADHD (Nigg, Nikolas & Burt, 2010). Studies have identified genetic risk factors (Faraone & Doyle, 2001) with heritability as high as 75% to 91% (Faraone et al., 2005; Levy, Hay, McStephen, Wood & Waldman, 1997) and causes, including prenatal compromises or delivery complications (Mick, Biederman, Prince, Fischer & Faraone, 2002), environmental toxins, dietary factors (e.g. nutritional deficiencies, nutritional surpluses), and psychosocial adversity (see Thapar, Cooper, Eyre & Langley, 2013, for a review). Moreover, there is robust evidence for neurobiological causes. Converging evidence suggests changed brain activation pattern. Thus, a large body of studies report fronto-striatal dysfunction and dopaminergic and noradrenergic imbalances of neurotransmission in the prefrontal cortex for the clinical presentation of ADHD (e.g. del Campo, Chamberlain, Sahakian & Robbins, 2011; Konrad et al., 2010; Russell, 2002). Furthermore, growing Functional/Magnetic Resonance Imaging research provides robust evidence of cortical thinning and smaller volumes of right frontal areas, dorsolateral prefrontal cortex, cerebellum, caudate, pallidum, and corpus callosum (Durstun et al., 2004; Makris et al., 2007; Seidman et al., 2006; Valera, Faraone, Murray & Seidman, 2007). The above structural and functional brain abnormalities subserves deficits in attention, executive functions, and cognitive processes, which in turn lead to inefficient behaviour and functional impairment (Barkley, 1997, 1999; Castellanos & Tannock, 2002). To date, there is no consensus on the causal determinants for behaviour associated with ADHD.

Behavioural symptoms

Neuropathological approaches of ADHD assume that an inefficient behavioural outcome is manifested through disturbances of neuropsychological functions, which are linked to frontal brain regions (see Castellanos & Tannock, 2002; Huang-Pollock & Nigg, 2003; Willcutt, Doyle, Nigg, Faraone & Pennington, 2005, for reviews). Especially in childhood, these disturbances are reflected in the presence of inappropriate levels of inattentive-disorganised and hyperactive-impulsive behaviour across different settings. Given that it has been refuted that children grow out of disorder (Barkley, Fischer, et al., 2002; Biederman, Petty, Evans, et al., 2010), it is widely accepted that ADHD core symptoms persist throughout development. However, in adults there is a significant decrease of symptoms accounted for by a reduction of hyperactivity and impulsivity. That is to say, the symptom severity and quality, as well as long-term effects across multiple life

domains change over time. A reason for this may be ongoing brain maturation and different responsibilities that face adults in daily life. As a result, they may experience more heterogeneous symptoms, primarily those marked by inattention. Moreover, adults are more likely to exhibit neuropsychological disturbances, which adversely affect their functional outcomes (see Hervey et al., 2004; Seidman, 2006, for reviews). In the adults' daily life, these symptoms lead to severe interferences. For example, symptoms of inattention are associated with disorganization and distractibility as well as increased sensitivity to stress. Hyperactivity results in fidgeting and excessive talk. Symptoms of impulsivity are expressed by excessive impatience and acting without thinking (Kooij et al., 2010). As detrimental as the negative effects of the symptomatology on the adults themselves are the adverse effects on social functioning. They may lead to frequent changes in partnerships (Robin & Payson, 2002), impaired parenting processes (Harvey, Danforth, McKee, Ulaszek & Friedman, 2003), reduced educational attainment or occupational performance (Biederman, Petty, Fried, Kaiser, et al., 2008; de Graaf et al., 2008; Gjervan, Torgersen, Nordahl & Rasmussen, 2012), unemployment and lower financial resources (Kessler, Adler, Ames, Barkley, et al., 2005; Kessler, Lane, Stang, Van Brunt & Trott, 2008), increased rates of criminality (Mannuzza, Klein & Moulton, 2008; Ziegler, Blocher, Bro & Rösler, 2003), risky driving and increased car accidents (Barkley, Murphy, Dupaul & Bush, 2002; Fischer, Barkley, Smallish & Fletcher, 2007), and exacerbated addictive illnesses (Ohlmeier et al., 2008).

Comorbidity

The general probability of comorbid diagnoses increases with age (Yang, L. et al., 2007) and in intensity (Kessler et al., 2006). Accordingly, the clinical picture of ADHD in adults is marked by an elevated prevalence of comorbid psychiatric conditions. This implies an increased heterogeneity of symptoms and subsequent complications in the diagnostic process. Prevalence rates of at least one other comorbid condition in adults with ADHD have been estimated as high as 87%. At least two other comorbid disorders have been found in 56% of the adults (McGough et al., 2005; Torgersen, Gjervan & Rasmussen, 2006). The most frequent reported comorbid disorders include anxiety disorders ($\approx 47\%$), mood disorders ($\approx 38\%$), impulse control disorders (e.g. Borderline Personality Disorder) ($\approx 20\%$), and substance use disorders (≈ 15 to 47% ; Kessler et al., 2006; McGough et al., 2005).

2.1.2 Current state of research: Neuropsychological functions in adult ADHD

Over the last 40 years, it has been demonstrated that the developmental alterations of ADHD were not only related to behavioural impairments but also to neuropsychological deficits (see Seidman, 2006, for a review). Until recently, most research on neuropsychological functions has been built upon studies on childhood ADHD. However, the findings are not always consistent across developments, depicting neuropsychological deficits often differently in adults with ADHD (Rashid, Morris & Morris, 2001; Schoechlin & Engel, 2005; Thaler, Bello & Etcoff, 2013; Valko et al., 2009; Valko et al., 2010). Yet, sometimes equal impairment has been indicated in children and adults (see Hervey et al., 2004; Woods, Lovejoy & Ball, 2002, for reviews). Generally found in adults with ADHD are a broad and heterogeneous range of deficient neuropsychological functions in comparison to healthy individuals (see Bridgett & Walker, 2006; Frazier, Demaree & Youngstrom, 2004; Hervey et al., 2004; Schoechlin & Engel, 2005, for reviews). Impairment has been identified in up to 50% of the adults (Lovejoy et al., 1999). This suggests that not all adults with ADHD show evidence of these deficits, but that does not imply that normal test scores rule out a diagnosis of ADHD (Seidman et al., 2004). In terms of gender differences, neuropsychological deficits have been found to be largely the same for boys and girls with ADHD (see Seidman, 2006, for a review). Similar findings have been reported in the adult literature (Biederman, Faraone, Monuteaux, Bober & Cadogen, 2004; Silva et al., 2013).

A debate surrounding neuropsychological research pertains to whether neuropsychological deficits are independent of comorbid psychiatric conditions or are in fact an artefact (Faraone et al., 2000; Marchetta, Hurks, De Sonneville, Krabbendam & Jolles, 2008; Marchetta, Hurks, Krabbendam & Jolles, 2008; Seidman, 2006; Silva et al., 2013). According to the common aetiology hypothesis, there is a negative additive effect on neuropsychological functions in adults with ADHD and comorbid conditions. This means that deficits are combined and are, thus, more pronounced compared to adults without comorbid conditions (described in Willcutt, Pennington, Olson, Chhabildas & Hulslander, 2005). However, research still remains inconclusive. Similarly, a discussion on the influence of psychopharmacological mediation (e.g. methylphenidate) on neuropsychological functions has been started. Some argue that a stable medication intake does not normalize performance on neuropsychological tests in adults with ADHD (Advokat, 2010; Müller et al., 2007), yet others support the idea of stimulants as cognitive enhancers (Aron, Dowson, Sahakian & Robbins, 2003; Turner, Blackwell, Dowson, McLean & Sahakian, 2005). Generally, it is indicated that the benefit of medication on performance is task specific (Kurscheidt et al., 2008).

In what follows, an overview of research findings on neuropsychological deficits in adults with ADHD is provided, individually for different cognitive domains and for general intellectual functioning. This outline is not exhaustive, but serves to illustrate the most important research findings to ensure that the full breadth of deficits in adults with ADHD is considered. The focus is on recent research, which is based on standardised neuropsychological assessment measures. Table 3 summarizes these findings.

Attention in adults with ADHD

Theories emphasize the central role of attention in ADHD for behavioural outcomes (Biederman, Mick & Faraone, 2000). Deficient deployments of attention are the most consistent finding in adults with ADHD. Across studies, the effects sizes of neuropsychological tests measuring attention range between medium and large (see Hervey et al., 2004, for a review). These effects were particularly reported on sustained attention and vigilance (Marchetta, Hurks, De Sonneville, et al., 2008; Schoechlin & Engel, 2005) but were not always verified (Epstein, Conners, Sitarenios & Erhardt, 1998; Holdnack, Moberg, Arnold, Gur & Gur, 1995). Some research found evidence of general inattention (e.g. errors of omission) and deficits in controlled attentional processes (e.g. response inhibition; Epstein, Johnson, Varia & Conners, 2001; Fischer, Barkley, Smallish & Fletcher, 2005; Pazvantoğlu et al., 2012; Walker, Shores, Trollor, Lee & Sachdev, 2000; Young & Gudjonsson, 2005). There are limited findings on adults with ADHD and deficits in selective and divided attention (Woods, Lovejoy, Stutts, Ball & Fals-Stewart, 2002; Young & Gudjonsson, 2005). Overall, the results on deficient attentional constructs are inconsistent and, at times, contradictory. It seems that attentional dysfunctions were found more often in more complex tasks with increased attentional demands, such as '4th Stroop Test' or 'Trail Making Test-B', compared to tasks with basic attentional demands, such as 'Digit Span Forward' (Pazvantoğlu et al., 2012; Rohlf et al., 2012; Schoechlin & Engel, 2005). In terms of comorbidity, research showed that comorbid disorders in adults with ADHD are positively related to attentional deficits (see Woods, Lovejoy & Ball, 2002, for a review). Others have found that sustained attentional performance is independent of comorbidity, but that adults with comorbid conditions are characterised by more severe problems of focused attention (Marchetta, Hurks, De Sonneville, et al., 2008). A large number of studies are inconsistent and the effect of comorbidity was not controlled for (see Woods, Lovejoy & Ball, 2002, for a review). Furthermore, research on adults with ADHD were medicated with stimulants provided evidence for an improvement of reaction times and increased hit rates in tests measuring sustained attention or vigilance. However, stimulus evaluation (index

of impulsivity) in demanding attentional tasks requiring more enhanced attentional abilities seemed not to be affected or even impaired by stimulants (Advokat, 2010; Barrilleaux & Advokat, 2009; Turner et al., 2005).

Executive functions in adults with ADHD

Since ADHD has been conceptualised as a condition of behavioural inhibition (Barkley, 1997), a large body of research primarily focused on aspects of executive functions, which have consistently been found deficient (see Willcutt, Doyle, et al., 2005, for a review). However, theories of executive functions as core deficits have increasingly been questioned (Castellanos, Sonuga-Barke, Milham & Tannock, 2006; Nigg, 2001). Nevertheless, much research noted poor performance on neuropsychological instruments that assume to measure executive functions and confirmed deficits in several domains. As such, small to medium effects have been found on tasks measuring attention distraction and large effects on tests of response inhibition, set shifting, and planning (see Hervey et al., 2004, for a review). Robust evidence was revealed for the impairment of response inhibition (Antshel et al., 2010; Bekker et al., 2005; Boonstra, Kooij, Oosterlaan, Sergeant & Buitelaar, 2010; Boonstra et al., 2005; Hervey et al., 2004; Murphy, Barkley & Bush, 2001; Nigg, Stavro, et al., 2005; Rapport, Van Voorhis, Tzelepis & Friedman, 2001; Woods, Lovejoy, Stutts, et al., 2002) and set shifting (Boonstra et al., 2010; Boonstra et al., 2005; Halleland, Haavik & Lundervold, 2012; McLean et al., 2004; Pazvantoğlu et al., 2012; Rohlf et al., 2012). Other aspects of executive functions, including planning, organisation, and problem solving have also been found to be deficient (McLean et al., 2004; Nigg, Stavro, et al., 2005; Schoechlin & Engel, 2005; Woods, Lovejoy & Ball, 2002; Young, Morris, Toone & Tyson, 2007). This could not be confirmed by all studies, which reported only small effect sizes, particularly in planning and problem-solving (Aycicegi-Dinn, Derwent-Ozbek, Yazgan, Bicer & Dinn, 2011; Holdnack et al., 1995; Pazvantoğlu et al., 2012; Riccio, Wolfe, Romine, Davis & Sullivan, 2004; Schoechlin & Engel, 2005). Furthermore, there has been no indication on the contribution of comorbid conditions in adults with ADHD on executive function test performance (Antshel et al., 2010; Boonstra et al., 2010). However, the influence of comorbid conditions could still not be ruled out completely (Rohlf et al., 2012). In terms of the effects of stimulants on executive functions research remains inconclusive. A recent review found that performance on tasks involving more complex functions, such as adaption, planning, and flexibility did not improve with stimulant intake and may even impair performance. This was explained by increased rates of arousal, which in turn facilitate impulsivity and depressed performance (see Advokat, 2010, for a review). Other studies support posi-

tive effects of stimulants on performance. As such, it was shown that processing of tasks-irrelevant stimuli reduces the latent inhibition effect in visual search tasks (Lubow, Kaplan & Manor, 2014).

Information processing speed in adults with ADHD

Speed of information processing is considered an important component of the deficits found in adults with ADHD. Studies reported slower information processing compared to healthy adults based on perceptual motor speed tasks (Holdnack et al., 1995; Lovejoy et al., 1999; Rapport et al., 2001; Seidman, 2006; Walker et al., 2000; Woods, Lovejoy & Ball, 2002). Collectively, effect sizes of processing speed were reported in small ranges (Holdnack et al., 1995) and were much lower compared to other cognitive functions. Larger effects were commonly found on tasks with aspects of executive and set-shifting components (e.g. 'Trail Making Test-B'; see Hervey et al., 2004, for a review). Other authors support this given the lack of evidence for differences on simple processing speed tasks, but on those with more cognitive demands (e.g. 'Trail Making Test-B', 'Digit-Symbol', 'Stroop'; Pazvantoglu et al., 2012; Walker et al., 2000). Some failed to confirm this (Holdnack et al., 1995). Limited research has been done on the effect of comorbidity on speed of information processing. Yet, it has been argued that comorbidity and ADHD in adults should receive specific focus given that deficits in task of information processing are likely to be attributable to comorbid conditions in adults with ADHD (Marchetta, Hurks, Krabbendam, et al., 2008). Moreover, stimulant medications seem not to interfere or to improve processing speed tasks (see Advokat, 2010, for a review).

Working memory in adults with ADHD

In meta-analytic work, working memory has been suggested to be a core feature in adults with ADHD, with up to 84% of the adults displaying clinically significant performance in working memory tasks (see Alderson, Kasper, Hudec & Patros, 2013, for a review). The deficits are explained by different theoretical models, such as Baddeley and Hitch's model of working memory (Baddeley, 1986). It proposes a central executive as an attention controller and coordinator for two slave systems, the phonological loop (temporary storage/rehearsal of verbal information) and the visuo-spatial sketchpad (temporary storage/rehearsal of visual/spatial information). Robust empirical evidence for verbal working memory deficits in adults with ADHD relative to healthy individuals suggests insufficient allocation of attention to the phonological loop and hence deficient encoding processes. Consistent small to medium effects on verbally based memory tasks but not on those involving visual-spatial stimuli have been reported in a meta-analytic studies (see Hervey et al., 2004; Schoechlin & Engel, 2005; Skodzik, Holling & Pedersen, 2013, for reviews). A large

number of studies confirmed this and demonstrated small to medium effects on 'Digit Span' tasks and 'Arithmetic' of the WAIS (e.g. Boonstra et al., 2005; Bridgett & Walker, 2006; Quinlan & Brown, 2003; Rohlf et al., 2012; Walker et al., 2000; Willcutt, Doyle, et al., 2005) or revealed impaired performance on other verbal working memory tests (e.g. Marchetta, Hurks, Krabbendam, et al., 2008; Schoechlin & Engel, 2005). In contrast, no effects on visual working memory tasks were found (see Skodzik et al., 2013, for a review) and only a limited number of studies indicated difficulties in spatial memory performance (e.g. McLean et al., 2004). A recent meta-analytic review reported similar effect sizes for verbal and spatial working memory domains. Large effect sizes were found for verbal tasks, particularly those placing greater demands on the central executive (e.g. 'Letter-Number Sequencing'; see Alderson et al., 2013, for a review). With regard to the influence of comorbid conditions, it has been revealed that verbal working memory deficits are independent of comorbidity (Marchetta, Hurks, Krabbendam, et al., 2008). Moreover, it has been suggested that stimulant medication does not enhance or even impair short-term memory acquisition, yet retention of information may be improved (see Advokat, 2010, for a review).

Visuo-spatial functions and language in adults with ADHD

Studies largely revealed intact visuo-spatial skills in adults with ADHD (Rapport et al., 2001; Seidman, 2006; Seidman, Biederman, Weber, Hatch & Faraone, 1998). There is little research that does not confirm these findings (e.g. Schreiber, Javorsky, Robinson & Stern, 1999). Moreover, ADHD in adults has been associated with difficulties in verbal fluency, which involves a rapid production of words. Some studies have reported a marked reduction of total word output on verbal fluency tasks (e.g. Dinn, Robbins & Harris, 2001; Lovejoy et al., 1999; Schoechlin & Engel, 2005; Tucha et al., 2005; Woods, Lovejoy, Stutts, et al., 2002) but other have not (e.g. Boonstra et al., 2010; Marchetta, Hurks, Krabbendam, et al., 2008) and suggested relatively intact verbal intellectual abilities (Holdnack et al., 1995). However, many verbal fluency tasks also tap onto executive functions (e.g. inhibition). Therefore, studies using 'pure' linguistic measures involving more semantic and lexical knowledge (e.g. Vocabulary subtest) found divergent results and reported relatively intact verbal abilities and rigorous crystallised intelligence (Pazvantoğlu et al., 2012; Rapport et al., 2001).

Intelligence in adults with ADHD

Results on general intelligence in adults with ADHD are inconsistent and it appears that their relationship is inconclusive at this moment (Goodwin, Gudjonsson, Sigurdsson & Young, 2011). Compared to healthy adults, studies reported significantly attenuated Full Scale Intelligence

Quotient (FSIQ) scores (e.g. Biederman et al., 1993; Boonstra et al., 2010; Hervey et al., 2004; Holdnack et al., 1995) of on average 2.94 points (see Bridgett & Walker, 2006, for a review) or a decrement of a 9 point magnitude (see Frazier et al., 2004, for a review). In this context, medium effect sizes occurred for general intelligence (see Kofler et al., 2013, for a review). A below average FSIQ, however, was not equally found in all studies, suggesting that the ADHD adult population is not always likely to be associated with an intelligence decrement (Nigg, Butler, Huang-Pollock & Henderson, 2002; Rapport, Friedman, Tzelepis & Van Voorhis, 2002; Seidman et al., 1998; Woods, Lovejoy, Stutts, et al., 2002). Moreover, it has been claimed that comprised FSIQ scores can be explained by the sensitivity of subtests to attentional abilities, working memory, and processing speed functions, which have often been found deficient in adults with ADHD (Harrison, DeLisle & Parker, 2008). This led to a debate whether or not the diagnosis of ADHD is valid in adults with a high FSIQ and whether a depressed FSIQ is at all applicable as an index. With regards to the former, it was found that an above average FSIQ leads to similar functional impairment, neuropsychological deficits, and comorbidities reported in those with an average FSIQ or in high FSIQ controls (Antshel et al., 2008; Antshel et al., 2010). This suggests that a high FSIQ does not automatically infer a good functional outcome, supporting the validity of an ADHD diagnosis in adults with above average intelligence. The question remained if the FSIQ can be used as a representative index due to prominent deficits in attention, working memory, and processing speed. To this end, the relationship between the FSIQ and the General Ability Index (GAI)⁴ in adults with ADHD has been examined. Given that the adults usually demonstrate a lower FSIQ than GAI it has been suggested that impairment in these abilities depress general intelligence (Harrison et al., 2008). Furthermore, in terms of comorbidity, some kind of interaction between the FSIQ and comorbid conditions has been proposed. Adults with ADHD, but without comorbidity appear similar to healthy controls on general intelligence measures compared to those adults with comorbidity (see Bridgett & Walker, 2006, for a review).

An overview of the research findings in adults with ADHD

In spite of much variability with regard to the application of assessment tools, the present overview suggests that the neuropsychological performance of adults with ADHD is highly variable and characterised by a wide variety of deficits. It can be concluded that there is little indication which impairments are truly specific (for a concise overview see Table 3) because all domains

⁴ The General Ability Index (GAI) refers to an alternate global composite score of general intellectual functioning composed solely of verbal and perceptual reasoning constructs, thereby eliminating processing speed and working memory abilities (Wechsler, Coalson & Raiford, 2008). For more information on its calculation please refer to section 4.2.1.

of cognitive functions revealed at least some degree of impairment. These deficits may be expressed in information processing and attention abilities, which in turn partially account for poor performance on tasks measuring other cognitive domains. Moreover, deficits in neuropsychological tasks may be exacerbated by comorbid conditions. Yet, an additive negative effect of comorbidity is unclear. In addition, there is inconsistent evidence that stimulants act as a cognitive enhancer of test performance.

Table 3. Summary of the current state of research in adult ADHD

Attention	Research provides consistent evidence of medium to large effect sizes in tests that are designed to measure attention. Impairment in adults with ADHD compared to controls is evident in tasks with high attentional demands, particularly in tests of sustained attention and controlled attentional processes.
Executive functions	A large number of studies found differences between adults with ADHD and controls in executive function tasks that assess response inhibition and set shifting. However, there is inconsistent evidence regarding other tasks of executive functions. Effect sizes are reported between small and large ranges.
Information processing speed	Effect sizes for tasks measuring processing speed are relatively small. Specifically confined to adult ADHD appear higher demands in processing speed load. Simple reaction time tasks with minimal processing demands appear to be unaffected.
Working memory	There is robust evidence for deficits in verbal working memory in adults with ADHD relative to controls. The largest effects are suggested to occur in tasks with demands on the central executive.
Intelligence	There is large variability in results pertaining to general intelligence, with studies reporting any differences in the FSIQ ^a relative to controls and studies indicating large and significant discrepancies. Limited evidence regarding FSIQ ^b -GAI ^b relationship exists but research so far indicates lower FSIQ ^b scores compared to higher GAI ^b scores.
Visuo-spatial functions and language	If tasks do not tap on executive and attentional controlled processes, crystallised and visuo-spatial knowledge seems to be relatively intact in adults with ADHD.

^aFull Scale Intelligence Quotient; ^bGeneral Ability Index.

2.1.3 The assessment process in adults with ADHD

The assessment of ADHD in children is much more established than in adults. During childhood, the diagnostic approach is commonly based on diagnostic criteria and clinical guidelines (e.g. American Academy of Child and Adolescent Psychiatry, 2007; Taylor et al., 2004). The point of convergence for children with ADHD is the exploration of informants' accounts and observational data (Görtz-Dorten & Döpfner, 2014). Additionally, there is a long tradition for the use of neuropsychological tests, which however have often been questioned in terms of positive and negative predictive power (see Seidman, 2006, for a review). Conversely, the assessment of adults

remains contentious and scarcely any guidelines exists (see Seixas, Weiss & Müller, 2012, for a review). This inconsistency leads to many challenges in clinical practice (Adler et al., 2009; Waite, Vlam, Irrera-Newcomb & Babcock, 2013) with the result that mental-health professionals do not feel confident enough to give a diagnosis of ADHD in adulthood (Adler et al., 2009). The purpose of the following section is to provide an overview of the assessment process for adult ADHD, including screening measures, standardised interviews, and neuropsychological instruments.

Screening measures and diagnostic interviews

Diagnosing ADHD in adults requires not only evidence of ADHD related behavioural problems but a retrospective childhood diagnosis. Therefore, the exploration of both current and past symptom burdens is necessary. Central to achieving this is the use of self-report screenings. Occasionally applied but not required are informant-reports to infer a diagnosis (Görtz-Dorten & Döpfner, 2014). Several screening tools to evaluate ADHD symptoms exist and many of them are also found to correlate with ADHD in adulthood (e.g. Conners, Erhardt & Sparrow, 1999; Rösler, Retz-Junginger, Retz & Stieglitz, 2008; Schmidt & Petermann, 2009a). The choice, which one to operationalize is, however, not universally agreed upon. Some examples of screenings frequently used in clinical practice are depicted in Table 4.

The main motivation to apply self-reports is to gain cost-effective collateral information to indicate whether adults meet the diagnostic criteria for ADHD. Subsequent to a positive screen, further assessment for symptoms, behaviour, and comorbidity is necessary. This is most often done by means of standardised behavioural ratings and structural diagnostic interviews. They assume to provide data on the perceived degree of psychological distress, the prevalence and intensity of behavioural deficits, and functional impairment as well as information on differential diagnoses (Surman, 2013). A systematic review of clinical guidelines on ADHD concludes that clinical interviews remain the gold standard of an assessment (see Seixas et al., 2012, for a review).

Although self-reports in form of screenings, standardised questionnaires or interviews constitute the fundamental basis to achieve diagnostic conclusions, their validity and accuracy has frequently been questioned. It has been indicated that both screenings and interviews can lead to false diagnostic results (see Meyer et al., 2001, for a review) and provide insufficient information to rule out a disorder (see Culpepper & Mattingly, 2010, for a review). Furthermore, it has been concluded that they are poor indicators of ADHD with a large number of false positives (McCann & Roy-Byrne, 2004). This becomes apparent in research using symptom rating scales only. They indicate that up to 39% of the questionnaires used in clinical practice, miss the diagnosis rate of

ADHD (Kooij et al., 2008). Considering this, it remains controversial if self-evaluations can serve as an inventory to determine a diagnosis of ADHD in adulthood alone.

One main reason for this is that they leave professionals dependent on the information of third parties and subject to many sources of errors. For example, adults with ADHD are typically considered to have poor self-reflection (Kooij et al., 2008). They often vary in the way they perceive their symptoms and differ in what they consider as clinically significant. In this context, the literature points out that adults with ADHD often underreport their symptoms and have a poor recall of retrospective symptoms. Sometimes, they appear too eager to achieve a diagnosis in order to explain daily life problems, which are unrelated to the condition (Jiang & Johnston, 2012; Kooij et al., 2008; McCann & Roy-Byrne, 2004; UK Adult ADHD Network, 2013; Zucker, Morris, Ingram, Morris & Bakeman, 2002). On the contrary, it has also been found that adults with ADHD were the best informants regarding their clinical symptoms (Kooij et al., 2008). Considering this, it may be hypothesised that diagnosing ADHD in adulthood solely on the ground of self-evaluation may result in under- or overestimation (Johnson & Conners, 2002; Weiss, M. & Murray, 2003). However, until now no concrete evidence for this has emerged (Kooij et al., 2010). One strategy to minimize sources of errors in the diagnostic process is to use the corroboration of other sources of information, for example objective psychometric techniques.

Standardised neuropsychological measures

Although the role of neuropsychological assessments in diagnosing ADHD in children is debated, neuropsychological data may be of particular relevance to the diagnostic process of ADHD in adults. Table 4 depicts selective examples of methods commonly used in research and clinical practice. In general, research holds up to the account that children with ADHD are more characterised by behavioural symptoms whilst adults with ADHD are considered to experience more decrements in cognitive functions (see Woods, Lovejoy & Ball, 2002, for a review). Despite the essence of cognitive inefficiency, there is yet no agreement of the utility of neuropsychological tests within the diagnostic process for ADHD in adulthood. Reasons for this may be related to the current understanding that neuropsychological tests are not conclusive for a diagnosis. Additionally, so far there are no guidelines to recommend neuropsychological information as collateral to draw diagnostic inferences (see Seixas et al., 2012, for a review). Consequently, the scientific literature advises that a diagnosis of ADHD in adults should not be made on the basis of this information alone and only if the diagnostic criteria are fulfilled (see Seidman, 2006, for a review).

Notwithstanding the limited utility in reaching diagnostic conclusions, authors in the field

of adult ADHD tend to advocate the idea of screening for neuropsychological functions to complement the diagnostic process (Ramsay, 2010). Accordingly, the mere documentation of symptoms and the developmental history that determines whether the adult meets the criteria for ADHD is only one part of the clinical evaluation. In addition to screenings and diagnostic interviews, it is suggested to apply psychometrically sound tools. They are suggested to address facets that are not of behavioural means and not specifically emphasised in the ICD-10 or the DSM-V (Surman, 2013). In this context, it is proposed that neuropsychological assessments will be most sensitive using a collective approach, that is, the incorporation of multiple procedures (see Woods, Lovejoy & Ball, 2002, for a review). In line with the view that ADHD in adults is very heterogeneous, a battery of tests may therefore be particularly helpful in the identification of cognitive strengths and weaknesses, and the integral components of ADHD related symptoms. Such information may lead to more realistic achievements that can be expected from an intervention (see Seidman, 2006; Woods, Lovejoy & Ball, 2002, for reviews). Despite these benefits, the discriminant validity of neuropsychological assessments still remains debated in the diagnostic process of ADHD. Research on children with ADHD indicates that the tests are limited in their discriminant validity but that, in fact, multiple tests increased the prediction status of ADHD (Doyle, Biederman, Seidman, Weber & Faraone, 2000). In adults, the sensitivity of neuropsychological tests has been low although the group differences to controls were significant. Therefore, they have not been considered useful to reliably discriminate between adults with ADHD and other psychiatric disorders (Holst & Thorell, 2013). Other studies also report poor accuracy rates and many false positive errors (Walker et al., 2000; Woods, Lovejoy & Ball, 2002).

Table 4. The assessment process for diagnosing ADHD in adults

Method	Objective	Procedure
Anamnesis	Exploration of developmental history	Establishment of a preliminary case history
Screenings	Application of self-report and/or informant screenings to assess the presence of ADHD related symptoms	e.g. ASRI-4 ^a , ASRS ^b , CAARS ^c , HASE ^d ,

Table 4. (continued)

Behaviour ratings and clinical interviews	Positive screens should be followed by full diagnostic assessment including standardised behavioural ratings and clinical interviews to evaluate current life-situation, clinical history, functional impairment, and comorbidity	e.g. ADHS-E ^e , BADDSt, CAADID ^g , DIS-L ^h
Psychometric assessment	Objective evaluation of neuropsychological functions with psychometric procedures	e.g. CPT ⁱ , Stroop-Task ^j , TAP ^k , TMT ^l , WAIS ^m

^aAdult Self-Report Inventory-4 (Gadow, Sprafkin & Weiss, 1999), ^bAdult ADHD Self-Report Scale Symptom Checklist (Kessler, Adler, Ames, Demler, et al., 2005), ^cConners' Adult ADHD Rating Scales (Conners et al., 1999), ^dHomburger ADHD-Scales for Adults (Rösler, Retz-Junginger, et al., 2008), ^eADHD Screening for Adults (Schmidt & Petermann, 2009a), ^fBrown Attention-Deficit Disorder Scales (Brown, 1996), ^gConners Adult ADHD Diagnostic Interview for DSM-IV (Epstein, Johnson & Conners, 2001), ^hStructural Diagnostic Interview Schedule (Robins, Helzer, Croughan & Ratcliff, 1981), ⁱContinuous Performance Test (Rosvold, Mirsky, Sarason, Bronsome & Beck, 1956), ^jStroop-Task (Golden, 1978), ^kTest of Attentional Performance (Zimmermann & Fimm, 2009), ^lTrail Making Test (Reitan, 1992), ^mWechsler Adult Intelligence Scale (Wechsler, 2008).

2.2 Neurological disorder with central deficits of attention (stroke)

As defined by the World Health Organization (1978), a stroke is an acute “neurological deficit of cerebrovascular cause that persists beyond 24 hours or is interrupted by death within 24 hours”. It leads to neuronal death and neuropsychological dysfunctions due to an abnormal cerebral blood circulation, originating either from ischemic or haemorrhagic cause. Worldwide, stroke is considered as the leading cause of long-term disabilities and death (see Feigin, Lawes, Bennett, Barker-Collo & Parag, 2009, for a review). Based on epidemiological research, it is the most common occurring neurological condition and the main cause of death in Germany, with approximately 196,000 first-ever strokes and 66,000 recurrent strokes diagnosed annually. Most commonly, strokes occur in the elderly, with approximately 50% of first-ever strokes in individuals above the age of 73 years, in Europe (Heuschmann et al., 2010). In the United States, the mortality rate is doubled in every 10 years between the ages of 55 and 85 years (American Heart Association, 2010). In terms of gender differences, the incidence rates of stroke in men are higher than in women whereas, in total numbers, more women suffer from stroke because of their increased life expectancy. Currently, 2% to 5% of the total health costs are caused by patients with a stroke in western countries. The total economic costs of first-ever ischemic strokes health care has been estimated to be 108 billion Euros for the next 20 years in Germany (Heuschmann et al., 2010). Despite of increasing incidence rates, long-term disabilities, and enormous costs stroke remains the least researched and funded disorder (American Heart Association, 2010). In order to better comprehend

the clinical picture of a stroke, the next section provides information to its pathogenesis and clinical symptoms. Moreover, an overview of the current state of research on neuropsychological outcomes is portrayed and the assessment process of stroke patients is addressed.

2.2.1 Definition of stroke

Stroke is a heterogeneous neurological disorder and occurs due to an acute focal disruption of blood flow and transportation of oxygen in the brain. The most common types of stroke are, ischemic (80%) and haemorrhagic (15%) strokes (Schubert & Lalouschek, 2006). Since ischemic stroke is the focus of the present research, haemorrhagic and other insults are neglected. A concise view on the pathogenesis of ischemic stroke follows, with an excursion to brain laterality to introduce the association between stroke localization and behavioural impairment. An overview of the clinical characteristics of stroke, including risk factors, neurological and behavioural symptoms is provided.

Pathogenesis

An ischemic stroke occurs from an interruption or absent blood flow in the cerebral arterioles and capillaries due to the formation of blood clots, as a result of plaque, a build-up of fatty deposits or other material (= thrombus, embolus). At the infarct core cellular death occurs due to a deprivation of oxygen and leads to permanent neurological injury. Hours to days following the infarct, secondary ischemic cascades of biochemical reactions may cause brain swelling and additional neuronal death. Next to many other causes, the most common pathological condition facilitating thrombotic artery occlusion is atherosclerosis⁵ and embolization is mostly provoked by atrial fibrillation⁶. Depending on the location in the brain, a thrombus can block large blood-supplying arteries (e.g. middle cerebral artery; MCA) or small penetrating cerebral arteries or arterioles. Ischemic stroke resulting from embolization most frequently reaches superficial branches of cerebellar/cerebral arteries (Hacke, 2010). The MCA is considered the most commonly affected artery by ischemic stroke (50.8%) because of its large vascular distribution (Ng, Y. S., Stein, Ning & Black-Schaffer, 2007). Therefore, it is additionally associated with greater risk of causing cognitive dysfunctions (Jaillard, Grand, Le Bas & Hommel, 2010). Neuroanatomically, the MCA is divided into four segments (M1 to M4) and supplies large portions of the right and left hemisphere

⁵ *Atherosclerosis* refers to the narrowing and hardening of arteries due to an accumulation of plaque (e.g. fat, cholesterol) builds up in its walls which in turn limit blood flow (Hacke, 2010).

⁶ *Atrial fibrillation* denotes an irregular heart contraction which increases the risk of stroke due to the formation of blood clots through turbulent blood flow in the heart chambers (Hacke, 2010).

cortex and subcortical regions. Roughly embracing the specific neuroanatomic sites, the MCA supplies the inferior and lateral frontal lobes, anterior lateral area of the parietal lobes, superior parts of temporal lobes, and the insula. In addition, deeper brain areas, such as the posterior limb of the internal capsules, the basal ganglia, the putamen, and the caudate, external globus pallidus are supplied as well. These areas have important roles for functional and cognitive abilities. For example, for motor functions (e.g. primary motor cortex), sensation (e.g. primary somatosensory cortex; parietal lobe), eye movement (e.g. frontal eye fields), language production (e.g. Broca's area; left frontal lobe), language comprehension (e.g. Wernicke's area; left temporal lobe), perception (e.g. lateral frontal, parietal lobes; right hemisphere), visuo-spatial perception (e.g. posterior parietal lobe; right hemisphere), and the visual field (e.g. optic radiations, striate cortex; parietal/temporal lobes) are just some of many to mention (Bradac, 2011; Hacke, 2010).

Brain laterality

The understanding of the brain's functional localization supports clinicians work in diagnosis and therapy. Many studies that involve patients with a stroke address the associations between localization and behavioural impairment. It has been suggested that a stroke can cause behavioural impairment with predictable regularity depending on its exact location (Lezak et al., 2004). The most consistent findings have been reported for the asymmetry of language and visuo-spatial functions (Hugdahl, 2000). This means, that a stroke occurring in the right hemisphere (RHS) usually causes impairment involved with right hemisphere functions (e.g. visuo-spatial deficits) and a stroke occurring in the left hemisphere (LHS) leads to deficits related to left hemisphere functions (e.g. language; Pulsipher, Stricker, Sadek & Haaland, 2013; Wilde, 2010). The roots of this research date back to the evolution of theories on the localization of brain functions by Gall and Spurzheim (1810-1819) and Brodmann (1909) in the 19th century. They have identified areas in the brain to designate distinctive and specific cerebral and cortical functions. Preceded by the former, Paul Broca (1863, 1865) has localised brain lesions responsible for aphasia and suggested a language specialization of the left hemisphere. In addition, the account of modularity by Fodor (1983), which indicates that mental abilities arise from multiple distinct processes, influenced much of the later research on brain-behaviour relationships. Grounded on these theories and on decades of research, it is generally assumed that the modularity of the different brain systems is relatively stable⁷ and that impairment results by means of regional differences in the brain.

⁷ It is proposed that the assumption of stable modularity of cognitive systems does not hold for children who are still developing. For more information please refer to Mrakotsky (2007).

Although some discontinuities between behavioural and neurological data may exist, the dominant tendencies for a functional organization of the adult brain cannot yet be negated (Lezak et al., 2004). At present, the general assumption that the adult brain is functionally organised is furthermore extended by the notion that the different areas of the cerebral cortex and of subcortical regions are spatially distributed, but not operating independently. Rather, they are functionally linked and connected in a complex integrative network where information is constantly processed and shared (see Sporns, Chialvo, Kaiser & Hilgetag, 2004; van den Heuvel & Hulshoff Pol, 2010, for reviews).

Risk factors

Several factors contribute to an individual's risk to suffer an ischemic stroke. These can be clustered into nonmodifiable or modifiable risk factors, which means they are either predetermined or can be changed. To illustrate, gender, age, low birth weight, and genetic predispositions account to nonmodifiable factors. In terms of gender and age, it has been found that males have a higher risk to suffer of an ischemic stroke than women. Increasing age (≥ 55 years) has been reported to substantially heighten the risk for a stroke by doubling its rate each 10 years (see Goldstein, L. B. et al., 2006, for a review). With respect to modifiable factors, studies have suggested that a high blood pressure increases the risk for an ischemic stroke. Moreover, smoking has been indicated to double the risk of a stroke by facilitating the development of atherosclerosis and the formation of thrombi. Additionally, diabetes has been identified as an independent effect for an ischemic stroke through an increased risk of atherosclerosis and arterogenic factors (e.g. hypertension, abnormal blood lipids, and obesity). It has furthermore been found that patients with persistent or paroxysmal atrial fibrillation have a three to four fold increased risk of an ischemic stroke. Since the prevalence of atrial fibrillation increases with age, about one quarter of the stroke patients older than 80 years can be referred to this risk factor. These strokes are particularly large and associated with a great impact on functional outcomes. In addition, non-cerebrovascular atherosclerotic vascular disease (e.g. cardiovascular disease) heightens the risk for an ischemic insult. At last, dietary factors, obesity, and physical activity have been linked to an ischemic stroke. Diets rich in vegetables, fruit, and low-fat reduce the risk while high levels of sodium, body fat, and overweight (Body Mass Index ≥ 25) increase the risk of a stroke. Regular physical activity has been indicated as a protective factor by controlling other aspects, such as weight, diabetes, and cardiovascular disease (see Goldstein, L. B. et al., 2006, for a review).

Motor and sensory impairment

Depending on the brain areas and the side affected by a stroke, diverse deficits and disabilities in different severities may occur. Motor impairment following stroke relates to weaknesses and paralyses of diverse body parts. Frequently, patients experience deficits such as sudden contralateral signs of paralysis in their limbs or parts of their body (= hemiparesis, hemiplegia, and faciobrachial paresis). These deficits have mostly been related to damage in the motor cortex. In addition, sensory abnormalities may be caused, such as hemisensory loss (= hypesthesia) or involuntary movements. Subcortical lesions involving thalamocortical radiations, which are the fibres connecting the cerebral cortex and the thalamus, can also lead to sensory impairment (Kumral, Topcuoglu & Onal, 2009).

Visual field and language deficits

An ischemic stroke in the MCA territory may obstruct the transmission of information to the occipital cortex and its subsequent interpretation. This may lead to a loss of the visual field (= hemianopsia). Depending on the exact location of the occlusion, different parts of the visual field in each eye may be missed, including the same, the right or left half of each visual field (= homonymous, right or left homonymous), the outer half of each visual field (= bitemporal), or the upper or lower half of each visual field (= superior, inferior). In addition to hemianoptic deficits post-stroke, are the impairments of language functions (= aphasia). Since approximately 95% of the population have their dominance for language functions in this hemisphere, principally regardless of their handedness, aphasia is the most frequent result after LHS (Hernandez-Cardenache & Johnson-Greene, 2013; Kumral et al., 2009). Given that there is considerable variability in the symptoms of language impairments, different forms of aphasia can be distinguished. Aphasia is categorised into non-fluent (e.g. global, transcortical motor, mixed transcortical, and Broca's) and fluent forms (Wernicke's, transcortical sensory, anomia, and conduction), of which the former are characterised by a lack of expressive ease or verbal facility and the latter are marked by deficiencies in auditory comprehension and repetition (Hernandez-Cardenache & Johnson-Greene, 2013).

Hemispatial neglect and anosognosia

Patients with hemispatial neglect lost the ability or have an impaired capability to direct their attention to events towards the contra-lateral side of the stroke (Buxbaum et al., 2004). Next to a range of aetiologies, hemispatial neglect originates from a stroke to either brain hemisphere. Most commonly and most severe, it occurs following RHS within the MCA territory. A reason for this is that the right brain regions are assumed to be primarily involved in abilities of attention,

memory, and perception (Kerkhoff, 2001; Ringman, Saver, Woolson, Clarke & Adams, 2004). It has been suggested that they are responsible to mediate and monitor the distribution of attention and to shift attention to both sides of the visual field. In contrast, the left hemisphere is restricted to its contra-lateral visual field and cannot compensate for these functions once the right hemisphere has been damaged. Therefore, RHS more commonly leads to severe contra-lateral spatial neglect (Mesulam, 2000). In addition, it has been noted that neglect following stroke is associated with a substantial capacity for recovery (Ringman et al., 2004). Despite good recovery rates, neglect accounts for the strongest predictors of general functioning post-stroke. It has been indicated to obstruct abilities which are essential for active engagement in rehabilitation (Nijboer, Kollen & Kwakkel, 2013). These abilities include cognitive functions, which have been found twice as high impaired in patients with neglect than in patients without (Lindén, Samuelsson, Skoog & Blomstrand, 2005). Sometimes also categorised into the spectrum of neglect is anosognosia, a self-awareness disorder, which is most frequently caused by RHS. It refers to a state in which patients lack awareness or deny the existence or the severity of motor, sensory, perceptual, behavioural or cognitive disabilities (Orfei, Caltagirone & Spalletta, 2009).

Emotional disturbances

Following a stroke, emotional disturbances and other neuropsychiatric symptoms may occur. Often, right MCA stroke is associated with emotional change, bluntness, and deficits in emotional awareness (Paradiso, Anderson, Boles Ponto, Tranel & Robinson, 2011). The reason for this is not clear, but it may have something to do with the right hemisphere being associated with the experience of emotions (Schwartz, G. E., Davidson & Maer, 1975). Particularly, apathy has been linked to a right MCA stroke and consequent damage to brain tissue in the insula, the basal ganglia as well as subsequent modification of brain activity in limbic and paralimbic areas (Paradiso et al., 2011). In general, emotional alterations after a MCA stroke are often clustered into symptoms for a major depressive disorder (Paradiso et al., 2011). According to the ICD-10, no universal diagnostic criteria for a post-stroke depression (PSD) exist. Internationally, diagnostic inferences are largely drawn from the DSM-IV (e.g. mood disorder due to a general medical condition). With the ICD-10, a PSD may be determined if the criteria for a major depressive disorder (F 32.0-2) or Dysthymia (F 43.1) have been met (Dohmen et al., 2006). Generally, it is assumed that a PSD develops within the first four to eight weeks after stroke onset (Aström, Adolfsson & Asplund, 1993; Ayerbe, Ayis, Wolfe & Rudd, 2013; Berg, Palomäki, Lehtihalmes, Lönnqvist & Kaste, 2003). Its identification is important since it has been associated with a lower quality of life, increased dis-

bility, enhanced cognitive impairment, and mortality (see Robinson & Spalletta, 2010, for a review).

The aetiology of a PSD is suggested to be multifactorial⁸. Often, the association between the stroke location and a PSD has been discussed to delineate a pathophysiological explanation. On the one side, it is assumed that the risk to develop a depression is linked to RHS (Paradiso et al., 2011; Schwartz, J. A. et al., 1993) or to the LHS regions (see Bhogal, Teasell, Foley & Speechley, 2004; Robinson & Spalletta, 2010, for reviews). On the other side, often no differences between RHS and LHS have been found (see Ayerbe et al., 2013; Carson et al., 2000, for reviews). An attempt to explain such divergent findings resulted in the argument that laterality effects are only evident during the acute stage and not months or years post-stroke. Thus, it is assumed that a LHS leads to a major depressive disorder in acute stages and a RHS to a delayed occurrence of a minor depression. A reason postulated is that RHS is assumed to determine a decrease of noradrenalin and serotonin, and in turn the upregulation of serotonin receptors. Such upregulation probably does not occur after LHS since there is a lesser decrease of noradrenalin and serotonin (Chemerinski & Robinson, 2000). Similar approaches postulate that a stroke obscures serotonin mechanisms and, hence, determines a PSD (Kohen et al., 2008).

2.2.2 Current state of research: Neuropsychological functions post-stroke

There is considerable literature on neuropsychological functions following stroke with consistent reports of high levels of cognitive sequelae. These deficits have been found eminently heterogeneous and constantly changing during recovery. The frequency of cognitive impairment at the acute stage was reported as high as 78% (Leśniak et al., 2008; Nys et al., 2007). At least three to 15 months post-stroke, cognitive dysfunctions or a worsening of deficits were found in up to 73% of the patients (Ballard, Rowan, Stephens, Kalaria & Kenny, 2003; Ballard et al., 2002; Leśniak et al., 2008; Planton et al., 2012; Zinn et al., 2004), with continuous lasting impairment for up to three years in one-third of the survivors (Patel, Coshall, Rudd & Wolfe, 2003). Recently, population-based data collected five years post-stroke indicated that still 30% to 50% of the stroke survivors performed below average on standardised cognitive assessment measures (Barker-Collo, Feigin, Parag, et al., 2010; Barker-Collo et al., 2012). Moreover, diffuse and generalised profiles of cognitive dysfunctions across many domains compared to healthy adults have been identified (Hostenbach, Mulder, van Limbeek, Donders & Schoonderwaldt, 1998). Specific cognitive patterns

⁸ Multifactorial indicates that a PSD may be determined through biological and environmental factors. Please refer to the review of Spalletta et al. (2006) for more information on other aetiological aspects than lesion location.

of disturbances have sometimes been reported in association to clinical aspects of the stroke (e.g. lesion size, laterality; Laures-Gore, Marshall & Verner, 2010; Lindén et al., 2005; Montour-Proulx et al., 2004; Wilde, 2010). Furthermore, cognitive impairment has been linked to stroke-related symptoms (e.g. depression, aphasia, motor deficits; Ayerbe et al., 2013; Planton et al., 2012; Pulsipher et al., 2013; Robinson & Spalletta, 2010; Yang, S. et al., 2013), with the question whether cognitive sequelae are deficits of their own or a consequence of these symptoms (Planton et al., 2012). Together, studies have suggested that deficient cognitive functions across different domains post-stroke are associated with higher mortality and health care costs (Claesson, Linden, Skoog & Blomstrand, 2005; Tatemichi, Paik, et al., 1994). Moreover, they predict long-term disability, functional recovery, and rehabilitation success (Barker-Collo, Feigin, Parag, et al., 2010; Heruti et al., 2002; Nys, van Zandvoort, de Kort, van der Worp, et al., 2005; Zinn et al., 2004).

A review of scientific studies on neuropsychological deficits post-stroke is outlined in the following section. Table 5 summarizes the present research findings for each of the cognitive domains. Due to the complexity of neuronal networks, the narrowly intertwined connectivity of the brain regions, and a lack of control on factors affecting neuropsychological functions, most of the published research relevant for the present thesis is based on heterogeneous information. This concerns data on distribution (e.g. anterior, frontal, temporal), neuroanatomical location (right, left hemisphere, subcortical, cortical involvement), and aetiological causes (e.g. middle, anterior, posterior cerebral artery stroke) of the strokes. Therefore, it is difficult to review homogenous findings regarding stroke type. In order to assure at least some degree of homogeneity, this review consists of a selection of relevant studies largely including patients with a first-ever, unilateral ischemic stroke at different stages of recovery. As such, the review differentiates between patients in acute care and those in rehabilitation. The present review is not exhaustive, but serves to illustrate the most important research findings to consider the full breadth of neuropsychological deficits post-stroke.

Attention in patients with a stroke

Accounting for the most prominent impairment of a stroke are the deficits of attention (Hostenbach et al., 1998), which have been linked to motor recovery and functional status two years post-stroke (Robertson, Ridgeway, Greenfield & Parr, 1997). At the acute stage, the levels of attention deficits were estimated between 25% and 92% (Barker-Collo, Feigin, Lawes, Senior & Parag, 2010; Hyndman, Pickering & Ashburn, 2008; Stapleton, Ashburn & Stack, 2001). With only limited recovery during 12 months after discharge (Hyndman et al., 2008), attention deficits have

been indicated as the most frequently affected cognitive function one year post-stroke (54%; Leśniak et al., 2008). There are many studies that indicated a wide array of attentional dysfunctions, including attentional switching, divided-, and selective attention (Hyndman et al., 2008; McDowd, Filion, Pohl, Richards & Stiers, 2003). Particularly, medium to large effects for continuous attention (Planton et al., 2012) and high levels of sustained attention deficits were found (Barker-Collo, Feigin, Lawes, et al., 2010; Hyndman et al., 2008; Pendlebury, Cuthbertson, Welch, Mehta & Rothwell, 2010). The highest frequencies of deficits have been emphasised after RHS (Robertson et al., 1997). This finding can be confirmed by functional imaging studies associating attention with frontal lobe and right hemisphere functioning in healthy individuals (Lewin et al., 1996; Müri et al., 2002; Pardo, Fox & Raichle, 1991). Some research has failed to provide evidence regarding lesion laterality of attention deficits involving stroke patients (Barker-Collo, Feigin, Lawes, et al., 2010; Hyndman & Ashburn, 2003). In terms of language deficits, LHS patients with aphasia have been found to perform worse on tasks measuring attention (e.g. vigilance, orientation) compared to a control group and LHS patients without aphasia. The size of the effects for these comparisons have been indicated as large (Pulsipher et al., 2013).

Executive functions in patients with a stroke

Depending on the definition of executive functions and the domains measured, deficits at the acute stage of stroke occurred in approximately in 19% to 75% of survivors (Leśniak et al., 2008; Zinn, Bosworth, Hoenig & Swartzwelder, 2007). Until four months after stroke, these deficits were estimated at 41% in the survivors and they regressed to nearly 3% after one year post-stroke (Leśniak et al., 2008; Pohjasvaara et al., 2002). Population-based evidence indicated that 30% to 50% of stroke patients perform significantly below average on tasks of executive functions five years after stroke (Barker-Collo, Feigin, Parag, et al., 2010; Barker-Collo et al., 2012). The executive domains mostly affected included initiation, abstract thinking, and response inhibition (Leśniak et al., 2008; Nys et al., 2007), flexibility (medium effects), problem-solving, and planning (medium to large effects; Planton et al., 2012; Pulsipher et al., 2013; Stricker, Tybur, Sadek & Haaland, 2010), interference, maintaining, and shifting of cognitive sets (Pohjasvaara et al., 2002). In terms of recovery, it has been suggested that especially deficits in executive functions have detrimental effects on functional outcomes (Leśniak et al., 2008). In this context, abstract reasoning was the least common function to recover six to ten months post-stroke compared to other (Nys, van Zandvoort, de Kort, Jansen, et al., 2005). Additionally, differences in executive functions of patients with LHS and RHS have been reported. While some studies found equally common im-

pairment in both groups (Pulsipher et al., 2013), others suggested that LHS strokes are more often predisposed to deleterious executive functions (Glosser & Goodglass, 1990; Nys et al., 2007; Vataja et al., 2003). Furthermore, large effects have been indicated for the performance of LHS patients with aphasia on tasks measuring executive functions (e.g. planning, problem solving, judgement, fluency) in comparison to a control group and to LHS patients without aphasia (Pulsipher et al., 2013).

Information processing speed in patients with a stroke

Next to attention abilities, research has emphasised processing speed as the main disturbed cognitive function post-stroke (Barker-Collo et al., 2012; Rasquin et al., 2004). Slow information processing was found in up to 70% of the patients two to four months post-stroke (Hostenbach et al., 1998). A recent population-based study reported severe deficits in up to 50% of the patients five years post-stroke. It was indicated that information processing speed is a major independent predictor for post-stroke functional outcomes (Barker-Collo, Feigin, Parag, et al., 2010). Marked deficits with large effects were particularly found on tasks with high attentional demands, such as 'Trail Making Test' and 'Digit Symbol Tasks' (Planton et al., 2012). On simple and complex reaction time tasks, right hemisphere patients have been reported to perform slower than controls and patients with LHS. Only with increasing tasks demands were LHS patients found deficient (Gerritsen, Berg, Deelman, Visser-Keizer & Jong, 2003). Recent evidence, however, indicated no difference in performance related to hemisphere of lesion for the domain of information processing speed (Barker-Collo et al., 2012; Planton et al., 2012). Large effects on the differences in processing speed performance have been reported between groups of LHS patients with aphasia, those without, and healthy controls. Here, the group of patients with aphasia has performed significantly worse (Pulsipher et al., 2013).

Working memory in patients with a stroke

Marked deficits have been indicated in verbal working memory functions (e.g. list learning, digit span) and spatial working memory abilities (e.g. figures, geometric stimuli) post-stroke. Particularly, large effects have been reported for free recall and verbal working memory. Medium effects have been found for recognition and cued recall (Planton et al., 2012). Proportions of impairment ranged between 23% and 55% three months and up to 36% one year post-stroke (Leśniak et al., 2008; Snaphaan & de Leeuw, 2007). It has been emphasised that working memory is the least affected ability compared to other cognitive functions two to four months post-stroke (30%; Hostenbach et al., 1998). Though others have suggested the opposite (88%; Jaillard, Naegele,

Trabucco-Miguel, LeBas & Hommel, 2009). After five years of stroke, population-based data reported no evidence regarding below average performance on verbal working memory tasks (Barker-Collo et al., 2012). Recovery rates were particularly poor for verbal working memory functions, especially after temporal lesions (Nys, van Zandvoort, de Kort, Jansen, et al., 2005). In terms of lesion laterality, past research indicated decreased performance in tasks with verbal working memory demands after right hemisphere damage compared to controls (Tompkins, Bloise, Timko & Baumgaertner, 1994; Tompkins, Scharp, Meigh & Fassbinder, 2008). However, in relation to patients with a LHS, the performance tended to be higher in verbal working memory (Laures-Gore et al., 2010; Pulsipher et al., 2013; Schouten, Schiemanck, Brand & Post, 2009). Some research found no differences between RHS and LHS groups but associated spatial working memory deficits with right hemisphere damage (Philipose, Alphs, Prabhakaran & Hillis, 2007). This finding is supported by functional imaging research suggesting right hemisphere involvement in spatial working memory and left hemisphere involvement in verbal working memory functions (D'Esposito et al., 1998). In terms of stroke related symptoms, it has been indicated that aphasia in LHS patients has an additional detrimental effect on verbal- and nonverbal working memory tests. Their performance has been reported significantly worse in comparison to LHS patients without aphasia and a control group. These effects have been reported in large ranges (Pulsipher et al., 2013).

Visuo-spatial functions and language in patients with a stroke

Other affected cognitive functions constitute visuo-spatial and constructional skills, which have been found impaired after both RHS and LHS. Typically, these deficits are observed in RHS patients two to four months after stroke onset (Hostenbach et al., 1998; Ryan, Bartels, Morris, Cluff & Gontkovsky, 2009; Tatemichi, Desmond, et al., 1994). Other research found visuo-spatial tasks sensitive but not specific to RHS. This limits the lateralizing characteristics of visuo-spatial tasks in stroke patients and suggests that both hemispheres may be involved in spatial processing (Nys et al., 2007; Pulsipher et al., 2013). A high prevalence of recovery in visual perception and construction six and ten months post-stroke has been indicated compared to other cognitive functions. The recovery of language was least common (Nys, van Zandvoort, de Kort, Jansen, et al., 2005). Particularly poor performance in language tasks (e.g. oral production, comprehension) after LHS compared to RHS has been reported (Nys et al., 2007; Pulsipher et al., 2013). Large effects have been found for the group differences between LHS patients with aphasia, those without, and

controls on language tasks. Thus, aphasia in LHS patients has been associated with detrimental effects on tasks with language demands (Pulsipher et al., 2013).

Intelligence in patients with a stroke

The literature on stroke reveals limited information about global intellectual functioning given that much emphasis has been placed on specific cognitive decline. With regard to studies on global intellectual functioning, below average scores of stroke patients compared to controls, with no differences regarding lesion location, have been reported (Zillmer, Waechtler, Harris, Khan & Fowler, 1992). Great discrepancies between the FSIQ and the GAI in patients with brain injuries have been found with a tendency for the GAI to be higher than the FSIQ (Harrison et al., 2008). In terms of lesion location, recent findings have indicated reduced performance following right MCA stroke on the Verbal Intelligence Quotient (VIQ), the Performance Intelligence Quotient (PIQ), and the FSIQ in comparison to controls (Paradiso et al., 2011). Earlier research concluded that RHS patients performed lower on the PIQ than LHS patients, and that the VIQ tapped left functions more than right (see Bornstein & Matarazzo, 1982, for a review). Verbal-performance discrepancy scores were within wide ranges (-10 to +10) for both RHS and LHS patients (Warrington, James & Maciejewski, 1986). More recent studies⁹ confirmed abnormal VIQ > PIQ after LHS, but concluded that discrepancy scores were relative insensitive to RHS. Most importantly, it has been recognised that VIQ-PIQ splits do not serve as predictive diagnostic marker of cerebral dysfunction for lateralised damage (Iverson, Mendrek & Adams, 2004; Ryan et al., 2009).

An overview of the research findings in patients with a stroke

Stroke patients in acute care experience a diffuse pattern of severe cognitive deficits. Despite some cognitive recovery during the first weeks post-stroke, a large number of patients exhibit continuous and lasting cognitive impairment during inpatient rehabilitation and up to five years after stroke onset. Table 5 briefly summarizes the current state of research in neuropsychological functioning of patients with a stroke.

Table 5. Current state of research of patients with a stroke

Attention	Attention deficits have been found the most common complaints post-stroke, with little recovery and persistent impairment for up to one year. A high rate of impairment is associated with RHS ^a and a predictor for negative functional outcomes.
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⁹ These studies involved a mixed group of patients with unilateral damage of different aetiologies (e.g. vascular, tumour, brain injury).

Table 5. (continued)

Executive functions	Consistent evidence supports impairment in executive functions, with relatively high frequencies up to five years post-stroke. Tendencies towards left hemisphere sensitivity to executive function deficits have been suggested.
Information processing speed	It is agreed upon a significant slowness of information processing post-stroke, with independent and major contributions to negative functional outcomes. No laterality effects for this cognitive domain have been indicated.
Working memory	There is evidence regarding verbal- and spatial working memory impairment post-stroke, however, there are inconsistent rates regarding frequencies of these deficits. Most research associates RHS ^a with spatial working memory impairment and LHS ^b with verbal working memory deficits.
Intelligence	There is limited data on global intellectual functioning post-stroke, however, earlier studies reported below average intelligence scores. Laterality effects have consistently been found for the VIQ ^c and the PIQ ^d .
Visuo-spatial functions and language	There is inconsistent evidence regarding laterality effects in visuo-spatial abilities. Typically, RHS ^a are associated with visuo-spatial impairment. Language deficits are primarily linked to LHS ^b , with little rates of recovery. Aphasia in LHS ^b patients has been associated with detrimental effects on language tasks and other cognitive tests.

^aRight hemisphere stroke, ^bleft hemisphere stroke, ^cVerbal Intelligence Quotient, ^dPerformance Intelligence Quotient.

2.2.3 *The assessment process in stroke rehabilitation*

Quantifying stroke patients' health status with standardised assessments is recognised as a fundamental part of the therapeutic process in acute care as well as in inpatient rehabilitation (Bland et al., 2013). Next to a functional evaluations¹⁰, assessments of cognitive functions have been indicated as essential (Heruti et al., 2002). Given that the neuropsychological sequelae of stroke is highly heterogeneous and constantly changing during recovery (see section 2.2.2), its assessment encompasses many different approaches and varies across the different phases of rehabilitation. For example, in acute care it is the priority to prevent sub-acute medical complications and to restore neurological functions. Hence, the focus of assessment is more on general health functions than on neuropsychological abilities. This is, because general health functions provide the best marker of prognosis in the acute phase and because the patients are physically and physiologically still incapable to endure neuropsychological assessments. As stroke survivors are beyond the acute phase, the priority of care turns towards the recovery of cognitive sequelae and their compensation. Thus, the focus of assessment shifts to a more thorough evaluation in order to

¹⁰ An analysis of a functional status is utilized to assess task-specific activities important for a patient's overall well-being, such as motor functions and mobility. Recommended assessment tools are among others the 'Barthel Index' (Duncan et al., 2005).

infer to daily activity levels, participation, and reintegration (Miller et al., 2010). Since the domain and the degree of neuropsychological impairments highly varies between the points of acute care and post-acute rehabilitation (e.g. due to spontaneous cognitive recovery), it is from utter importance to ascertain the presence of deficits at admission to inpatient rehabilitation. Reasons for an early implementation of neuropsychological assessments are to enhance clinical decision making, to determine realistic rehabilitation aims, appropriate treatment strategies, and to improve rehabilitation outcomes (Heruti et al., 2002). Given that the present investigation involves patients in the post-acute phase of recovery, the following overview outlines current practices of neuropsychological assessments in inpatient rehabilitation facilities. Table 6 displays the rehabilitation process of stroke patients using the six-phase model of the German Federal Rehabilitation Council (BAR; Bundesarbeitsgemeinschaft für Rehabilitation, 1995). Integrated in this model is an exemplary selection of common standardised neuropsychological assessment instruments utilised during the different phases of recovery.

Table 6. The rehabilitation process for patients with a stroke

BAR ^a	Classification	Level of independence	Assessment method	Procedure
A	Acute intensive medical treatment in stroke units or intensive care	Biological autonomy	Screenings	e.g. K-SNAP ^b , MMSE ^c , MoCA ^d , NIHSS ^e , NRS ^f , SIS ^g
	(A) Maintenance of vital functions			
B	(B) Early stages of rehabilitation involving medical therapeutic options to maintain vital functions and biological autonomy (e.g. ventilation)			
C	Outpatient/inpatient rehabilitation after discharge from acute care	(C) Slow, vegetative, instable, mobilisation obstructed	Screenings, standardised neuropsychological instruments	e.g. Boston Naming Test ^h , MMSE ^c , MoCA ^d , NCSE ⁱ , NRS ^f , TMT ^j , WAIS ^k , WMS ^l
	(C) Curative care and high degrees of nursing to achieve functional autonomy			
D	(D) Follow-up treatments with the aim of social and occupational reintegration	(D) Medical stability, largely independent, self-care		

Table 6. (continued)

E	Recovery phase	(E) Outpatient treatments to achieve social and occupational reintegration	Social autonomy	n.a.	n.a.
F	Recovery phase	(F) Long term support and follow-up care to maintain functioning in specialists units			

Note. Modified from (Kaesberg, Fink & Kalbe, 2013) and (Ackermann & Schönle, 2012).

^aBundesarbeitsgemeinschaft für Rehabilitation, ^bKaufmann Short Neuropsychological Assessment Procedure (Kaufman & Kaufman, 1994), ^cMini Mental Status Examination (Folstein, Folstein & McHugh, 1975), ^dMontreal Cognitive Assessment (Nasreddine et al., 2005), ^eNational Institute of Health Stroke Scale (National Institute of Neurological Disorders and Stroke, n.d.), ^fNeurologischer Reha Score (Thilman, Nachtmann & Scharff, 2006), ^gStroke Impact Scale (Duncan et al., 1999), ^hBoston Naming Test (Kaplan, Goodglass & Weintraub, 1983), ⁱNeurobehavioral Cognitive Status Exam (Kiernan, Mueller, Langston & Van Dyke, 1987), ^jTrail Making Test (Reitan, 1992), ^kWechsler Adult Intelligence Scales (e.g. Wechsler, 2008), ^lWechsler Memory Scale (Wechsler, 2009b).

Screening techniques

In acute and sub-acute phases of the rehabilitation of a stroke, patients are still bedridden and severely impaired in their functional abilities. Therefore, neuropsychological assessment procedures are mostly limited to short screenings or remain incomplete. In these settings, screenings should be indispensable for initial profile analysis of cognitive impairment because they can be used as early warning systems about prognosis of post-hospital care. It is suggested that further comprehensive assessments are needed in post-acute stages of recovery in order to examine the patients' cognitive status thoroughly and to evaluate for neuropsychological syndromes (e.g. neglect). Such information is critical for drawing conclusions on patients' care, intervention planning, and recovery (Kaesberg et al., 2013). However, professionals oftentimes base their overall cognitive assessments on screening techniques. For example, the Mini Mental Status Examination (MMSE) is most routinely used on admission to rehabilitation (Heruti et al., 2002) and clinical practice guidelines recommend its implementation in stroke patients in inpatient rehabilitation facilities (Duncan et al., 2005). The accuracy of screening tests is, however, limited as they lack reliability. Information may not be well reflected and may lead to under- or overestimation of impairment. Moreover, they often misidentify deficits or let patients with deficits slip through the screen. Nevertheless, screening techniques should still be used with the knowledge of its limita-

tions and with the prospect of a further careful neuropsychological study to disentangle and identify underlying impairment and its severity (Lezak et al., 2004).

Standardised neuropsychological assessments

Recent approaches to stroke management demand early evaluations of neuropsychological deficits with reliable instruments in rehabilitation (Miller et al., 2010). The use of validated standardised measures of cognitive outcomes post-stroke is also suggested in the scientific literature (see Barker-Collo & Feigin, 2006, for a review). Thus, both in clinical practice as well as in research standardised neuropsychological assessments are recognised as an essential component for individuals with a stroke in inpatient rehabilitation facilities (Bland et al., 2013). Particularly, the use of comprehensive assessment batteries, which encompass a broad range of cognitive functions, are emphasised (see Barker-Collo & Feigin, 2006, for a review). The goal of a thorough neuropsychological examination post-stroke is twofold. The first is to determine the presence of impairment and the need for treatment. Since neuroimaging devices determine only an anatomical diagnosis of a stroke, neuropsychological assessments should be used to identify the nature of cognitive impairments and strengths, as well as to document those abilities that are inconsistent with findings from neuroimaging (Lezak et al., 2004). The second goal is to qualify and quantify deficits, to track changes, and adapt treatment procedures and intervention (Miller et al., 2010). By fulfilling these goals a variety of benefits are promoted. These include continuity of care, enhanced clinical decision making, and increased prognostic estimations. Thereby, the patients' potential to take active part in rehabilitation can be facilitated and the success rate of therapy may increase. Making clinical decisions without initial prognostic information is inefficient and very problematic (Bland et al., 2013; Heruti et al., 2002). Notwithstanding these benefits a routine use of standardised neuropsychological assessments in inpatient rehabilitation continuous to be challenging due to various reasons. These include low adherence rates of clinicians to cognitive assessment (Bland et al., 2013) and the lack of consensus on standardised instruments (Barker-Collo & Feigin, 2006; Gottesman & Hillis, 2010).

3 The present empirical investigation

The previous sections provided an overview of substantial research with inconsistent results regarding intellectual and cognitive functioning in adults with ADHD or stroke. Moreover, it was pointed out that neuropsychological assessments may play an important role in disentangling and identifying deficits in these areas, which may ultimately serve diagnosis and intervention. However, research on intellectual and cognitive functioning in adults with ADHD and in stroke patients in inpatient rehabilitation facilities has revealed some limitations and methodological issues. It is, thus, essential to improve our knowledge concerning these aspects. The following two sections describe the rationale and overall aims of the present investigation as well as the issues of previous research concerning adults with ADHD or stroke.

3.1 Rationale and aims of the studies on adults with ADHD

Prior research challenges the validity and accuracy of self-reported information and suggests that objective data on neuropsychological functioning adds valuable information to the diagnostic process and to inform treatment plans. The contribution of neuropsychological assessments to the diagnostic process is confined by various factors in scientific research. These include: (a) that no cognitive impairment has yet been identified as a specific marker for ADHD in adulthood. This implies that the absence of cognitive dysfunctions does not rule out a diagnosis whilst its presence does not guarantee one and/or might just as well be accounted for by other variables; (b) the application of single instruments measuring specific cognitive functions lose information and decreases sensitivity and negative predictive power since ADHD in adulthood is marked by a heterogeneous symptomatology causing a wide array of deficits; (c) comprehensive neuropsychological assessments encompassing many cognitive domains are not yet standardised and there is no consensus on appropriate assessment tools to evaluate the deficits in ADHD in adulthood. These three reasons limit the ability to draw firm guidelines and conclusions of neuropsychological assessments in clinical practice. Thus, there is ample need for consent on a tool with sufficient specificity and sensitivity to detect the cognitive deficits in adults with ADHD. In line with the view that ADHD in adults is very heterogeneous, a battery of tests can be essentially helpful in the identification of cognitive functioning. To this end, the current thesis seeks to examine the fundamental barriers to diagnosing ADHD in adults by addressing the identification of neuropsychological functions with

a battery-approach by means of the WAIS-IV. In addition, the status of self-evaluations for the diagnostic process is considered and it is discussed whether self-evaluations reflect the results found in objective psychometric tests. Accordingly, the global aims of the present research are threefold:

- Implementation of an objective approach that attempts to identify a general cognitive pattern of adults with ADHD by means of the WAIS-IV.
- Implementation of an objective approach to examine whether deficits found in the WAIS-IV are related to ADHD in adulthood or whether they actually form an artefact of comorbidity or medication at time of assessment.
- Implementation of a subjective approach that examines the status of self-evaluation in the assessment of ADHD in adulthood.

Methodological issues

For the above aims, the current research addresses some methodological difficulties present in prior studies, which may have caused conflicting findings and variability concerning neuropsychological performance in adults with ADHD. The first issue relates to the variability of neuropsychological assessment tools. Discrepant outcomes are likely to be due to considerable diversity of methods and differences in test utilization and administration. In this regard, there is a heavy use of tests across studies examining neuropsychological functions with limited power and sensitivity (e.g. screenings, short forms, flexible test-batteries). This may have contributed to an unclear pattern of effects, inaccurate conclusions, and may have further complicated the comparability of results. Moreover, many studies drew conclusions upon scores from single tests or a combination of individual subtests from a test battery (see Frazier et al., 2004, for a review). Therefore, the formal psychometric requirements for combining subtests and comparing their results may not have been met (e.g. test invariance, metric units, norms). Such lack of equivalence between tests may have constrained the validity of results. In order to overcome this, the current study addresses this issue by applying a psychometrically sound and fully standardised assessment battery (WAIS-IV). The second problem pertains to adequate statistical power. Many studies on neuropsychological functioning in adults with ADHD have a lack of statistical power due to small sample sizes and non-random group matching procedures. This may heighten the risk of Type II error and unreliable results on differences between patients and controls. A sufficiently large sample in accordance with a priori power analyses was included in the current research to derive more valid results.

Moreover, a random matching procedure was applied to add a control sample matched according to sex, age, and education. The third issue concerns the identification of adults with ADHD. As such, a potential source of error in prior studies may have been the considerable differences in the diagnostic procedures and the general lacking consensus of general diagnostic criteria. To ensure a clinically valid diagnosis of ADHD in adulthood, the present research made use of multiple sources of information in diagnostic decisions (e.g. clinicians' diagnosis, rating-scale based on ICD-10). The fourth issue relates to control variables on test performance. In terms of comorbidity it is debated to whether cognitive deficits are related to ADHD in adulthood or, in fact, are an artefact of comorbidity. Yet, only limited research addressed the influence of comorbidity in adults with ADHD at all, extensively or accurately. Regarding stimulants and other psychopharmacological medication intake at the time of assessment, it is indicated that they yield positive or negative effects on cognitive performance. Yet medication intake and long term influence on performance is not always considered in studies. As they may serve as possible confounders, the present research carefully controlled for the effect of comorbidity and medication status in the analyses and explicitly reported the levels of comorbid psychiatric conditions and medication.

3.2 Rationale and aims of the study on stroke

It has been claimed that the identification of neuropsychological deficits of stroke patients early at rehabilitation admission improves overall treatment success by tailoring therapeutic intervention to strengths and weaknesses of the patient. Despite its importance for functional outcomes, neuropsychological sequelae after stroke are often neglected in clinical practice, particularly at the point of rehabilitation. While neuropsychological outcomes post-stroke have been broadly discussed in the literature and dysfunctions have often been reliably linked to stroke, studies continue to provide a largely heterogeneous picture of cognitive sequelae post-stroke at the point of rehabilitation. Studies report diffuse cognitive profiles at different stages of post-stroke recovery and across various pathological stroke types with different aetiologies. Furthermore, clinical factors have been found that may be associated with these deficits. The heterogeneity of results may be attributed to different reasons: (a) only specific areas of neuropsychological functions were assessed and so far little has been done to employ a full standardised assessment battery to measure general cognitive functions in early stages of rehabilitation; (b) there is a lack of a standardised neuropsychological assessment instrument in scientific research and clinical practice. The present thesis addresses these issues by applying the following aims:

- Specifying the cognitive sequelae post-stroke by evaluating patients' cognitive status, their strengths and weaknesses at the point of rehabilitation with the WAIS-IV battery.
- Examining if language deficits, neglect, hemianopsia, and PSD have an additional effect on WAIS-IV performance in stroke patients.

Methodological issues

Working to achieve these aims, the current investigation addresses methodological issues of prior studies, which may underlie the variability in the literature. The first issue pertains to the variability of tests across studies to measure cognitive functions post-stroke. The inconsistency may be due to the lack of standardization in the selection of measures and of standardization of tests for stroke patients. Therefore, a large number of studies utilize tests from diverse sources. This means, that these tests may differ in their metric and evolve the issue of multiple comparisons and increases the probability of type I error. Moreover, there are still tests used without a metric, with no representative sample or normed at all. Since valid inferences regarding cognitive deficits are dependent on the degree of equivalence of the normative sample of the tests, the comparability of results may be increased and the type I error may be reduced by applying standardised measures with appropriate statistical methods (e.g. Bonferroni correction). The current investigation intended to make up for these shortcomings by means of a standardised test battery, the WAIS-IV. The second issue denotes inadequate statistical power. A large number of studies on cognitive functions post-stroke are limited by small sample sizes and/or a lack of a control group or inadequate matched controls. This increases the risk of type II error. In order to provide sufficient power to detect differences between the groups, the present research calculated the necessary sample size with a priori power analyses. Moreover, a random group matching procedure from a large pool of data provided a perfect match to the control group according to sex, age, and education. The third issue relates to the heterogeneity of the stroke sample. Inconsistencies may have resulted from grouping together different stroke types. In an attempt to draw more reliable inferences from the data, the current investigation included a more homogenous group of patients. The last issue relates to potential factors influencing neuropsychological performance. As such, stroke related variables including lesion characteristics, motor and language deficits or sensory impairment have often not been taken into sufficient account. The present investigation attempted to sufficiently describe and consider the impact of these variables in the interpretation of the results.

Empirical Research

4 Methods

The following part introduces the methods of the present research, including the design, sample selection, and the assessment. The central results of studies I, II, and III are presented. More detailed information, which is not fully considered in the following, can be found in the individual studies (see Appendix B to D).

4.1 Target population and sample composition

The first part outlines the demographic characteristics of both samples of the present investigation. Following this, the sample selection, recruitment, and composition are addressed separately. The individual recruitment procedures and the sample selection for adults with ADHD or stroke until the point of publication are depicted in a Consort-Flow diagram in Figure 2 and 4. Details on in- and exclusion criteria and specific clinical characteristics for each of the samples are provided. Concisely, the matching procedure of the control sample and its composition is outlined.

4.1.1 Demographic characteristics

In total, $N = 124$ adults with ADHD and $N = 112$ patients with first-ever unilateral, ischemic stroke underwent assessment. Details of demographic characteristics are portrayed in Table 7. At the time of assessment, the majority of patients lived in the northern regions of Germany (e.g. Bremen, Southern Saxony, North Rhine-Westphalia). Large differences in the male: female ratio indicates an unbalanced gender proportion for both groups. Higher rates of male ADHD cases are in accordance to scientific surveys and meta-analytic data (Kessler et al., 2006; Simon et al., 2009), however, not always significant (Zwaan et al., 2012). Additionally, more stroke incidents in men are in line with age-specific stroke rates (Heuschmann et al., 2010; Reeves et al., 2008). With respect to education levels, 47% adults with ADHD reported a school degree equivalent to 13 years of education (= Gymnasium) and 52% accomplished their degree in 9 (= Volks- Hauptschule) to 10 years (= Realschule). Although, these characteristics differ from some studies with much lower education rates (≥ 12 years of school (16%); Zwaan et al., 2012), they are largely consistent with those of others (≤ 11 years of school (16%), = 12 years (33%), ≥ 13 years (51%); Kessler et al., 2006). The educational degrees in the sample of stroke coincides with annular statistical reports involving elderly adults (≥ 65 years) in Germany (Arbeitsgruppe Bildungsberichterstattung, 2012). The current level of occupation for the samples (at the time of assessment) is categorised into

standardised codes of the International Labour Organization (2008). For patients with stroke, these levels are indicated for past and current occupations.

Table 7. Demographic characteristics of the total samples (*N*)

	ADHD	Stroke	
<i>N</i>	124	112	
Gender ratio (male: female), <i>n</i>	75:49	81:31	
Age, <i>M</i> , <i>SD</i> (min.-max.) <i>y</i>	37.9, 11.4 (17-71)	65.8, 11.4 (38-88)	
Educational Degree, <i>n</i> (%)			
Volks-Hauptschule ^a	18 (15.5)	76 (67.8)	
Realschule ^b	42 (36.2)	18 (16.1)	
Gymnasium ^c	54 (46.6)	15 (13.4)	
Still in school	2 (1.7)	-	
Special needs school	-	2 (1.8)	
no degree	-	1 (0.9)	
ISCO^d, <i>n</i> (%)			
	Current	Past	Current
ISCO 1-3 ^e	24 (20.7)	23 (21.5)	5 (4.6)
ISCO 4-5 ^f	24 (20.7)	15 (14.0)	12 (11.3)
ISCO 6-8 ^g	9 (7.8)	57 (53.3)	14 (13.1)
ISCO 9 ^h	2 (1.7)	3 (2.8)	1 (0.9)
Housewife	3 (2.6)	9 (8.4)	-
Retired/disabled/unemployed	28 (24.1)	-	75 (70.1)
Student/PhD	26 (22.4)	-	-
State of Living			
Bremen	74 (59.7)	1 (1.7)	
Southern Saxony	29 (23.4)	50 (86.2)	
North Rhine Westphalia	4 (3.2)	7 (12.2)	
Berlin	4 (3.2)	-	
Hamburg	6 (4.8)	-	
Other	7 (5.7)	-	

^a9 years of school, mandatory basic school, ^b10 years of school, intermediate type of advanced school, ^c13 years of school, high school equivalent, ^dInternational Standard Classification of Occupation (International Labour Organization, 2008), ^eoccupation with higher education (e.g. manager, health professionals, engineering associate), ^foccupation involving clerical support, service and sales (e.g. secretary, cook, service), ^goccupation involving agriculture (e.g. farmer, fisher), craft work (e.g. carpenter, bricklayer), and machine operators (e.g. truck driver), ^helementary occupations with no further education after school (e.g. cleaner, helper).

4.1.2 Sample of adults with ADHD

Studies I and III followed a quasi-experimental cross-sectional design with a non-probability sample of adults with ADHD. Their recruitment of the total sample, the study procedure, and the study sample characteristics' are as follows.

Recruitment and procedure

Initial power analyses were performed to calculate a sufficient sample size to test the hypotheses. A reasonable minimum desired effect size was set at medium ranges¹¹ and at significance levels of .05. This value indicates a sufficient degree of power to determine group differences with sample sizes of $N = 218$. Due to reasons of exclusion, an approximate 10% drop-out rate was estimated and acknowledged for sample recruitment. Therefore, more patients were assessed as indicated by power analyses. The recruitment took place between April 2012 and May 2013. One part of the data was provided by an outpatient psychiatric clinic where diagnoses and treatment for the adults have taken place or took place at the time of recruitment. During recruitment, 364 adults with an ADHD diagnosis received a letter with information regarding the present investigation. All patients had the voluntary option to respond to this letter and take part in this research. A second part of the data was retrieved via press releases in local newspapers. Suspected ADHD cases were screened on the telephone for eligibility according to a set of predefined inclusion criteria (ADHD diagnosis; ≥ 17 years), excluding those with intellectual disability and with a history of severe head injury. Medication and comorbidity were no reason for exclusion. All individuals who reported to be clinically diagnosed with ADHD in child- or adulthood were invited and scheduled for assessment. A copy of a clinical report for diagnostic verification was asked for. In addition to clinical reports, the diagnoses of all participants were verified with a diagnostic screening that provides diagnoses according to the criteria of the ICD-10. This screening was especially important for diagnostic confirmation of those participants who forgot their copy ($n = 11$). Prior to assessment, written informed consent was obtained from all participants. The assessment took place in a one-to-one setting in the premises of the University of Bremen or at the outpatient clinic. In terms of anonymity, each participant received a numerical code and his or her data was scored blindly. On request, the participants received their assessment results in form of a written report. Doing this, they voluntarily revealed their identity. Details of the recruitment procedure, assessment enrolment, and sample selection for data analysis are displayed in Figure 2. The total sample size differentiates between study I ($N = 123$) and study III ($N = 124$) because of ongoing assessments whilst the manuscript of study I had been completed and submitted for publication.

¹¹ Most prior research involving adults with ADHD indicates medium effect sizes for neuropsychological functioning. For details, please refer to section 2.2.2.

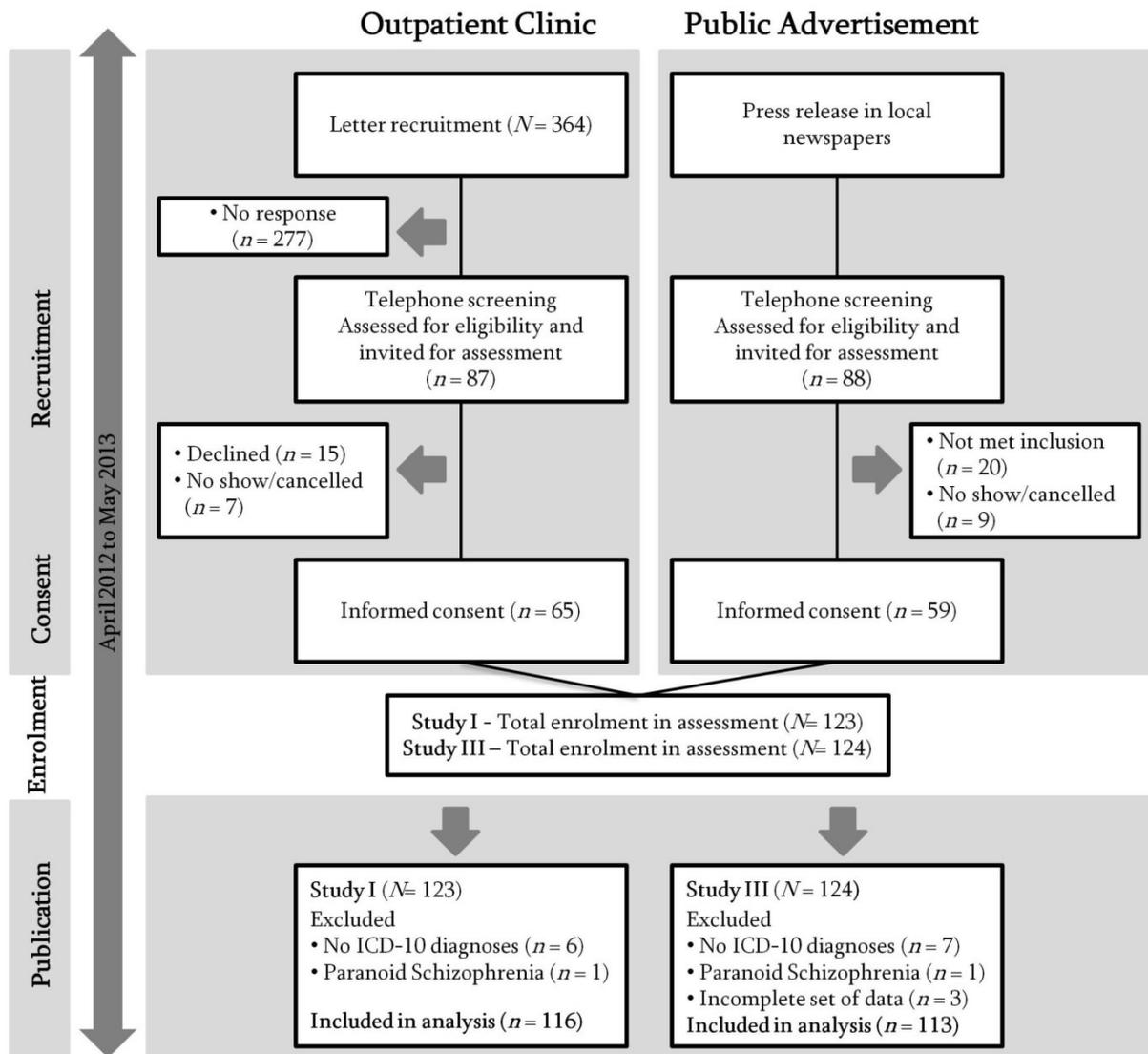


Figure 2. Consort-Flow diagram through the phases of recruitment and sample selection for data analysis for studies I and III.

Note. The difference in N between the studies is a result of on-going recruitment after the completion of the manuscript for study I. Therefore, the additional patient assessed was only included in study III. ICD-10, Internal Classification of Diseases – Tenth Edition.

Clinical characteristics

All participants included in the studies I and III met the ICD-10 criteria for ADHD. According to the DSM-IV-TR criteria, a substantial proportion of the patients fulfilled the criteria for ADHD combined type (85%). Of all the participants, 80% received a diagnosis of ADHD in adulthood (≥ 18 years) compared to the rest who were diagnosed with ADHD during childhood. In terms of comorbidity, 73% of the adults with ADHD had a least one comorbid condition. Figure 3 displays the percentage share of comorbid conditions in the sample. Moreover, prior to assessment the participants did not refrain from their medication treatment. That means that in both studies approximately 60% of the adults were under medical treatment in form of stimulants or antide-

pressants (e.g. Ritalin, Methylphenidate, Selective Serotonin Reuptake Inhibitor (SSRI), Tricyclic Antidepressants). For more detailed information please refer to Appendix B.

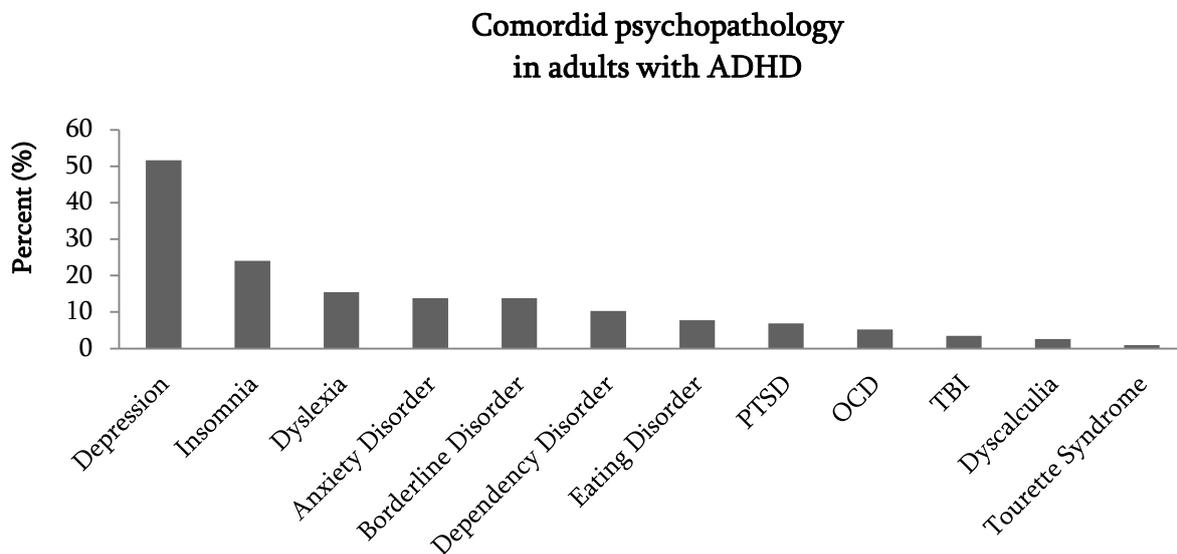


Figure 3. Percentage share of comorbid conditions in adults with ADHD.

OCD, Obsessive compulsive disorder; PTSD, Post traumatic stress disorder; TBI, traumatic brain injury.

4.1.3 Sample of ischemic stroke patients

The present research was confined to patients with a first-ever unilateral, ischemic stroke who had been admitted to inpatients rehabilitation settings. The study followed a quasi-experimental cross-sectional design with a non-probability sample. The recruitment of the total stroke sample, the study procedure, and the clinical characteristics are described in the following.

Recruitment and procedure

A priori power analyses estimated a total sample size to reveal medium to large effects¹² in the statistical analyses and at significance levels of .05. This value indicates a sufficient degree of power to determine group difference with sample sizes of $N = 172$. A 10% drop-out rate was estimated and acknowledged for sample recruitment thus more patients than indicated by the power analyses were recruited and assessed. The participants got recruited through two inpatient rehabilitation facilities between May 2012 and December 2012. Details on the process of pre-selection, recruitment procedure, assessment enrolment, and sample selection for data analysis of the published studies are displayed in Figure 4. A first pre-identification of patients fitting the inclusion

¹² Much of the previous research involving stroke patients indicate effect sizes in medium to large ranges for neuropsychological functioning. For more information please refer to section 2.2.2.

criteria was achieved with an electronic database, which contained anamnestic information, medical, and therapeutic records. Since the medical records, which were in all cases provided by the acute hospital were most often incomplete, the strokes could not be verified by neuroimaging data. Therefore, experienced neuropsychologists in charge of supervision for the present research confirmed the criteria for inclusion (see Table 8). This was done by means of clinical reports in the database and/or following consultation with neuropsychologists. Patients identified as eligible were visited by those responsible of the investigation and were informed about the study and asked for their interest to participate. Patients who agreed to participate were scheduled for evaluation. Each participant gave his or her written informed consent prior to assessment. Test administration took place in private rooms in the rehabilitation facility on average 15 days ($SD = 9$) after the patients' admission. This means, that on average the participants benefitted from two weeks of therapy prior to assessment. Therapeutic interventions were different for each patient but majorly included mobility training, computer-based therapy, occupational- and speech-language therapy, and healthy education. The next part provides a detailed overview of the clinical characteristics of the study sample by addressing the type of stroke, the time between first-ever stroke onset and assessment, as well as clinical symptoms and signs.

Table 8. In- and exclusion criteria for recruitment and sample selection of stroke

Inclusion	Exclusion
Ischemic causes of stroke	Impaired consciousness
First-ever stroke	Severe motor impairment
Unilateral	Severe auditory and/or visual deficits
Isolated MCA ^a territory infarct	Global aphasia, no means of communication
	Acute psychiatric illness
	Evidence bilateral, multiple stroke (preexisting cognitive impairment)

^aMiddle cerebral artery.

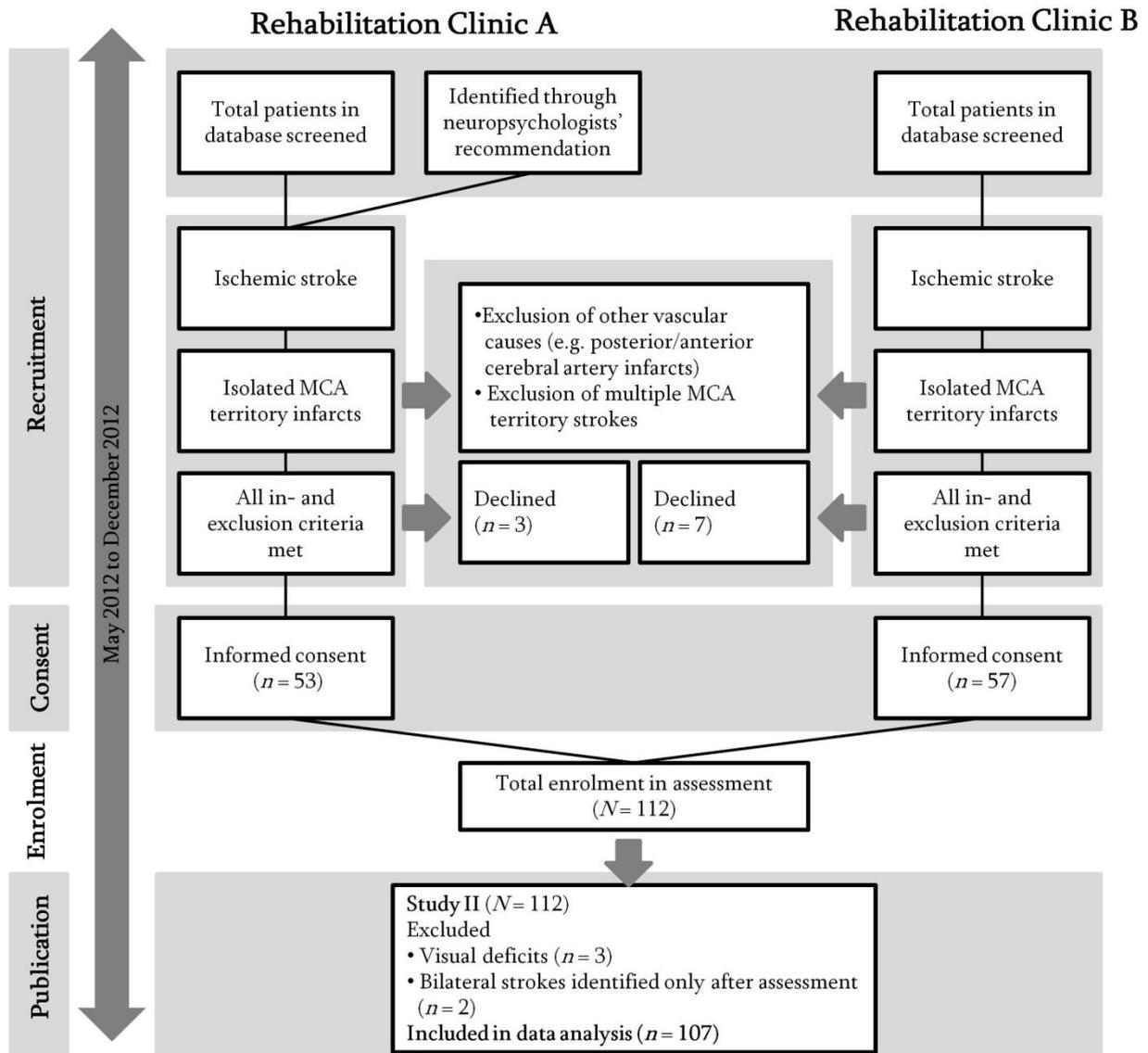


Figure 4. Consort-Flow diagram through the phases of pre-selection, recruitment, and sample selection for data analysis for study II.

MCA, Middle Cerebral Artery.

Stroke type

The current investigation focused on patients with first-ever unilateral, ischemic stroke following MCA occlusion. Ischemic causes and MCA occlusion were selected to derive conclusions from a homogenous group of patients. In order to obtain a fairly large study sample, ischemic stroke was chosen as it is considered the most frequent stroke type and MCA occlusion was selected as it presents the most commonly affected artery by ischemic stroke (see section 2.2.1). Moreover, choosing patients with a first-ever stroke raised the possibility to rule out prior stroke related sequelae, particularly preexisting cognitive impairment. Unilateral strokes were selected in order to derive conclusion on domain specific differences in cognitive functions and to rule out more severe sequelae due to bilateral infarcts.

Time since stroke

The majority of patients were assessed within one year post-stroke (87%), with an average time of 24 weeks between stroke onset and assessment. All of these participants were in rehabilitation phase D, which indicates a period in rehabilitation in which the patients are largely independent their actions, receiving only minimal amount of care (see Table 6). The rest of the patients (13%) had a stroke onset more than 1 year prior to assessment and received rehabilitation service due to persistent stroke related motor and cognitive deficits. Of these patients, none showed aphasic symptoms, neglect or hemianopsia. However, more than 50% of them received a diagnosis for PSD. The sample selected for study II consisted of $n = 58$ patients with RHS and $n = 49$ with LHS.

Clinical symptoms and signs

In terms of behavioural sequelae, some deserved specific focus during assessment. As such, it occurred that some patients needed to use their non-dominant hand to accomplish a task with motor demands if their dominant upper limb was affected by paresis (27%). In addition, patients with language impairment received particular simple and slow test instructions. In tasks with verbal demands, these patients were given enough time to accomplish the tasks and were encouraged to use other modes of communication. For example, patients who experienced problems with word finding or phrasing a word were encouraged to write down an answer, use their hands to point to objects or make signs. Confirmed by logopaedic records, none of the participants with language impairment had problems in comprehending speech. Some patients with LHS experienced deficits in language production, word finding, spontaneous speech or utterances of words. The classification of patients with language impairment was achieved by clustering them into two groups based on the general categorisation of the forms of aphasia (non-fluent vs. fluent, see section 2.2.1). One group consisted of LHS patients with fluent speech but trouble with word finding, thus, speaking around words ($n = 12$) and the other group of LHS patients with non-fluent communication due to impairment of spontaneous speech and utterances of words ($n = 17$). Moreover, 11% and 19% of the patients of study II demonstrated hemianopsia and neglect, respectively. This was determined by appropriate tests of the neuropsychologists at the rehabilitation facility. For more information on clinical sample characteristics please refer to Appendix C. Supplemental to the published studies, PSD is addressed in the current thesis. Since no ICD-10 criteria exist to draw post-stroke diagnostic inferences, a diagnosis was determined based on method commonly used in other studies (e.g. Ayerbe et al., 2013; Dohmen et al., 2006). Thus, a diagnostic category of

depression was accepted if (a) the stroke patients received an ICD-10 diagnosis of major depressive disorder (F32.0) or Dysthymia (F43.1) by the neuropsychologists in rehabilitation clinics and if (b) those patients without a diagnosis achieved a cut-off for abnormality on a standard depressive screening (see section 4.2.2). If data on the depressive status was missing or negative, patients were considered as not depressed. In total $n = 38$ patients met the criteria for a PSD. Of these patients meeting the criteria, $n = 11$ received antidepressants (e.g. SSRI).

4.1.4 Control group

The control group was selected from the German normative sample of the WAIS-IV ($N = 1,454$). The normative sample is representative for the German-speaking population of individuals between 16:0 and 90:11 years, stratified for sex, education, and geographical regions. For a detailed description of the sample please refer to (Petermann, 2012). The control samples for studies I to III were randomly drawn using Data Analysis and Statistical Software (STATA; StataCorp., 2013) and matched according to sex, age, and education. The large data pool of the normative sample ensured that each adult with ADHD and each patient suffering from stroke has a perfect random match. Thus, no statistical differences occurred between these variables.

4.2 Assessment

The assessment methods used for the present investigation included the WAIS-IV, which was administered in strict accordance to its standardised administration procedure by doctoral-level graduates and master students with extensive training. Standardised questionnaires were included in the assessment to determine diagnostic criteria and symptom severity. All tools included in the assessment were scored blind to the identity of the participants.

4.2.1 Wechsler Adult Intelligence Scale- IV

The WAIS-IV provides a normative sample of adults between 16:0 and 90:11 years and is considered a standardised instrument to examine intellectual and cognitive functions. The following lines outline the structure and the framework of the WAIS-IV and its psychometric properties. Furthermore, its clinical utility is addressed and its application in the present research is outlined.

Structure and test framework

The WAIS-IV was utilised to determine a psychometric cognitive profile for each participant. This profile included the calculation of global intellectual functioning (FSIQ) and specific cognitive functions represented by four indices: Verbal Comprehension Index (VCI), Perceptual

Reasoning Index (PRI), Working Memory Index (WMI), and Processing Speed Index (PSI) ($M = 100$, $SD = 15$). The four indices are composed of a sum of various subtest scaled scores ($M = 10$, $SD = 3$) and the FSIQ is derived from the sum of ten scaled scores. More information on the WAIS-IV structure and the test framework are depicted in Figure 5. Table A1 displays a description of each subtest, what it measures, its CHC ability, and cognitive capacities needed for efficient performance. Furthermore, essential information for the psychometric profile was added by the GAI ($M = 100$, $SD = 15$), an optional index for global intellectual functioning, solely composed on verbal and perceptual reasoning abilities. Thus, it is derived from the sum of scaled scores on the VCI and the PRI, excluding those of the WMI and the PSI (see Figure 5). The GAI may only be determined if the size of difference between the highest and the lowest index is larger than $1.5 SD$ (≥ 23 IQ points) and if the size of the standard score difference between the VCI and the PRI is less than $1.5 SD$ (≤ 23 IQ points). A further index standard score of the WAIS-IV is the Cognitive Proficiency Index (CPI), which reflects an individual's ability to process information. However, in the present research the CPI received no special attention. Additional focus was put on the calculation of scaled process scores ('Block Design No Time Bonus' [BDN], 'Digit Span Forward' [DSF], 'Digit Span Backward' [DSB], and 'Digit Span Sequencing' [DSS]). They are based on the performance on the corresponding subtests ($M = 10$, $SD = 3$).

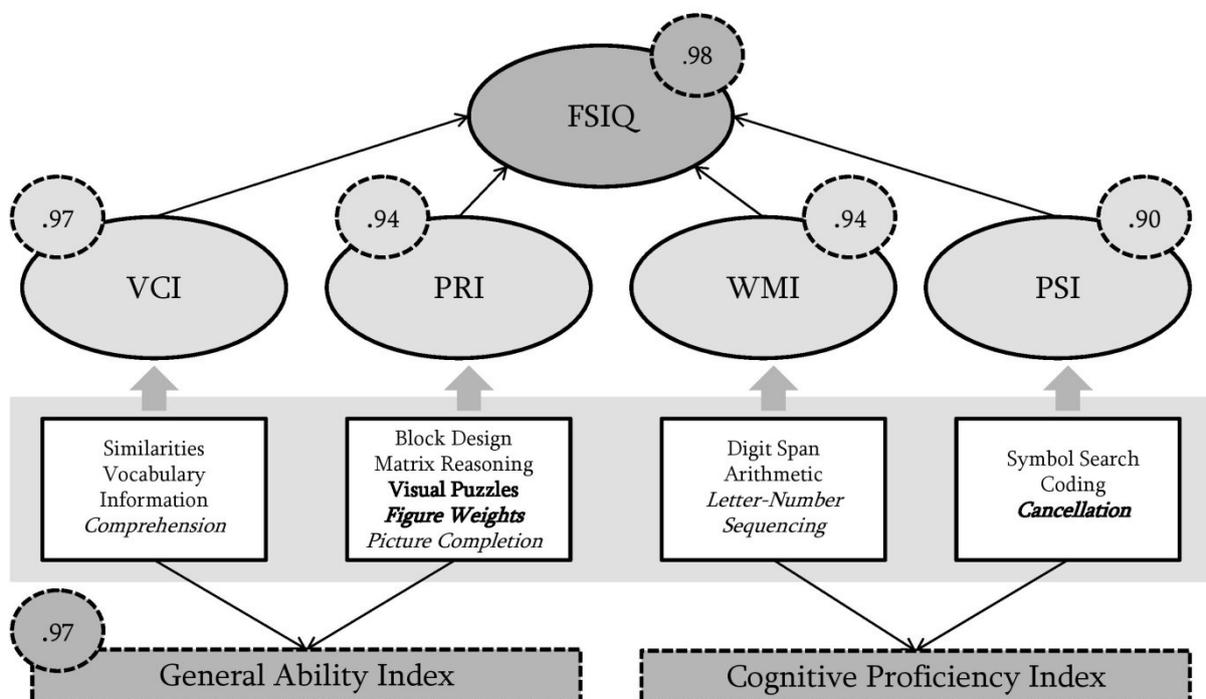


Figure 5. Structure of WAIS-IV with average reliability coefficients (Petermann, 2012). *Note.* Supplemental subtests in cursive letters; new subtests in bold letters; all remaining subtests are core subtests. FSIQ, Full Scale Intelligence Quotient; PRI, Perceptual Reasoning Index; PSI, Processing Speed Index; VCI, Verbal Comprehension Index; WMI, Working Memory Index.

Psychometric properties

The WAIS-IV reliability has been proven strong with good to excellent reliabilities for the composite score, indices, and subtests. This accounts for a reliable interpretation of the WAIS-IV and provides the fundamental requirements for psychometric testing. Recent structure analytic studies confirm this (Benson, Hulac & Kranzler, 2010; Canivez & Watkins, 2012; Weiss, L. G., Keith, Zhu & Chen, 2013). For the German adaption of the WAIS-IV, the average reliability coefficients¹³ of the composite scores are indicated as follows: VCI, .97; PRI, .94; WMI, .94; PSI, .90; FSIQ, .98. For the subtests, reliability coefficients ranged between .78 and .94. (Petermann, 2012) Additionally, strong evidence related to the scale's validity is provided from comparison with other measures of cognitive functions (Petermann, 2012; Wechsler et al., 2008). This supports the utilization of the WAIS-IV as an instrument to assess cognitive functions. Evidence from special group studies (Wechsler et al., 2008) supports its clinical utility and it is assumed that the WAIS-IV measure the same functions in both healthy individuals and in adults with clinical disorders (Weiss, L. G. et al., 2013).

Clinical utility

The WAIS-IV encompasses profound structural changes, content, and administration modification. These make it more adaptable to clinical and neuropsychological settings in contrast to its predecessors. On subtest level several changes have been made to increase its implication for neuropsychological application. These are (a) improved user friendliness (e.g. reduced discontinuous rules, decreased testing time), (b) developmental appropriateness (e.g. reduced sensory and motor demands, explicit instructions, reduced time bonuses), and (c) increased emphasis on neuropsychological constructs. This was done by emphasizing reasoning and working memory abilities and by putting less weight on crystallised knowledge: The measurement of fluid reasoning has been enhanced by adding the subtests 'Visual Puzzles' (VP) and 'Figure Weights' (FW) and revising the subtest 'Matrix Reasoning' (MR). Greater emphasis has been placed on the examination of processing speed and working memory by reducing visual acuity and motor demands in processing speed subtests. Particularly, neurological patients with reduced psychomotor speed gain advances through a reduced focus on speed. In these patients, the FSIQ may not be as much affected as the predecessors. Moreover, the subtests 'Digit Span' (DS), 'Arithmetic' (AR), and 'Letter-Number Sequencing' (LNS) now reflect the advances in the understanding of working memory abilities. Working memory demands were increased (e.g. addition of 'Digit Span Sequencing' [DSS]), time

¹³ For more information on reliability of the WAIS-IV please refer to Petermann (2012).

bonuses were eliminated, and AR items contain reduced verbiage and mathematical knowledge. The subtests of the VCI include an enhanced reasoning with crystallised words. Even though prior crystallised knowledge is required for all subtests to perform, less weight is put on pure crystallised knowledge (e.g. recite learned facts/words), particularly on the subtest 'Similarities' (SI; Wechsler et al., 2008).

Application

The application of the WAIS-IV in adults with ADHD or stroke differed to the extent that the former group was exposed to ten core subtests and two supplemental subtests (LNS, CA) and the latter group was assessed with nine core subtests and one supplemental subtest ('Picture Completion' [PC]). A reason to replace 'Block Design' with PC in the stroke sample was due to reasons of high motor demands of the task and expected motor deficits due to stroke. The administration time of the WAIS-IV required approximately 90 minutes in the ADHD sample and 50 to 60 minutes in the sample of stroke patients.

4.2.2 Self-reports

Several questionnaires were used in the assessment of the present research. Adults with ADHD or stroke filled out the questionnaires by themselves following WAIS-IV administration. Some stroke patients had difficulty reading the items and received help by the examiner accordingly. Adults with ADHD filled out the ADHD questionnaires presented below, the stroke patients filled out the Beck's Depression Scale-II only.

ADHD diagnostic questionnaire and symptom severity

The self-report questionnaire ADHS-SB (Rösler, Retz-Junginger, et al., 2008) was utilised to examine diagnostic criteria of ADHD and to estimate the degree of symptom severity. Participants responded to 22 four-choice items (0-3) on the scales attentiveness, hyperactivity, and impulsiveness. An item was counted as fulfilled when a participant chose a symptom to be slightly, mild or severely pronounced. A diagnosis according to the ICD-10 (World Health Organisation, 1992) or DSM-IV-TR (American Psychiatric Association, 1994) could be given if the specific algorithm was met and if retrospective ADHD symptomatology was verified with four additional items. Hence, when a participant attained a minimum of 6 points on items 1 to 9 (attentiveness), a minimum of 3 on items 10-14 (hyperactivity), and a minimum of 1 on items 15-18 (impulsiveness) a diagnosis according to the ICD-10 was given. Following the DSM-IV-TR algorithm, the criteria for the combined type (ADHD-C) were met when a participant attained a minimum of 6 points on

the items of attentiveness and a minimum of 6 on the items of hyperactivity and impulsiveness. Criteria for the predominantly inattentive type (ADHD-I) and the hyperactive-impulsive type (ADHD-H) were met when either 6 points of the according items were attained. Symptom severity was determined by using unpublished data from the workgroup of Stieglitz. For details please refer to Rösler, Retz-Junginger, et al. (2008). Thus, a raw score which was 1 SD above the mean was accounted for as clinically significant; attentiveness ($M = 15.03$, $SD = 6.08$), hyperactivity ($M = 7.67$, $SD = 3.58$), impulsiveness ($M = 5.65$, $SD = 3.22$), hyperactivity/impulsiveness ($M = 13.29$, $SD = 5.95$), total scale ($M = 28.34$, $SD = 10.76$). The average administration time was 7 minutes. Its internal consistency has been reported between $\alpha = .72$ and $\alpha = .90$. Re-test reliability of the questionnaire ranges between $r_{tt} = .78$ and $r_{tt} = .89$.

ADHD screening for adults

The ADHS-E screening for adults is a dimensional assessment instrument based on the diagnostic criteria of ADHD (Schmidt & Petermann, 2009a). It is a standardised instrument with normative data to determine the degree of symptom severity of ADHD. Next to a global score of severity, the screening permits to evaluate the degree of impairment on five scales: 'Emotion and Affect', 'Attentional Control', 'Unrest and Hyperactivity', 'Impulsivity and Disinhibition', and 'Stress-Tolerance'. The scale 'Emotion and Affect' assesses the control of mood and emotions in stressful situations and in those where solution strategies need to be applied. The scale 'Attentional Control' captures deficits in selective attention, which is often followed by thoughtless actions and disinhibition. Such impulsivity is assessed with the scale 'Impulsivity and Disinhibition' and particularly focused on negative situations. The scale 'Unrest and Hyperactivity' comprises the core symptoms of ADHD according to the ICD-10 and the DSM-IV-TR. Behaviour in stressful situations and coping with stress is evaluated with the scale 'Stress-Tolerance'. The degree of severity is considered clinically significant for t -scores above 70 ($\leq 16^{\text{th}}$ percentile). The administration time was on average 5 to 7 minutes. Internal consistency of the scales determined with the clinical sample ranges between $\alpha = .64$ and $\alpha = .77$. Re-Test reliability of the screening ranges between $r_{tt} = .82$ and $r_{tt} = .94$.

Beck's Depression Scale-II

Post-stroke depressive symptoms were assessed with the Beck's Depression Scale in its second edition (BDI-II; Hautzinger, Kühner & Keller, 2006). On 21 four-choice items, participants responded by selecting an answer based on their emotional, vegetative or behavioral symptoms over the last two weeks. A total score of 63 could be achieved. Scores from 0 to 13 indicated a

'minimal depression', 14 to 19 a 'mild depression', 20 to 28 a 'moderate' and scores ranging from 29 to 63 indicated a 'severe depression'. The duration of the rating scale was approximately 7 minutes. Test-retest reliability is $r_{tt} = .93$, with internal consistency of $\alpha = .93$. The BDI-II has been demonstrated a suitable measure of depression in patients in neurorehabilitation settings and is suggested to provide a reasonable approximation of severity of depression (Siegert, Walkey & Turner-Stokes, 2009). The cut-off scores to determine a diagnosis of depression vary greatly across studies (≥ 10 to ≥ 19 ; see Hackett, Yapa, Parag & Anderson, 2005, for a review). Due to the screening character of the BDI-II and low specificity rates (Lincoln, Nicholl, Flannaghan, Leonard & Van der Gucht, 2003), the cut-off of the total BDI-II score to accept a PSD was set relatively high at ≥ 20 .

4.3 Data management and statistical analyses

The information on data management and analyses in the first part of the following section provides an outline of the statistical analyses used in the present research. Table 9 depicts an overview of the statistical methods employed in studies I, II, and III. For more detailed information regarding statistics please refer to the attached studies in the Appendices B, C, and D. A second part outlines supplemental statistical analyses, which were not published in the studies but were nevertheless included in the present research in order to contribute to its understanding.

In the first stage of data management, the data was entered into Excel spreadsheets and analysed in terms of frequencies and proportions in order to identify typing errors, which were consequently being cleaned. Following this, the data was entered into SPSS software 20.0 (SPSS Inc, 2011) and STATA for analyses, with a level of significance set at $p < .05$. Prior to all analyses, the suitability of statistical methods and their requirements were checked and the assumptions were confirmed. Variables that were not normally distributed were reanalysed with non-parametric statistical methods (e.g. Kruskal-Wallis-Test). None of the statistics deviated strongly from non-parametric outcomes. Therefore, only parametric results were reported. Both the ADHD and the stroke sample were randomly matched to a control group according to age, gender, and education. Therefore, there were no differences between the groups on these demographic variables. Next to eta squared effect sizes (η^2) (Bortz & Döring, 2006), additional effects were calculated and interpreted with Cohen's d formula (Cohen, J., 1988).

For an additional understanding of the present research, supplemental statistical analyses were performed, which were not included in the published studies. These included the calculation of further descriptive statistics of the WAIS-IV. With respect to ADHD, MANOVA was conduct-

ed to examine the effect of ADHD on the subtests LNS and CA. Additional Cohen's *d* effect sizes were calculated. Regarding the stroke sample, independent sample *t*-tests examined the differences between the GAI of patients with stroke and controls. Further analyses on the FSIQ – GAI discrepancy comparison between RHS and LHS patients were conducted. In addition, independent sample *t*-test investigating the difference between the GAI in RHS and LHS patients were performed and two-paired sample *t*-tests examined the difference between the FSIQ and the GAI for the respective groups. Pearson's chi-square tests were conducted to examine whether the observed distribution in the FSIQ < GAI discrepancy in both groups fitted the expected distribution. To further investigate the effect of language impairment on WAIS-IV performance, an additional MANOVA was carried out. Given that language impairment was only present in LHS patients, the entire sample of stroke patients was subdivided into three groups: RHS, LHS without language deficits (LHS⁻), and LHS with language deficits (LHS⁺). Subsequent post-hoc analyses with Bonferroni criterion of significance were performed to examine the difference between these groups. With respect to PSD, MANOVA examines the effect of PSD on WAIS-IV performance. Linear regression analyses were conducted to control for the variables side of stroke and the time between stroke onset and assessment. Possible interaction effects for side of stroke and PSD were examined.

Table 9. Statistical analyses utilised in the present research

	Analyses	Description of statistical method and effects measured
Study I	Pre-analysis	<ul style="list-style-type: none"> • STATA^a: Random Matching according to age, gender, and education • MANOVA: Main effect of gender, age, and education on WAIS-IV^b subtests and composite scores
	Main analysis	<ul style="list-style-type: none"> • MANOVA: Main effect of group^c on WAIS-IV^b subtests, process-, and composite scores • Effect size calculation (Cohen's <i>d</i>) • MANOVA: Main effect of medication and comorbidity on WAIS-IV^b subtests, process-, and composite scores • Two-paired-samples <i>t</i>-test: FSIQ^d < GAI^e discrepancy between group^c • Pearson's chi-square test: Distribution of FSIQ^d < GAI^e discrepancy between ADHD and control
Study II	Pre-analysis	<ul style="list-style-type: none"> • STATA^a: Random Matching according to age, gender, and education • MANOVA: Main effect of gender, age, and education on WAIS-IV^b subtests, process-, and composite scores • Independent-Sample <i>t</i>-test: Time since stroke between LHS and RHS • MANOVA: Effect of hemiparesis on WAIS-IV^b subtests, process-, and composite scores

Table 9. (continued)

	Main analysis	<ul style="list-style-type: none"> • MANOVA: Main effect of groups^e on WAIS-IV^b subtests, process-, and composite scores • MANOVA: Main effect of side of stroke^f on WAIS-IV performance • Post Hoc Analyses with Bonferroni criterion for significance: Differences of aphasia1^g, aphasia2^h, no aphasia on WAIS-IV^b subtests, process-, and composite scores • Effect size calculation (Cohen's <i>d</i>) • MANCOVA: Main effect of side of stroke^f on WAIS-IV subtests, process-, and composite scores, including covariatesⁱ
Study III	Pre-analysis	<ul style="list-style-type: none"> • Expectation-Maximization algorithm for missing values • Pearson product-moment-correlation: Relationship between WAIS-IV^b and ADHD-E^j scales
	Main analysis	<ul style="list-style-type: none"> • Independent-Sample <i>t</i>-tests: Difference between impaired and non-impaired adults with ADHD (ADHS-E^j) across WAIS-IV^b composite scores and subtests • Stepwise regression analyses: Equation^k; $WAIS_i = \beta_0 + \sum_{j=1}^n ADHD_j$

^aSTATA; Data Analysis and Statistical Software, ^bWechsler Adult Intelligence Scale - Fourth Edition, ^cdefined as ADHD and control, ^dFull Scale Intelligence Quotient, ^eGeneral Ability Index, ^fdefined as RHS and control; LHS and control, ^gLHS and RHS hemisphere stroke, ^hLHS patients with fluent speech, trouble word finding, speaking around words, ⁱLHS patients with non-fluent communication due to impairment of spontaneous speech, utterances of words, ^japhasic symptoms, neglect, and hemianopsia, ^kADHD Screening for Adults, ^k $\in \{1,2,3,4,5\}$ denotes 5 ADHS-E scales: 'Emotion and Affect', 'Attentional Control', 'Unrest and Hyperactivity', 'Impulsivity and Disinhibition', 'Stress-Tolerance'.

5 Results

The following section displays the main results of the present investigation based on the aims provided in the previous chapter (see section 3). For detailed statistical findings please refer to the Appendices B to D. Supplemental results, which were not published in the studies, are included if they contribute to the further understanding of the present research aims.

5.1 Cognitive profiles in adults with ADHD or stroke

The current research demonstrates that the WAIS-IV characterizes adults with ADHD or stroke with distinguishable cognitive profiles. Together, these results provide important insights into the sensitivity of the WAIS-IV on different levels of analyses to discriminate between adults with ADHD and healthy individuals, as well as between patients with stroke and healthy individuals. The following lines provide the most essential results of study I and II, separately for adults with ADHD or stroke patients.

Neuropsychological functions on the WAIS-IV of adults with ADHD - difference to a matched control group (study I)

Overall, the analyses reveal a significant decrement across many cognitive domains and global intellectual functioning in adults with ADHD compared to the matched control group. For the most part, composites and standard scores do not fall below the normative mean. On composite level up to 28.4% and on subtest level up to 20.7% of the adults with a diagnosis of ADHD performed more than 1 *SD* below the population mean. Descriptive statistics, *p*-values, and effect sizes are portrayed in Table 10. The results indicate significant differences to controls on the composite score level. The largest effects were found for the WMI ($d = 0.64$), the FSIQ ($d = 0.60$), and the PSI ($d = .44$). Figure 6 graphically displays the mean composite scores of adults with ADHD and matched controls along with the 95% confidence interval. Specifically sensitive to discriminate adults with ADHD and controls are subtests with working memory and processing speed demands. Here, the largest effects were found for subtests AR ($d = 0.64$), DS ($d = 0.45$), CD ($d = 0.41$), and VP ($d = 0.40$). The average subtest scores of adults with ADHD and controls along with the 95% confidence interval are portrayed in Figure 7. Additionally, on process score level large effects were observed on DSB ($d = 0.43$) and DSF ($d = 0.42$).

FSIQ-GAI discrepancy comparison between adults with ADHD and a matched control group (study I)

The results indicate a significant difference in the GAI between adults with ADHD and matched controls ($d = 0.42$), with 13.8% of the adults with ADHD performing more than 1 SD below the population mean (see Table 10). To evaluate whether the GAI serves as a more appropriate measure of overall intellectual ability than the FSIQ, discrepancy analyses between the two composites were performed. The findings demonstrate a significant difference between the GAI and the FSIQ ($d = 0.37$). In total, 58% of the adults with ADHD showed a pattern of FSIQ < GAI discrepancy, with 43% obtaining a significant discrepancy. The magnitude is relatively large (min = -1, max = -14). Even though not all adults with ADHD and their matched controls fulfilled the requirements for the GAI calculation (see section 4.2.1), the results provide some evidence for meaningful differences in significant GAI < FSIQ discrepancies between the groups. Upon examination of this data, it was verified how rare or common the adults' discrepancy was in the normative sample population. Thus, absolute values of the difference scores were determined by ability level. Here, the FSIQ < GAI pattern of 25% of the adults with ADHD was ranked below the 16th percentile and 19% ranked below the 10th percentile. There are significant differences at both percentile ranges between adults with ADHD and controls.

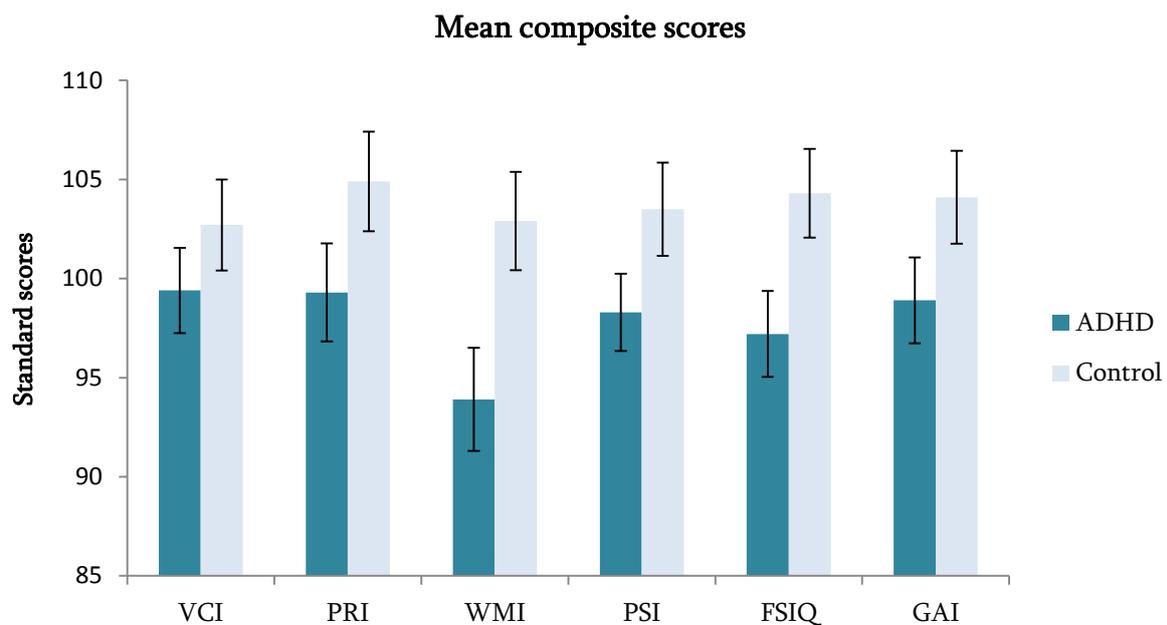
Table 10. Cognitive profiles on the WAIS-IV of adults with ADHD

Subtest/Process/ Composite scores	<i>n</i>	<i>M</i>	<i>SD</i>	Confidence interval (95%)	<i>p</i>	<i>d</i> ^w	% 1SD ^x
VCI ^a	116	99.4	11.8	97.9 - 101.6	.044	0.27	13.8%
SI ^b	116	9.9	2.5	9.5 - 10.4	.076	0.23	10.3%
VC ^c	116	9.7	2.5	9.3 - 10.2	.065	0.24	10.3%
IN ^d	116	10.1	2.6	9.6 - 10.6	.084	0.23	11.2%
PRI ^e	116	99.3	13.6	96.8 - 101.8	.002	0.41	17.2%
BD ^f	116	10.1	2.9	9.6 - 10.6	.029	0.29	9.5%
MR ^g	116	9.9	2.5	9.5 - 10.4	.025	0.30	12.1%
VP ^h	116	9.8	2.8	9.3 - 10.3	.002	0.40	15.5%
WMI ⁱ	116	93.9	14.3	91.3 - 99.5	.000	0.64	28.4%
DS ^j	116	8.9	2.7	8.4 - 9.4	.001	0.45	17.2%
AR ^k	116	8.9	2.9	8.4 - 9.4	.000	0.64	20.7%
LNS ^l	107	9.8	3.0	9.2 - 10.4	.097	0.23	10.1%
PSI ^m	116	98.3	10.7	96.4 - 100.3	.001	0.44	7.8%
SS ⁿ	116	9.8	2.2	9.4 - 10.2	.010	0.34	6.9%
CD ^o	116	9.6	2.3	9.2 - 10.0	.002	0.41	6.9%
CA ^p	107	9.5	2.6	9.0 - 10.0	.053	0.27	14.7%
FSIQ ^q	116	97.2	11.9	95.0 - 99.4	.000	0.60	19.0%

Table 10. (continued)

GAI^f	116	98.9	11.9	96.7 - 101.1	.001	0.42	13.8%
BDN^g	116	10.5	3.8	9.8 - 11.2	.777	0.04	7.8%
DSFⁱ	116	9.1	2.5	8.7 - 9.6	.001	0.42	16.4%
DSB^u	116	8.9	2.7	8.4 - 9.4	.001	0.44	18.1%
DSS^v	116	9.6	2.7	9.1 - 10.1	.022	0.30	12.9%

^a Verbal Comprehension Index, ^bSimilarities, ^cVocabulary, ^dInformation, ^ePerceptual Reasoning Index, ^fBlock Design, ^gMatrix Reasoning, ^hVisual Puzzles, ⁱWorking Memory Index, ^jDigit Span, ^kArithmetic, ^lLetter-Number Sequencing, ^mProcessing Speed Index, ⁿSymbol Search, ^oCoding, ^pCancellation, ^qFull Scale Intelligence Quotient; ^rGeneral Ability Index, ^sBlock Design No Time Bonus, ^tDigit Span Forward, ^uDigit Span Backward, ^vDigit Span Sequential, ^wCohen's *d* effect size, ^xFrequency of adults more than 1 standard deviation below the population mean.

**Figure 6.** WAIS-IV composite scores of adults with ADHD and matched controls.

Note. Error bars indicate 95% confidence intervals. FSIQ, Full Scale Intelligence Quotient; GAI, General Ability Index; PRI, Perceptual Reasoning Index; PSI, Processing Speed Index; VCI, Verbal Comprehension Index; WAIS-IV, Wechsler Adult Intelligence Scale - Fourth Edition; WMI, Working Memory Index.

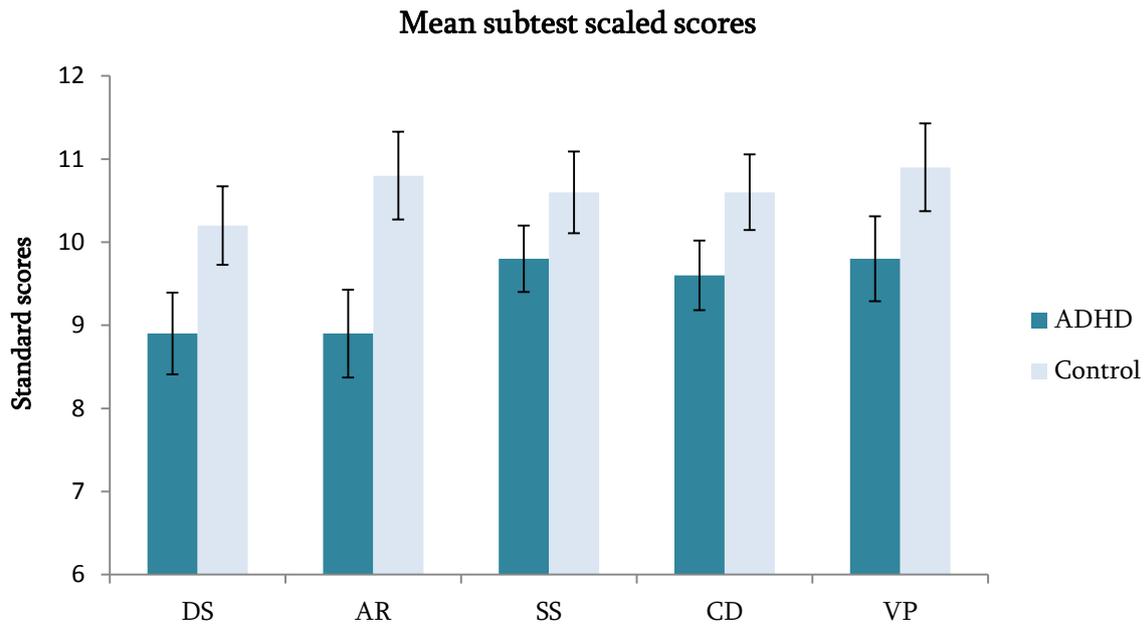


Figure 7. Selected WAIS-IV subtest scaled scores of adults with ADHD and matched controls.

Note. Error bars indicate 95% confidence intervals. AR, Arithmetic; CD, Coding; DS, Digit Span; SS, Symbol Search; VP, Visual Puzzles; WAIS-IV, Wechsler Adult Intelligence Scale - Fourth Edition.

Neuropsychological functions on the WAIS-IV of RHS patients - difference to a matched control group (study II)

The analyses on patients with RHS reveal a significant decrement across all cognitive domains and global intellectual functioning compared to the matched control group. On the composite level, frequency statistics indicate that up to 74.1% of the patients perform more than 1 *SD* and 22.4% more than 2 *SDs* below the mean of the control group. On subtest level, this occurred for up to 62.1% (≥ 1 *SD*) and 17.2% (≥ 2 *SD*). Table 11 displays descriptive statistics, *p*-values, and effect sizes for these patients. Statistical analyses furthermore indicate significant differences to the control group on all composite scores, with the largest effects found for the FSIQ ($d = 1.69$), the GAI ($t(114) = -8.40$, $p < .001$, $d = 1.56$), the PRI ($d = 1.64$), and the PSI ($d = 1.39$). Figure 8 portrays the means of all composites along with the 95% confidence interval for RHS patients and controls. An evaluation of subtest differences between the groups indicates significant decrements on all subtests for RHS patients. The largest effects are observed for predominantly nonverbal subtests; MR ($d = 1.45$), VP ($d = 1.36$), and SS ($d = 1.23$).

Neuropsychological functions on the WAIS-IV of LHS patients - difference to a matched control group (study II)

The analyses on patients with LHS indicate significant decrements across all cognitive domains and global intellectual functioning compared to the matched control group. Frequency sta-

tistics on composite scores reveal that up to 77.6% of the patients perform more than 1 *SD* below the population mean and up to 28.6% more than 2 *SDs*. Across the subtests, up to 69.4% of the LHS patients performed more than 1 *SD* and up to 20.4% more than 2 *SDs* below the normative sample. Descriptive statistics, *p*-values, and effect sizes for the patients are portrayed in Table 11. Differences to the controls additionally reveal significant effects across all composite scores, with the largest effects occurring for the VCI ($d = 1.15$), the FSIQ ($d = 1.12$), and the PSI ($d = 1.08$). The GAI differed significantly between patients and controls ($t(96) = -4.99, p < .001, d = 1.01$). In Figure 8, the composition of these scores is displayed along with the 95% confidence interval. Across all subtests, with the exception of subtest PC ($p = .122$), the LHS patients perform significantly worse than matched controls. The largest effects occurred primarily on subtests with verbal and processing speed demands; SI ($d = 1.39$), CD ($d = 1.14$), VC ($d = 1.01$). On process score level effect sizes occurred up to $d = 1.09$ for DSF.

Neuropsychological functions on the WAIS-IV - difference between RHS and LHS patients (study II)

Further statistical analyses have been performed on subgroup-specific effects on the WAIS-IV. Here, performances of RHS and LHS patients were compared to evaluate the WAIS-IV sensitivity to hemispheric dysfunction. For statistical details please refer to Appendix B. The results indicate a significantly lower PRI ($d = 0.48$) after RHS and lower performances on the subtests with visuo-spatial demands in RHS patients compared to LHS; MR ($d = 0.51$) and PC ($d = 0.42$) (see Figure 9). These subtests seem most challenging and specifically sensitive for patients with RHS. In addition, significantly lower performance was found in LHS patients primarily for subtests with verbal demands; SI ($d = 0.47$) (see Figure 9). Process scaled score DSF ($d = 0.22$) was found significantly decremented in patients with LHS. No significance but a tendency of overall disadvantage was found for patients with LHS on the VCI and the WMI and related subtests.

FSIQ-GAI discrepancy comparison between RHS and LHS patients (no included in study II)

The GAI for LHS and RHS patients was calculated (see Table 11). By reducing the FSIQ by working memory and processing speed abilities, the estimate of the GAI resulted in a mean of 79.4 ($SD = 9.9$) in RHS patients and a mean of 80.0 ($SD = 12.1$) in LHS patients, with no significant difference between them ($t(105) = -.293, p > .05$). Paired-two-sample *t*-tests indicate significant differences between the FSIQ and the GAI in RHS patients ($t(58) = 3.06, p < .01$) and in LHS patients ($t(49) = 3.63, p < .001$). In order to determine the significance of the FSIQ < GAI discrepancy pattern in these groups, the significant value at the .15 level was utilised. Altogether, 79% of the RHS

patients show a pattern of $FSIQ < GAI$ performance, with 55.2% obtaining a significant discrepancy. The magnitude of the $FSIQ < GAI$ was large (min. -1, max. -10). In contrast, 76% LHS patients show a pattern of $FSIQ < GAI$ performance, with 61% obtaining a significant discrepancy. Here, the magnitude was again quite large (min. -1, max. -8). There are no statistically meaningful differences between significant $FSIQ < GAI$ discrepancies of RHS and LHS patients. By determining how rare or common the difference score is relative to the normative sample (by the ability level), the results demonstrate a $FSIQ < GAI$ pattern in 17% of RHS patients below the 16th percentile and 10% below the 10th. In contrast, 29% of the LHS patients showed this pattern below the 16th percentile and 22% below the 10th. There were no significant differences at both percentile ranges between the groups (16th; $\chi^2(1, 83) = 2.59, p < .05$; 10th; $\chi^2(1, 83) = 3.51, p < .05$).

Table 11. Cognitive profiles on the WAIS-IV of stroke patients

Subtest/Process/ Composite scores	Right stroke patients (<i>n</i> = 58)						Left stroke patients (<i>n</i> = 49)					
	<i>M</i> (<i>SD</i>)	Confidence interval (95%)	<i>p</i>	<i>d</i> ^t	% 1SD ^u	% 2SD ^v	<i>M</i> (<i>SD</i>)	Confidence interval (95%)	<i>p</i>	<i>d</i> ^t	% 1SD ^u	% 2SD ^v
VCI ^a	82.2 (13.0)	78.9 - 85.6	>.001	1.16	56.9%	17.2%	78.3 (12.4)	74.8 - 81.8	>.001	1.15	77.6%	22.4%
SI ^b	6.7 (2.7)	6.0 - 7.4	>.001	0.97	51.7%	12.1%	5.5 (2.1)	4.9 - 6.1	>.001	1.39	69.4%	12.2%
VC ^c	6.4 (2.7)	5.7 - 7.1	>.001	1.13	50.0%	17.2%	5.9 (2.7)	5.1 - 6.7	>.001	1.01	69.4%	16.3%
IN ^d	7.4 (2.9)	6.7 - 8.2	>.001	0.90	44.8%	5.2%	6.9 (2.8)	6.1 - 7.7	>.001	0.79	57.1%	6.1%
PRI ^e	80.2 (9.4)	77.8 - 82.6	>.001	1.64	63.8%	17.2%	85.8 (13.6)	82.0 - 89.6	>.001	0.69	49.0%	12.2%
MR ^f	6.3 (1.6)	5.9 - 6.7	>.001	1.45	62.1%	1.7%	7.4 (2.6)	6.7 - 8.1	.003	0.63	42.9%	4.1%
VP ^g	7.0 (1.8)	6.5 - 7.5	>.001	1.36	44.8%	0.0%	7.4 (2.4)	6.7 - 8.1	>.001	0.88	32.7%	6.1%
PC ^h	7.0 (2.6)	6.3 - 7.7	>.001	0.80	41.4%	8.6%	8.2 (3.0)	7.4 - 9.0	.122	0.32	32.7%	6.1%
WMI ⁱ	85.3 (14.2)	81.7 - 89.0	>.001	1.06	46.6%	19.0%	81.7 (15.8)	77.3 - 86.1	>.001	0.94	61.2%	28.6%
DS ^j	7.3 (2.9)	6.6 - 8.1	>.001	0.96	39.7%	13.8%	6.7 (3.2)	5.8 - 7.6	>.001	0.85	55.1%	18.4%
AR ^k	7.5 (2.5)	6.9 - 8.1	>.001	0.99	37.9%	1.7%	7.0 (2.8)	6.2 - 7.8	>.001	0.84	49.0%	6.1%
PSI ^l	78.8 (11.1)	75.9 - 81.7	>.001	1.39	72.4%	15.5%	79.7 (13.9)	75.8 - 83.6	>.001	1.08	73.5%	22.4%
SS ^m	6.2 (2.4)	5.6 - 6.8	>.001	1.23	62.1%	12.1%	6.8 (2.7)	6.0 - 7.6	>.001	0.87	44.9%	14.3%
CD ⁿ	5.9 (2.3)	5.3 - 6.5	>.001	1.17	62.1%	12.1%	5.8 (2.6)	5.1 - 6.5	>.001	1.14	69.4%	20.4%
FSIQ ^o	77.8 (10.7)	75.1 - 80.6	>.001	1.69	74.1%	22.4%	77.8 (13.3)	74.1 - 81.5	>.001	1.12	73.5%	26.5%
GAI ^p	79.4 (9.9)	76.9 - 82.0	>.001	1.56	69.0%	15.5%	80.0 (12.1)	76.6 - 83.4	>.001	1.01	71.4%	10.2%
DSF ^q	7.9 (2.7)	7.2 - 8.6	>.001	0.84	36.2%	5.2%	6.8 (2.8)	6.0 - 7.6	>.001	1.09	38.8%	14.3%
DSB ^r	7.3 (2.9)	6.6 - 8.1	>.001	0.94	43.1%	10.3%	7.4 (3.2)	6.5 - 8.3	.009	0.54	38.8%	8.2%
DSS ^s	8.4 (3.2)	7.6 - 9.2	.002	0.59	29.3%	3.4%	7.7 (3.1)	6.8 - 8.6	.005	0.58	40.8%	6.1%

^aVerbal Comprehension Index, ^bSimilarities, ^cVocabulary, ^dInformation, ^ePerceptual Reasoning Index, ^fMatrix Reasoning, ^gVisual Puzzles, ^hPicture Completion, ⁱWorking Memory Index, ^jDigit Span, ^kArithmetic, ^lProcessing Speed Index, ^mSymbol Search, ⁿCoding, ^oFull Scale Intelligence Quotient, ^pGeneral Ability Index, ^qDigit Span Forward, ^rDigit Span Backward, ^sDigit Span Sequential, ^tCohen's *d* effect size, ^ufrequency of adults more than 1 standard deviation below the population mean, ^vfrequency of patients more than 2 standard deviations below the population mean.

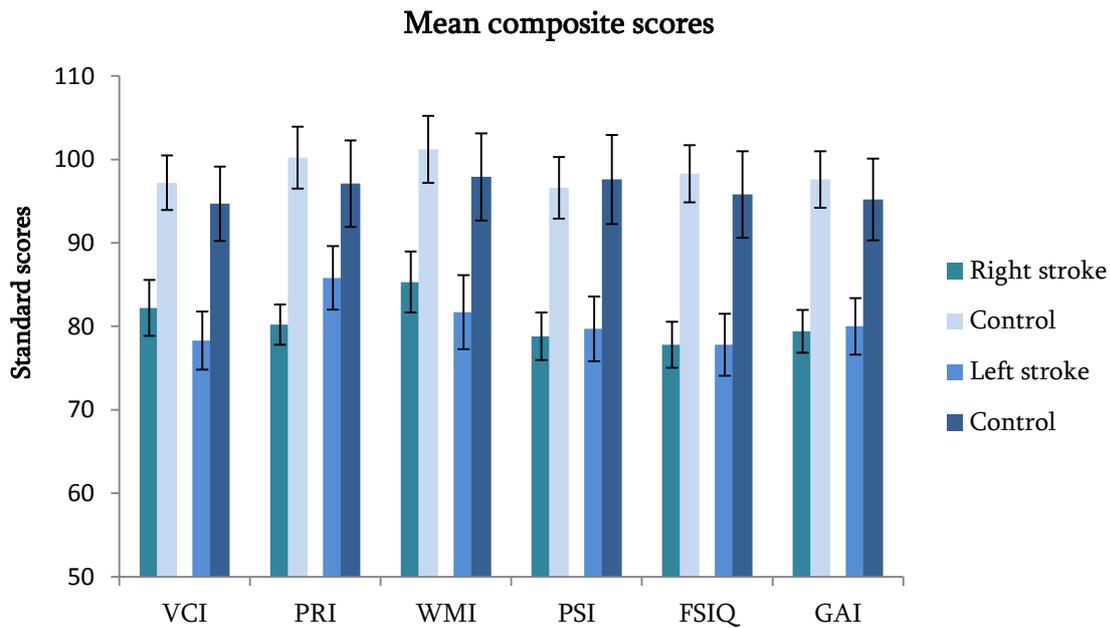


Figure 8. WAIS-IV composite scores of patients with right hemisphere stroke and left hemisphere stroke with individually matched controls.

Note. Error bars indicate 95% confidence intervals. FSIQ, Full Scale Intelligence Quotient; GAI, General Ability Index; PRI, Perceptual Reasoning Index; PSI, Processing Speed Index; VCI, Verbal Comprehension Index; WAIS-IV, Wechsler Adult Intelligence Scale - Fourth Edition; WMI, Working Memory Index.

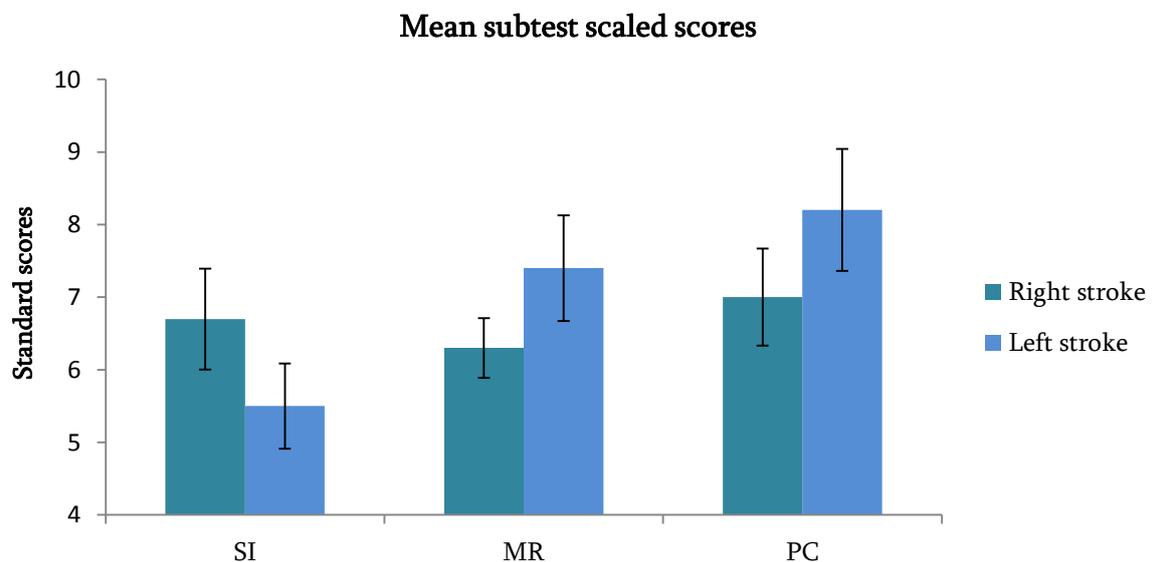


Figure 9. Selected WAIS-IV subtest scaled scores of patients with right hemisphere stroke and left hemisphere stroke

Note. Error bars indicate 95% confidence intervals. MR, Matrix Reasoning; PC, Picture Completion; SI, Similarities; WAIS-IV, Wechsler Adult Intelligence Scale - Fourth Edition.

5.2 Additional influences of confounding variables

Several variables that possibly impact intellectual and cognitive performance of the WAIS-IV were considered in the ADHD and the stroke sample. The following section provides the most essential results of the present research.

The effect of comorbidity and medication at the time of assessment on WAIS-IV performance in adults with ADHD (study I)

Adults with ADHD differ significantly from the matched control group in diverse cognitive and global intellectual functioning. Within the ADHD group comorbidities and medication at the time of assessment had no significant effect on WAIS-IV performance. As no significant part of these deficits can be explained by other psychiatric disorders or by medication intake, the differences between adults with ADHD and matched controls are robust with respect to these variables.

The contribution of language deficits, neglect, and hemianopsia on WAIS-IV performance in patients with stroke (study II)

Statistical analyses on language deficits in LHS patients indicate a negative contribution of these deficits on subtests requiring verbal skills, working memory, and processing speed functioning. As indicated by post-hoc analyses, performances of LHS patients with good comprehension abilities but non-fluent communication are significantly different from those LHS patients with no language deficits in the composites WMI and FSIQ, as well as in the subtests VC, AR, and VP. Similarly, there are no differences to those LHS patients with trouble of word finding. This implies that that language deficits in LHS patients, particularly those of ‘non-fluent communication’ (e.g. impairment of spontaneous speech, utterances of words) may have an impact on performance on these composites and subtests. Further adjustment for language deficits in analyses comparing RHS and LHS patients on WAIS-IV performance gives some confidence to these conclusions. Moreover, adjustment for neglect and hemianopsia in the analyses reduces some error variance on subtests with language, working memory, visuo-spatial, visuo-analytic, and processing speed demands (DS, VC, AR, SS, CD). These results indicate that the negative contribution of RHS on visuo-spatial and processing speed subtests increased after adjustment.

The difference between stroke patients with language deficits and without (not included in study II)

To further investigate the contribution of language impairment on WAIS-IV subtest performances, the effect of stroke (RHS, LHS⁻, LHS⁺) on WAIS-IV subtest performance was examined using

MANOVA. Results confirm a main effect of stroke, Wilks's $\lambda = .614$, $F(20, 190) = 2.26$, $p < .001$. Further univariate F statistics reveal significant effects on SI $F(2, 104) = 3.18$, $p < .05$, DS $F(2, 104) = 3.21$, $p < .01$, MR $F(2, 104) = 6.75$, $p < .01$, VC $F(2, 104) = 3.77$, $p < .05$, AR $F(2, 104) = 5.22$, $p < .01$, and SS $F(2, 104) = 4.01$, $p < .05$. In order to examine the difference between RHS, LHS⁻, and LHS⁺ stroke on subtest performance, additional post-hoc analyses with Bonferroni criterion of significance were performed. Results indicate significant differences between RHS and LHS⁺ stroke patients on subtests SI ($\Delta 1.36$, $p < .05$), AR ($\Delta 1.47$, $p < .05$), between RHS and LHS⁻ on MT ($\Delta -1.97$, $p < .001$) and SS ($\Delta -1.66$, $p < .05$), as well as between LHS⁻ and LHS⁺ on MR ($\Delta 1.51$, $p < .05$), VC ($\Delta 2.03$, $p < .05$), AR ($\Delta 2.28$, $p < .01$), and SS ($\Delta 1.87$, $p < .05$).

The contribution of PSD on WAIS-IV performance in patients with stroke (not included in study II)

Further supplemental analyses were performed in order to examine the effect of PSD on WAIS-IV performance in stroke patients. Descriptive statistics show that $n = 21$ RHS patients and $n = 17$ LHS patients have a PSD. Of all patients with a diagnosis of PSD, 66% ($n = 25$) had their first-ever stroke onset 2 to 6 weeks before the assessment, 13% ($n = 5$) 18 to 31 weeks, and 21% ($n = 8$) of the patients were assessed ≥ 1 year post-stroke. MANOVA reveals that there was no main effect of PSD on WAIS-IV subtests and composite scores (Wilk's $\lambda = .823$, $F(16, 90) = 1.14$, $p > .05$). Additional F -tests in the univariate analyses show significant differences between stroke patients with PSD and those without, indicating a decrement on MR ($F(1, 105) = 3.99$, $p < .05$), SS ($F(1, 105) = 3.98$, $p < .05$), VP ($F(1, 105) = 6.96$, $p < .01$), and PRI ($F(1, 105) = 7.20$, $p < .01$) for patients with PSD. Figure 10 portrays these selected WAIS-IV subtest scaled scores of stroke patients with PSD and those without. Further linear regression analyses the effect of PSD, controlled for the side of stroke as well as for the time between stroke onset and assessment on WAIS-IV performance (see Table 12). The analyses indicated no difference in the effect of PSD (all p 's $> .05$). Moreover, no interaction effects between side of stroke and PSD were found (see Table 13).

Table 12. The effect of PSD adjusted for side of stroke and time since stroke on each WAIS-IV subtest scaled and composite scores

Subtest/ Composite scores	PSD ^a		PSD side ^r		PSD time ^s	
	β (SE)	p	β (SE)	p	β (SE)	p
VCI ^a	0.87 (2.60)	.738	0.94 (2.58)	.718	0.96 (2.62)	.713
SI ^b	-0.05 (.51)	.916	-0.04 (.49)	.944	-0.06 (.51)	.910
VC ^c	0.44 (.56)	.437	0.44 (.56)	.429	0.45 (.56)	.420
IN ^d	-0.07 (.57)	.906	-0.06 (.58)	.918	-0.37 (.58)	.949
PRI ^e	6.22 (2.31)	.008	6.13 (2.26)	.008	6.09 (2.34)	.010

Table 12. (continued)

MR^f	0.87 (.43)	.048	0.85 (.42)	.046	0.86 (.44)	.051
VP^g	1.09 (.41)	.009	1.09 (.41)	.010	1.07 (.42)	.011
PC^h	0.98 (.58)	.093	0.96 (.57)	.094	0.93 (.58)	.109
WMIⁱ	3.24 (3.02)	.285	3.30 (3.02)	.276	2.94 (3.01)	.330
DS^j	0.56 (.61)	.363	0.57 (.61)	.354	0.48 (.61)	.427
AR^k	0.61 (.54)	.259	0.62 (.54)	.252	0.58 (.54)	.285
PSI^l	4.00 (2.49)	.112	3.99 (2.51)	.114	3.87 (2.51)	.125
SS^m	1.01 (.51)	.048	1.00 (.51)	.050	0.99 (.51)	.055
CDⁿ	0.45 (.49)	.366	0.45 (.49)	.364	0.43 (.49)	.394
FSIQ^o	4.10 (2.38)	.088	4.10 (2.39)	.089	3.98 (2.39)	.098
GAI^p	3.34 (2.19)	.130	3.33 (2.21)	.134	3.31 (2.21)	.137

^aVerbal Comprehension Index, ^bSimilarities, ^cVocabulary, ^dInformation, ^ePerceptual Reasoning Index, ^fMatrix Reasoning,; ^gVisual Puzzles, ^hPicture Completion, ⁱWorking Memory Index, ^jDigit Span, ^kArithmetic, ^lProcessing Speed Index, ^mSymbol Search, ⁿCoding, ^oFull Scale Intelligence Quotient, ^pGeneral Ability Index, ^qPost-Stroke Depression, ^rside of stroke, ^stime between stroke onset and assessment.

Table 13. Interaction between PSD and side of stroke on each WAIS-IV subtest scaled and composite scores

Subtest/ Composite scores	PSD ^a		Side ^r		PSD ^a *Side ^r	
	β (SE)	<i>p</i>	β (SE)	<i>p</i>	β (SE)	<i>p</i>
VCI^a	4.64 (3.82)	.228	8.28 (4.16)	.049	-6.78 (5.17)	.193
SI^b	0.54 (0.74)	.463	1.82 (0.80)	.025	-1.06 (0.99)	.291
VC^c	1.10 (0.83)	.186	1.25(0.90)	.168	-1.21 (1.12)	.284
IN^d	0.77 (0.85)	.371	1.50 (0.93)	.110	-1.52 (1.16)	.193
PI^e	10.05 (3.34)	.003	-0.85 (3.62)	.814	-7.16 (4.51)	.116
MR^f	1.35 (0.63)	.034	-0.48 (0.68)	.481	-0.91 (0.85)	.288
VP^g	1.67 (0.61)	.007	0.32 (0.67)	.627	-1.07 (0.83)	.196
PC^h	1.97 (0.84)	.021	0.01 (0.91)	.990	-1.83 (1.13)	.108
WMIⁱ	7.64 (4.46)	.090	8.77 (4.85)	.074	-7.92 (6.03)	.192
DS^j	1.36 (0.91)	.137	1.62 (0.99)	.104	-1.44 (1.23)	.242
AR^k	1.35 (0.79)	.094	1.41 (0.87)	.109	-1.33 (1.08)	.219
PSI^l	7.22 (3.72)	.055	3.01 (4.04)	.458	-5.89 (5.03)	.243
SS^m	1.58 (0.75)	.039	0.14 (0.82)	.865	-1.05 (1.02)	.304
CDⁿ	0.98 (0.74)	.190	0.83 (0.80)	.302	-0.96 (1.00)	.342
FSIQ^o	8.39 (3.52)	.019	5.13 (3.83)	.183	-7.85 (4.77)	.102
GAI^p	7.33 (3.25)	.026	4.15 (3.53)	.243	-7.30 (4.39)	.099

^aVerbal Comprehension Index, ^bSimilarities, ^cVocabulary, ^dInformation, ^ePerceptual Reasoning Index, ^fMatrix Reasoning,; ^gVisual Puzzles, ^hPicture Completion, ⁱWorking Memory Index, ^jDigit Span, ^kArithmetic, ^lProcessing Speed Index, ^mSymbol Search, ⁿCoding, ^oFull Scale Intelligence Quotient, ^pGeneral Ability Index, ^qPost-Stroke Depression, ^rside of stroke.

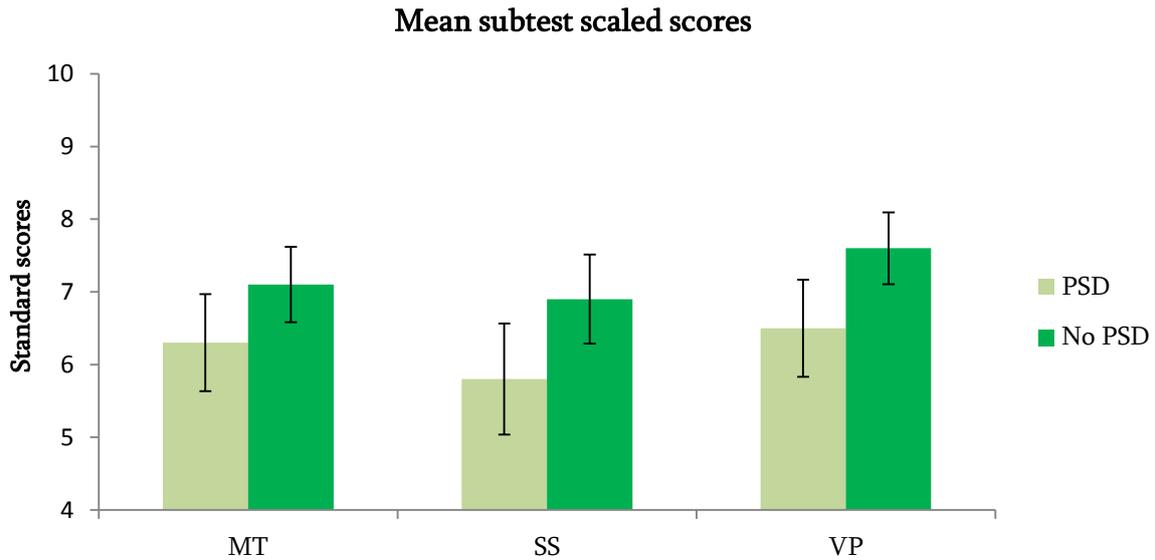


Figure 10. Selected WAIS-IV subtest scaled scores of stroke patients with Post-Stroke Depression and those without.

Note. Error bars indicate 95% confidence intervals. MR, Matrix Reasoning; SI, Similarities; SS, Symbol Search; VP, Visual Puzzles; WAIS-IV, Wechsler Adult Intelligence Scale - Fourth Edition.

5.3 The status of self-evaluation in the assessment of ADHD in adults

The status of self-evaluation in the assessment of adults with ADHD was addressed by examining the relationship between cognitive functions and self-reported symptoms in a sample of adults with ADHD. The following lines provide the most essential results of study III. For detailed statistical results please refer to the Appendix D.

The relationship between self-reported symptoms and WAIS-IV performance (study III)

The statistical analyses demonstrate that the association between self-reported ADHD symptoms and WAIS-IV performance is limited. Only few significant correlations can demonstrate some kind of relationship. Further statistics reveal that, based on self-reported ADHD symptoms, impaired and non-impaired adults with ADHD do not differ in WAIS-IV performance. However, it is noteworthy that there is a tendency of those patients evaluating their symptoms as particularly severe to perform better in WAIS-IV subtests compared to those reporting less severe symptoms. The missing relationship is supported by further analyses to predict WAIS-IV performance in the form of composite scores with self-reported symptoms. Here, only symptoms of the scale 'Emotion and Affect' as well as 'Stress-Tolerance' can be considered as significant predictors, especially for the composite PRI and the FSIQ. Thus, the results of this study go some way to con-

clude that the status of self-evaluation of adults with ADHD is somewhat questionable due to a missing link between objectively measured impairment and that which is self-rated.

6 Discussion

Based on the information gained by the literature review, neuropsychological functioning in adults with ADHD or stroke is often neglected in clinical practice and current research is limited and inconsistent. Nevertheless, it is of great importance to study the deficits in each of these groups and the related clinical aspects that may function as confounding factors. This is of high relevance particularly against the backdrop that neuropsychological impairment serves specific purposes for diagnostic decision making and treatment in both groups. Therefore, the investigation aimed to examine the neuropsychology of adults with ADHD and first-ever unilateral, ischemic stroke patients in rehabilitation, in comparison to healthy individuals. The aim was to generate cognitive profiles with the WAIS-IV and determine whether intellectual and cognitive functioning vary as a function of clinical characteristics in these groups. Moreover, the current investigation aimed to examine the status of self-evaluation in adults with ADHD with the purpose to address the issue of diagnostic accuracy. Clinical practice may benefit from integrating the newly acquired knowledge of the current investigation. Adding to the existing literature of management and care, this section will highlight the important clinical aspects the present research has raised. The following is devoted to address the application of the WAIS-IV in adults with ADHD or stroke, illustrating in detail specific clinical implications and conclusions that can be drawn from the results. Moreover, directions for future research and clinical practice will be addressed as well as general limitations.

6.1 ADHD in adulthood

This section discusses the implication of the WAIS-IV in adults with ADHD by pointing out the most central results of the current investigation and their importance for clinical care. Subsequently, it is outlined how to increase the neuropsychological interpretation of the WAIS-IV in order to provide more accurate cognitive profiles of adults with ADHD. Moreover, the role of neuropsychological assessments in the diagnostic process and therapy is discussed. Specific shortcomings will be addressed, important issues of neuropsychological performance to diagnosis and treatment are discussed, and directions for future research and practice are denoted.

Implications of the WAIS-IV in adults with ADHD

In line with prior research (see section 2.1.2), the current findings illustrate heterogeneous WAIS-IV profiles. Statistically meaningful patterns in adults with ADHD on the level of composites, with compromised scores compared to the control group, could be demonstrated. More specifically, relatively low scores on the WMI, PSI, and FSIQ could be identified as being somewhat characterizing for their cognitive profile. In order to enrich the quality and breadth of the interpretation of the WAIS-IV for adults with ADHD in clinical practice, these composites with respect to their corresponding subtests are given further notice.

Working memory

Adults with ADHD are identified with a statistically lower WMI compared to the control sample. In accordance with the results, this reflects difficulties in verbal short-term memory (DSF), verbal working memory (DSB, DSS, AR), and fluid reasoning (AR). These findings are congruent to other studies (see section 2.1.2) and suggest that the WAIS-IV can in fact serve as a measure to provide broad information about strengths and weaknesses in verbal working memory functions in adults with ADHD. This corroboration furthermore strengthens the following conclusive implications for clinical care. In practice, clinicians can use the information of a lower WMI and corresponding subtest pattern to infer to considerable impairment in real-life situations. For example, occupational difficulties can be parallel with these deficits to explain mental fatigue, stress or making more errors. This information provides a first indication for the nature of behavioural symptoms causing impairment in occupational settings, such as increased time to process and recall information, to keep in mind information and meetings, or to sustain and shift attention (Weiss, L. G., Saklofske, Coalson & Raiford, 2010). Information of working memory deficits may not only be taken as an indication of behavioural daily-life impairment but as a basis for treatment. It can specifically be used to plan therapeutic sessions with the aim to strengthen the core functions of working memory and related abilities (e.g. attention). With intensive memory trainings, both the symptoms and the underlying neuropsychology of the adults could be targeted. Trainings on working memory abilities have been found to alleviate attention deficits and reduce symptoms in children and college students with ADHD (Beck, Hanson, Puffenberger, Benninger & Benniger, 2010; Gropper, Gotlieb, Kronitz & Tannock, 2014; Klingberg, Forssberg & Westerberg, 2002), with effects of increased performance on working memory tasks up to several months after training (e.g. Holmes, Gathercole & Dunning, 2009). Promising results on the efficacy of these trainings have also been reported in adults with ADHD from neuroimaging studies

(Olesen, Westerberg & Klingberg, 2004). The results support a neural adaptation associated with the cognitive training and suggest that trainings can directly affect the neural systems underlying working memory abilities. This means that they can change the dopamine functioning in individuals with ADHD, which may lead to a persistent improvement in behavioral and cognitive deficits since dopamine is involved in learning (see Rutledge, van den Bos, McClure & Schweitzer, 2012, for a review).

Given that adults with ADHD may be deficient in other working memory constructs as well, one source of weakness of the WAIS-IV is that it involves only measures for verbal working memory. Thus, further notice, if necessary, should also be given to visual working memory, for example. By viewing the present status of research in adult ADHD, however, scarce evidence has been found on deficient performance in memory tasks involving visual material (see Skodzik et al., 2013, for a review). The data points more strongly towards encoding deficits of verbal material and a disruption of the phonological loop (see Hervey et al., 2004, for a review). Therefore, verbal working memory deficits found in the WAIS-IV may be used as a fundamental basis for neuropsychological functioning, though clinicians are still advised to apply further neuropsychological evaluation. This is also because verbal working memory is closely interrelated and theoretically overlapping with other higher-order functioning, such as attention and executive functions. Therefore, these abilities should receive a more thorough focus.

Processing speed

The current research further demonstrates differences between adults with ADHD and the controls on the PSI and relating processing speed subtests (CD, SS). This indicates impairment in the functions necessary for successful performance in these tasks, including visuo-motor processing speed, cognitive flexibility, and attention. A pattern like this should gain specific notice as prior studies examining the same construct demonstrate similar results (see section 2.1.2). In settings of clinical care this information may be of particular importance. A PSI pattern to the one found in the present investigation may imply impairments in processing information efficiently (e.g. comprehend and integrate information). This can be proven useful in helping to understand the nature of behavioural problems in adults with ADHD. As such, symptoms of mental fatigue, increased errors (Barrilleaux & Advokat, 2009; Kooij et al., 2010), and the tendency to invest less effort (Wiersema, van der Meere, Antrop & Roeyers, 2006) may be related to deficient processing speed abilities. By paralleling such behavioural consequences with a subtest pattern of the PSI, the clinician might be able to unravel that an extra causal force of this outcome may in fact be the

additional cognitive effort adults' invest to keep up and compensate for lower processing speed abilities. By having gained this insight, the clinician can help the adult to be aware of the impact of these underlying causes on behavioural outcomes and target specific treatment programs to improve the deficits. Furthermore, it is important to consider the wide-ranging adverse effects of processing speed on other cognitive functions (e.g. working memory, executive functions) and thus on areas of daily-life (Weiss, L. G. et al., 2010). Considering the long-lasting and severe implications of processing speed deficits for adults with ADHD, detections of these deficits should obtain high priority. Hence, clinicians are encouraged to administer the PSI subtests in order to gauge a first comprehensive picture of strengths and weaknesses regarding processing speed functions. Moreover, it is suggested to corroborate and complement the results with clinically relevant behavioural problems and a more detailed neuropsychological evaluation on working memory and executive functions.

General intelligence

The medium effects of adult ADHD on the FSIQ found in the present research are largely in accordance with earlier studies demonstrating attenuated scores in general intelligence (see section 2.1.2 and Bridgett & Walker, 2006, for a review). Supported by the general underachievement of adults with ADHD in daily-life (Kooij, 2013), the determination of the FSIQ in clinical practice continues to be important for several reasons.

Based on the finding that intelligence seems to be a strong predictor for occupational success and achievement (Gottfredson, 1997; Kuncel, Hezlett & Ones, 2004), the FSIQ can provide an indication for an overall functional potential in adults with ADHD in clinical practice. Thus, one reason to the importance of the FSIQ is that it can be used to better apprehend deficits in adults with ADHD. Another reason is that it can serve as a foundation for general cognitive functioning from which other and more specific cognitive abilities can be compared. That means, a lowered neuropsychological test performance could be attributed to compromised intellectual functions (see Bridgett & Walker, 2006, for a review). Furthermore, it has been argued that the FSIQ can offer an index for compensation (Kooij, 2013). As such, adults with a high FSIQ would have a general advantage in daily-life compared to those with a low IQ. This would suggest that especially those with lower intelligence scores are in the need of increased support and therapy. Although this seems like an ensuring argument, earlier findings show that above average intelligence in adults with ADHD does not prevent dysfunctioning (e.g. sustained attention, mental fatigue, more accidents, lower quality of life; Antshel et al., 2008).

Despite the benefits that can be drawn from the FSIQ for clinical practice, it has been asserted that the FSIQ can become invalid if there is too much subtest scatter among the indices (Lichtenberger & Kaufman, 2009c). The present investigation provides two reasons to believe that caution should be warranted in the interpretation of the FSIQ in adults with ADHD. First, their cognitive profiles were found largely heterogeneous and characterised by discrepancies across the indices. Second, the WMI and the PSI were significantly impaired to those of controls. Due to a too large amount of subtest scatter and because the FSIQ heavily relies on working memory and processing speed abilities, both reasons provide an indication for the argument that the FSIQ may in fact be unreliable in adult ADHD. Especially with the application of the WAIS-IV, the latter is essential to keep in mind since the test structure fundamentally shifted the components of the full scale, with more notions of the WMI and the PSI (Wechsler, 2009a). Given that these indices are in particular negatively affected by core symptoms of ADHD in adulthood the corresponding subtests are often marked by increased errors and omissions. Thus, adults with ADHD can be disadvantaged on these subtests and their performance may not reflect their true deficits. Prior research supports this phenomenon by concluding that performance increases when ADHD symptoms remit. This suggests that these symptoms can undermine global intellectual performance and may hide true capabilities (Goodwin et al., 2011). Considering this, it is important to interpret the WAIS-IV global intellectual functioning with respect to behavioural correlates of ADHD. That is, to evaluate whether lower performance is an artefact from this behaviour or are in fact due to deficits in neuropsychological functions.

One way to do this is to use an alternative measure calculated without working memory and processing speed abilities. The WAIS-IV offers such an approach by means of the GAI. Analyses of the present investigation indicate that underlying memory and processing speed deficits may have produced the significant and uncommonly large variabilities among the four indices. An implication of this is that a meaningful interpretation of the FSIQ cannot be guaranteed and the GAI may be regarded as a supplementary composite for the overall functioning of adults with ADHD as it provides a more realistic view on the adults' degree of capability. This can be of great assistance in practice because it provides the clinician with information to what extent deficits in working memory and processing speed impair global intellectual functioning and thereby new learning, achievement, and functional potential. Moreover, the discrepancy between the FSIQ and the GAI can be used to establish information regarding whether the adult with ADHD is similar to individuals with the same or other conditions with comparable symptomatology. It should not be

used to establish a diagnosis or to differentiate between patients but more as a source of neuropsychological interpretation to target strength and weaknesses during therapeutic intervention (Lichtenberger & Kaufman, 2009a).

In general, clinicians need to be aware that the GAI does not replace the FSIQ and is not a more valid estimate of global intellectual functioning (Loring & Bauer, 2010). Rather, it functions as a supplementary composite, which can omit ADHD related neuropsychological deficits and symptoms compromising overall functioning. In clinical decision making, it should be kept in mind that deficits in working memory and processing speed functions should not be neglected. They are essential abilities accounting for overall cognitive functioning. Neglecting them nonetheless would lead to unrealistic outcomes and at worst to inadequate achievement expectation and incorrect clinical decision making (Weiss, L. G. et al., 2010). Similar conclusion can be drawn if neuropsychological performance is not interpreted within the context of confounding factors. Results of the current investigation concerning comorbidity and stimulants are denoted in the following part.

Confounding factors

Issues in the interpretation of WAIS-IV emerge by considering variables that influence neuropsychological performance. One problem concerns the elevated rates of comorbid psychiatric conditions and another denotes the influence of stimulants or other psychopharmacological medication on performance. There is much discussion in the literature whether neuropsychological deficits are due to ADHD or are in fact an artefact of comorbidity (Faraone et al., 2000; Marchetta, Hurks, De Sonneville, et al., 2008; Marchetta, Hurks, Krabbendam, et al., 2008; Seidman, 2006; Silva et al., 2013). Others discuss the positive or even negative influence of stimulants (see section 2.1.2; Advokat, 2010; Müller et al., 2007; Turner et al., 2005). In this regard, the present investigation provides statistical data indicating that neither comorbidity nor medication explain the deficits found in adults with ADHD on the WAIS-IV. It can be suggested that the differences between adults with ADHD and controls are robust with respect to these variables. Despite the absence of statistically meaningful effects and due to the pronounced inconsistency in the literature, clinicians are advised to comprehensively evaluate comorbidity in order to consider the increased heterogeneity of symptoms. The same applies for medication. For a more accurate interpretation of the WAIS-IV in clinical practice, the following section discusses how the results can be interpreted on a neuropsychological level.

Interpretation of the WAIS-IV on a neuropsychological level

Not only on a cognitive psychological, but also on a neuropsychological level, does the WAIS-IV offer essential information to clinical care in adults with ADHD. The key for a neuropsychological interpretation of the results is the combination of indices and individual subtest scores, if necessary also task items and behavioural observations during test administration. Together these sources of information enable a more accurate interpretation of strength and weaknesses.

Considering the WAIS-IV's multi-factorial and dynamic nature, a neuropsychological interpretation of the performance can be enhanced. The multi-factorial basis refers to the interacting and complex cognitive traits¹⁴ needed for an effective subtest performance. In order to more narrowly define strengths and weaknesses in adults with ADHD the level of interpretation of the clinician should therefore be on subtest scaled scores. Limiting the interpretation to the composite scores alone may be insufficient because selective defects in specific subtests may be obscured. The reason for this is that the composites are unitary in nature and may therefore not be an adequate representation of the complex, multiple cognitive constructs that are required for successful subtest performance. This limitation becomes especially apparent when the subtests significantly differ from each other (McCloskey, Hartz & Scipioni, 2009).

Moreover, additional sources of the WAIS-IV can be utilised to increase a neuropsychological interpretation in adults with ADHD. These include, behavioural observations, subtest answering patterns (e.g. omissions), and the overall performance throughout testing (e.g. successive decrease indicative of deficits in sustained attention). Such qualitative information increases the likelihood to reliably detect variations in performance, thus indicating which abilities should be evaluated in a more specific neuropsychological follow-up testing. As such, particularly rare or unusual behaviour should be documented and emphasis should be given to variations during subtests with demands on different modalities.

Furthermore, as ADHD in adults is marked by heterogeneous symptoms, deficits may be demonstrated on one cognitive domain by means of a single test, but not in another by means of another single test. For that reason, the application of a combination of tests measuring many cognitive domains is more likely to yield information useful for clinical practice. The WAIS-IV core battery can offer a way to obtain broad information about the adults' cognitive status. More essentially, their weaknesses (interfering behaviour) and strengths (promoting behaviour to good

¹⁴ Primary and secondary capacities depicted in Table A1.

scores) can be determined from their cognitive profile. Both, strengths and weaknesses should be integrated into therapy, particularly in resource-oriented approaches. Moreover, the administration of a battery can be helpful in order to shed some light on unexplained sources of intra-individual variability. Since ADHD in adults is marked by large irregularities in behaviour, with sporadic or inconsistent attention (see Kofler et al., 2013, for a review), the behavioural responses through a prolonged testing time in combination with subtests comprising multiple and overlapping cognitive domains heightens the possibility to detect deficient performance. In fact, the administration of a whole battery in adults with ADHD has been favoured by previous research. A battery of tests was reported to increase sensitivity and negative predictive power (Lovejoy et al., 1999). Likewise, the present investigation illustrated that the application of the WAIS-IV battery detects reliable differences between adults with ADHD and controls. In order to further enhance the neuropsychological interpretation of the WAIS-IV there is the option to administer supplemental subtests (McCloskey et al., 2009). Although the current research could not detect significant effects of ADHD on supplemental subtests (LNS, CA), they can still offer a more varied perspective on the adults' use of cognitive constructs in clinical practice. A more differentiated view on ADHD allows for appropriate reactions for informed clinical decision making in the diagnostic process. Since many challenges to identify ADHD in adulthood still exist until today, the next section pertains to the issue of diagnosing ADHD in adulthood and discusses the general role of neuropsychological information in the diagnostic process.

Neuropsychological information for diagnosis

Even though the field of diagnostic expertise for adults with ADHD has progressed, the condition remains highly under-diagnosed (Kessler et al., 2006). The negative effects of unrecognised and untreated ADHD are far-reaching and detrimental, adversely affecting academic and occupational functioning, life-coping abilities, interpersonal relationships, and cognitive functioning. Resulting problems, such as hopelessness, self-denigration, and shame can lead to traumatised experiences and enhance the risk of other psychiatric disorders or chronic abnormal functioning (Ramsay, 2010; Waite et al., 2013). In an effort to prevent these negative effects, there is a necessity to improve and standardize the diagnostic process of adult ADHD. One approach for improvement has come from authors suggesting more corroborative objective data in form of neuropsychological information (Surman, 2013). Despite this suggestions, neuropsychological assessments are inherently underutilised in day-to-day clinical practice (Johnson & Conners, 2002; Woods, Lovejoy, Stutts, et al., 2002). This might be explained by the fact that many practitioners have not

been adequately trained in the assessment of ADHD in adulthood and that might hold opinions on the disorder based on misinformation that builds upon out-dated research findings (Waite & Ramsay, 2010; Waite et al., 2013). In general, the unique contribution of neuropsychological information for the diagnostic process of ADHD in adults remains unknown.

ADHD in adults is most commonly diagnosed based on the qualitative evaluation of perceived behavioural symptoms in form of self-evaluations. This approach by itself has been claimed to be problematic as the reliability of such questionnaires and interviews has often been questioned (see section 2.1.3; McCann & Roy-Byrne, 2004). It has been found that diagnostic decisions solely based on self-reports lead to a large number of false positive cases and thus to false diagnoses (Culpepper & Mattingly, 2010; McCann & Roy-Byrne, 2004; Meyer et al., 2001). The unreliability of self-evaluations is furthermore illustrated by studies examining the correlation between objectively measured deficits and actual symptoms reports. As such, results of these studies indicate only a modest, if any, relationship between neuropsychological dysfunctions and self-reported symptoms (Biederman, Petty, Fried, Black, et al., 2008; Chaytor, Schmitter-Edgecombe & Burr, 2006). The present research provides comparable findings. This suggests that self-evaluations may indeed be poor indicators for a diagnosis of ADHD if used in isolation. Apparently, they cannot fully reflect on the dimensions of functional impairment and thus provide insufficient information for diagnosis.

The inability of self-evaluations to reflect impairment caused by ADHD in adulthood may be due to different reasons of which one relates to the fact that ADHD symptoms are often not salient enough for the adults. As a result, it is the clinician's responsibility to determine a threshold at which the symptoms become clinically significant. This proves to be especially problematic because ADHD symptoms occur to a certain degree in all individuals of a healthy population (see Asherson, 2005, for a review). Furthermore, self-evaluations contain many sources of errors and biases because of a decreased self-reflection and memory deficits for retrospection. Additionally, a decreased motivation and the willingness to cooperate may result in unreliable responses. Whilst many adults consult a clinician by themselves, there are some who get motivated by third parties, thus trivializing their symptoms and associated problems in daily life. A last issue denotes subjectively manifested symptoms. Since ADHD received particularly great attention in the media and because the internet is full of inadequate information, there are many adults who assume to have ADHD (information bias). Also difficult to recognize are those adults aggravating or simulating

ADHD due to specific motives (Culpepper & Mattingly, 2010; McCann & Roy-Byrne, 2004; Meyer et al., 2001).

Considering this, there is need for more specific methods to differentiate between normal and abnormal symptom manifestations. However, apart from diagnostic classification systems that are based on behavioural criteria, no measurement with a threshold that decides on the presence or absence of ADHD in adulthood exists. Ultimately, a symptom approach to diagnosis by diagnostic classification systems seems to be a dead end as far as comprehending the nature of ADHD. At this point, it is worth emphasizing the implementation of standardised neuropsychological tools. There are several arguments in favour for their administration: (a) to provide initial aid to recognize differences to the normative population, (b) to rule out learning disorders, (c) to a priori define the behaviour of interest more narrowly and to isolate more specific facets of ADHD in a controlled setting. In all, adding a clear metric to the diagnostic process of ADHD in adulthood permits to depict the endophenotype of ADHD closer and thus its underlying pathophysiology. Moreover, a more specific augmentation and support for the behavioural symptoms outlined in the DSM-V can be provided. Given that the DSM-V essentially lacks any theoretical foundation about the nature of ADHD in adulthood, neuropsychological tests can be useful to develop a better theory relative to this classification system. Considering this, neuropsychological tests may in fact complement the diagnostic assessment process and guide objective clinical decision making. In the choice of test it is especially important to be aware of the heterogeneity of ADHD. As such, tests that cover a broad range of cognitive functions should be used. Given that the WAIS-IV can provide a broad indication of many cognitive domains it may serve as a foundation to portray the cognitive functioning in adults with ADHD. More specifically, it may serve as a basis for more precise neuropsychological tests as well as to interpret self-evaluations and behavioral problems.

Although it appears that data of neuropsychological assessments can benefit diagnostic decision making, there is insufficient sensitivity and specificity to generate diagnostic inferences from neuropsychological information itself. Although this and other research (see section 2.1.2) indicate reliable differences between adults with ADHD and controls, abnormal scores do not predict ADHD in adulthood. The same accounts for the reverse: normal scores are not predictive of an absence (Lovejoy et al., 1999). This suggests that there is currently insufficient data to support neuropsychological testing for including or excluding ADHD in adults. Moreover, neuropsychological tests have been associated with weak discriminatory power to distinguish between adults with ADHD and other psychiatric disorders or neurological conditions with similar frontal

lobe deficits (e.g. anxiety disorder, traumatic brain injury; see Woods, Lovejoy & Ball, 2002, for a review). Therefore, if used on their own they are limited in deriving conclusions for diagnoses. The discriminant validity of neuropsychological assessments has been much debated. Until now, research largely indicates poor accuracy rates, many false positive errors of neuropsychological assessments (Walker et al., 2000; Woods, Lovejoy & Ball, 2002), and a reduced ability to distinguish adults with ADHD from those with other psychiatric conditions (Holst & Thorell, 2013). Studies on the WAIS-IV suggest that the discrepancy between the FSIQ and GAI, in fact, may be valuable in a differential diagnostic process (e.g. Harrison et al., 2008). However, there is abundant room for future research in determining this. For example, in order to enhance our understanding of discriminant validity of neuropsychological measures, further research is needed to examine cognitive deficits associated with frontal-subcortical circuits. An accurate differentiation between conditions with similar dysfunctioning is crucial because many adults initially present for treatment with more obvious symptoms of comorbid conditions (e.g. anxiety) instead of ADHD. A reason for this is that ADHD cannot be directly observed and behavioural effects fall along a continuum of severity. Therefore, many adults perceive their ADHD symptoms as individual personality traits and consider them as ‘normal’, whereas those of comorbid conditions usually interfere with their daily functioning (Stieglitz, Nyberg & Hofecker-Fallahpour, 2012).

Taken together, the status of self-evaluation and the corroboration of neuropsychological assessments in the diagnostic process of adult ADHD must continue to be investigated so that no over-hasty conclusions are drawn for clinical practice thereby disregarding the usefulness of self-reports. To claim that self-reports are not useful is not the intention of the current thesis. In fact, self-evaluations are indispensable to provide key personal information about the adult presented for ADHD. These cannot be obtained by any other mean. Nevertheless, more awareness to the benefits of neuropsychological information should be raised in clinical practice. The present investigation therefore contends that the application of a multi-method framework should be central in the assessment for adult ADHD. Ideally, this should comprise a thorough anamnesis, information from self-evaluations, and behavioural observations, as well as a thorough neuropsychological assessment. Bringing together these elements, a clinician may be enabled to consider the salience of behavioural responses in a daily life context, concurrently recognizing their individual nature and how they fit together into a coherent pattern characterizing ADHD. Future studies on the current topic pertaining to the combination of measurements of diagnostically relevant symptoms and signs are needed. In addition to the role of neuropsychological assessments in diagnostic deci-

sion-making, it should be discussed whether it also serves specific purposes for treatment planning and therapy.

Neuropsychological information for treatment planning and therapy

Even though there is still insufficient evidence to support neuropsychological testing to inform treatment plans for adults with ADHD, treatment approaches often target neuropsychological functioning (e.g. problem-solving approaches, skill-based modules). This suggests that neuropsychology in fact plays an important role in the adult's improvement of symptoms. Given the fact that some adults with ADHD have cognitive impairment it may also adversely impact therapeutic intervention and functional recovery. Thus, it should indeed be addressed whether neuropsychological tests sensitive to ADHD in adulthood can be used as supplementary material to inform treatment plans and to develop realistic treatment goals. Throughout therapeutic intervention, neuropsychological information may serve different purposes. The arguments addressed in the following lines are premature and tentative because they have not been validated.

A first step to the treatment of adult ADHD constitutes the education about the disorder (Ramsay, 2010). For psychoeducation, neuropsychological information may hold particular importance as it can assist the adult to acquire additional awareness of the origins of perceived failures and how their daily functioning is affected. Moreover, it may promote an understanding about functional gains from consistent attendance in therapeutic sessions. Therefore, by integrating a cognitive element in psychoeducation, the apprehension of ADHD and the motivation for further therapy may increase. Thus, further adjustment of individual therapy in clinical settings may be enabled.

The central building block of treatment is a multimodal therapy, a combination of pharmacology (e.g. Methylphenidate) and psychotherapy. Stimulants provide an essential foundation for psychotherapy through immediate symptom relief and by reducing barriers previously interfering with learning. However, stimulants do not teach long-term coping strategies and skills to manage daily life and thus additional psychosocial therapy modes¹⁵ are most commonly applied (e.g. Cognitive-Behavioural Therapy, CBT; Ramsay, 2010). It has been argued that noteworthy impeding factors to effective psychosocial therapy in adult ADHD are cognitive and general intellectual impairment (Stieglitz et al., 2012). This may be especially true for CBT as it attempts to

¹⁵ These include for example psychoanalytic and psychodynamic approaches, cognitive remediation, and coaching. For a comprehensive overview on psychosocial therapy and alternative treatments please refer to Ramsay (2010).

improve previously problematic life domains by learning and practicing coping skills of negative schemata and maladaptive belief systems (Ramsay, 2010). Since cognitive functioning and intelligence are essential resources for learning and are closely intertwined with higher-order functioning (e.g. planning, organization), it can be suggested that if these domains are deficient, the acquisition of new information and strategies will be more challenging (Pesok, 2013) and the acquisition and the use of compensatory strategies may be obscured (see Mongia & Hechtman, 2012, for a review). In this regard, recent research has demonstrated that impaired complex prospective memory has negative effects on CBT in adults with ADHD. It has been suggested that by neglecting cognitive deficits in CBT, the adults are unable to self-initiate and execute previously agreed upon intentions for behavioural changes (Fuermaier et al., 2013). Considering this, generally observed therapeutic effects may decline and adults will necessarily require a more comprehensive therapy. It may therefore be argued that the identification of deficits in cognitive and intellectual domains goes hand in hand with addressing psychosocial therapy in adults with ADHD.

The negative impact of neuropsychological impairment on the adults' functional recovery seems immense since a failure to acquire strategies can lead to a cascade of malfunctions. Beginning with the maintenance of ADHD symptoms and increased failure in daily life, continuous underachievement, and functional impairment, neuropsychological deficits lead to a promotion of the development of negative cognitive schemata and belief systems. Decreased therapeutic motivation and progressively increasing avoidance and negative mood may be the result (Knouse & Safren, 2010). Considering this, it appears that neuropsychological testing in clinical practice provides valuable information on treatment modes involving cognitive aspects. Integrating these arguments with the current results and the findings of prior research, the Wechsler Scales and other sensitive measures to ADHD in adulthood can be suggested to establish a basis for intervention in clinical practice by providing information on the degree of cognitive resources necessary to develop and implement compensatory strategies. In this regard, it is noteworthy that helping adults to identify their personal strengths and available resources is equally relevant during therapy as addressing coping strategies (Ramsay, 2010).

Directions for future research and clinical practice

Future research should more specifically address the incremental validity of the WAIS-IV in adults with ADHD especially with regards to diagnosis and treatment planning. In addition, as cognitive signs and neurobehavioral functions change during development, thus sometimes found differently in adults with ADHD (see Seidman, 2006, for a review), further work should go some

way to address (a) the development of even more specific diagnostic criteria for ADHD in adults and (b) to expand and revise existing guidelines for assessment and diagnosis.

With regard to the first assertion, subtle but important changes have been made in the DSM-V. In its newest edition it offers a more separated view on ADHD in adulthood. However, its reliability of adult ADHD has not been examined in field trials yet. In children and adolescents with ADHD, considerably low inter-rater reliability scores on a DSM-V diagnosis have been found (Ghanizadeh, 2013). Thus, there is an urgent necessity for future research to examine the DSM-V reliability in adult ADHD. Furthermore, the criteria of the DSM-V are still not distinct enough compared to those of children. Therefore, the development of a more differentiated view on the disorder in adults should be achieved. One way to do this is to narrowly portray dysfunctional neuropsychological processes as the underlying basis for the behaviours of interest. Some authors even recommend a new subtype of ADHD predominately marked by neuropsychological impairment (e.g. Biederman et al., 2006; Nigg, Willcutt, Doyle & Sonuga-Barke, 2005). In order for this idea to progress, greater understanding is required to what extent, and under what circumstances neuropsychological functions predict ADHD related behaviour. Moreover, future work is required to examine how many of the adults with ADHD can realistically be characterised with neuropsychological impairment and if a reasonable threshold can be determined to distinguish between ADHD with and without such deficits. For example, a research question could address the clinical significance in overall functioning between these groups. In that case, this may indicate additional need for supplementary therapeutic intervention that directly target ADHD with comorbid neuropsychological impairment. However, as a first step towards a more neuropsychological oriented view in the DSM-V, it is worth to consider the inclusion of more items that stress those neuropsychological deficits characterizing ADHD in adulthood. This may aid the diagnostic process to the extent that less room is left for a clinician to interpret neuropsychological data on their own.

Considering the second claim, only scarce published material on guidelines for adults with ADHD exists, with no international agreement on assessment and diagnosis (see Seixas et al., 2012, for a review). Next to the lack of agreement across continents, there is not even a consensus on a European level. In Germany, there are many well-established guidelines that cover the child- and adolescent population (e.g. Deutsche Gesellschaft für Kinder- und Jugendpsychiatrie und Psychotherapie, 2007). However, for adults, the only guidelines formulated are based on a consensus among one group of experts (Ebert, Krause & Roth-Sackenheim, 2003). They convey the message that neuropsychological examination is not a necessary step in the diagnostic process of

ADHD in adults. However, often the scientific literature suggests otherwise and it is claimed that neuropsychological testing can provide valuable data to complement the diagnostic process (e.g. Ramsay, 2010; Surman, 2013; Woods, Lovejoy & Ball, 2002). Therefore, the questions about the specific benefits of neuropsychological testing for diagnosis and therapy arises and it should be discussed whether the necessity of the implementation of neuropsychological testing should be incorporated into existing guidelines.

Specific limitations to the research of adults with ADHD

It is of considerable importance to address specific issues that limit the conclusions that were drawn from the research on adults with ADHD. It is important to mention that despite statistical differences to the control group, the results of adults with ADHD indicate a substantial degree of distributional overlap. This means that the adults on average scored within the normative range. Yet, on an individual level, some demonstrated abnormal performance whereas a large proportion did not. Therefore, interpreting the effect of group on the composite scores and subtests as evidence for a single core deficit in all adults is problematic. It may be argued that given the variation in performance of adults with ADHD in general, the performance of the WAIS-IV is a natural result of variability occurring in a healthy population and therefore certainly unremarkable. However, previously this variability has been found specific for adults with ADHD (see Kofler et al., 2013, for a review). Consistent with that view, it has been suggested that abnormal scores in neuropsychological functioning were predictive of ADHD, but that in fact normal scores did not predict an absence of ADHD in adults (Lovejoy et al., 1999). Considering this, it is important for clinicians employing the WAIS-IV or other neuropsychological tests to not overinterpret test scores but at the same time to not rejecting them as meaningless. That is also in light of limited ecological validity of neuropsychological information in general. One possible approach to this problem would be to support the results of the neuropsychological assessments with additional sources of behavioural observation during testing as outlined above. Insights into peaks and valleys of performance due to obvious decreased concentration or motor restlessness can be a key interpretive and possibly relevant for diagnosis and treatment. The direct observation of more internal symptoms (e.g. inner agitation) influencing performance becomes more difficult. In this regard, it may be important to consider self-evaluated information gained by reports. Furthermore, clinicians should be encouraged to parallel the results with every-day behavioural problems to receive a more differentiated and objective view on ADHD problems.

Concluding remarks

The present research has extended the available knowledge on neuropsychological functioning in adults with ADHD and provides implications for the argument that intellectual and cognitive deficits are an integral component of ADHD in adulthood. It can be concluded that the WAIS-IV is sensitive to ADHD in adults and effective to provide a broad indication of their cognitive and intellectual functioning. Concluding on the results and the implications drawn for clinical practice, professionals should be encouraged to administer the full battery of the WAIS-IV in order to determine both strength and weaknesses. Particular focus should be given to subtests composing the WMI and the PSI, as well as the calculation of the GAI. These abilities can be essential corroborative in the diagnostic process of ADHD. Together, the results of the current investigation suggest that, similar to its predecessors, the WAIS-IV continues to serve as valuable measurement and as a fundamental part in the neuropsychological assessment of adults with ADHD. Particularly, as it offers additional, broad, and objective information compared to that of self-evaluations. Considering this, it may be suggested that the WAIS-IV provides a foundation for the diagnostic process as it generates a basis for further and more specific neuropsychological testing. However, it is necessary to keep in mind that under no circumstances diagnostic conclusion can be reached from these tests because they are not adequate to draw diagnostic inferences alone. Rather, they should be considered as important building blocks, which provide additional sources of information. Therefore, the present research corroborates with the conclusions of other authors that comprehensive neuropsychological assessments in adults with ADHD should not be overlooked and that they can complement the diagnostic process of adult ADHD, but that they cannot draw diagnostic inferences. Furthermore, it appears that neuropsychological information can be utilised to aid adults to become aware of their cognitive strengths and weaknesses, which, in turn, can be integrated into intervention. Together, the findings implicate that it is no longer adequate to neglect neuropsychological outcomes in adults with ADHD. With this, the current investigation emphasizes the need to revise current guidelines for management and care. These should not only define the conditions by behavioural symptoms alone but also by their neuropsychological functioning. In this way, a clearer line may be drawn between ADHD in adulthood and that occurring in childhood. Thus, current diagnostic criteria should account for the neuropsychological categorization of symptoms heterogeneity and their overlap with many other psychiatric disorders. To this end, diagnosis and treatment should expand their horizon to current neuropsychological models that characterize ADHD in its various presentations in adulthood.

6.2 Ischemic stroke patients

The current investigation indicates severe impairment of intellectual and cognitive functioning with moderate to large effect sizes of RHS and LHS patients compared to controls and some indication for dominant hemispheric dysfunction. In this respect, composites and subtests were found to be somewhat characterizing for a cognitive profile which may differentiate between RHS and LHS patients. Overall, these results are largely consistent with those of other studies indicating a wide array of neuropsychological deficits in patients with stroke during post-acute rehabilitation (see section 2.2.2). To facilitate the clinical interpretation of the WAIS-IV, the following section discusses the most important results concerning subtest performance and composite scores with respect to hemispheric dysfunction. Furthermore, their clinical implications are considered and the general role of neuropsychological assessments to treatment plans in rehabilitation is portrayed. Specific limitations to the research on stroke are provided, including directions for future research and practice.

Right hemisphere stroke

With an emphasis on the effect sizes occurring for RHS, the largest effects were found for predominantly nonverbal spatial subtests (MR, VP) and visuo-motor processing speed subtest (SS, CD). For the composites, the largest effect occurred for the PRI. In comparison, a less deviant pattern of effect sizes was observed for patients with LHS. In addition, the trend for RHS patients for visuo-spatial impairment could be supported by subgroup specific results, which illustrate meaningful differences in the subtests MR and PC, as well as the PRI. It could be argued that these effects can be explained by neglect and hemianopsia beyond that of a pure visuo-spatial deficit in RHS patients. To this end, the data gave preliminary indications that the effects of visuo-spatial and processing speed subtests increased after adjusting for neglect and hemianopsia. It is therefore likely to infer that patients with RHS experience considerable impairment in reasoning with visual stimuli, visual perceptual processing/discrimination, and attention despite neglect and hemianopsia. Previous research links patients with RHS (without neglect) to deficits in visuo-spatial and attentional functioning and supports the current findings. Hemianopsia was not specifically mentioned (Pulsipher et al., 2013). With regard to the present research, this would suggest that these WAIS-IV subtests and composite score have some degree of sensitivity to damage in the right hemisphere. Moreover, it can be assumed that these subtests fairly assess those domains of visuo-spatial processing that are more dependent on the right hemisphere than on the left. A reason for

this sensitivity may be attributed to the neuroanatomical structures in the right hemisphere affecting these subtests (e.g. right parietal lobe). In this regard, neuroimaging research indicates that even though visuo-spatial processing is linked to both hemispheres, there is still a trend that RHS patients experience a greater degree of visuo-spatial impairment than LHS patients (Ng, V. W. K. et al., 2000). This is also in line with the literature which holds the view of localization of brain functions and brain modularity (see section 2.1.1).

Left hemisphere stroke

The current research furthermore illustrates that the largest effect sizes for LHS occurred for subtests with predominately verbal and processing speed requirements (SI, VC, CD), and for the composite VCI. The smallest effects, in comparison, were observed for visuo-spatial subtests. This provides a preliminary implication that LHS patients performed better than RHS patients on the PRI and related subtests. In accordance with these results, previous studies have reported corroborating findings related to a left hemisphere bias in primarily verbal deficits compared to visuo-spatial impairment (Pulsipher et al., 2013; Wilde, 2010). Even though the subtests with verbal and processing speed requirements seem particularly demanding for LHS patients in comparison to controls, subgroup-specific analyses indicate only meaningful differences in the subtest SI between RHS and LHS patients. In contrast, the VC and IN were equally impaired in both samples. This may suggest that the subtest SI is more dependent on the left hemisphere. The main difference of SI to other language based subtests is that it requires the ability to reason with auditory verbal content (Gf). It seems possible that this ability may be deficient due to damage in the left hemisphere. This suggests a possible hemispheric asymmetry of brain areas employed in performing a reasoning task that is to a large extent verbal in nature. In this regard, the literature points out that the prefrontal and temporal lobes, particularly in the left hemisphere, are involved in retrieving and manipulating lexical and semantic information stored in memory and the integration of this information (Lee, Choi & Gray, 2007). Conversely, the cognitive requirements for the subtests VC and IN may be more dependent on both hemispheres. They primarily require the abilities to retrieve semantic knowledge from long-term memory, Gc, as well as expressive and receptive language abilities. In contrast to earlier findings, however, recent research has found that Gc and crystallised long-term memory stores are represented in distinct left-hemisphere regions (Damasio, Grabowski, Tranel, Hichwa & Damasio, 1996; Gläscher et al., 2009). Since long-term memory is fundamental for Gc (Lee et al., 2007), the non-significant differences in VC and IN may depend on deficits in the declarative knowledge storage in the temporal lobe of RHS pa-

tients, thereby interfering with the expression of Gc. It may be worthwhile for future research to consider whether RHS patients have particular deficits in encoding and retrieving information from long term storage. Future neuroimaging research should also aim to investigate the underlying neural mechanism of fluid and crystallised intelligence represented in WAIS-IV subtests.

Furthermore, the current investigation indicates a trend of LHS patients to receive on average more clinically noteworthy results (≥ 2 SDs) across the WAIS-IV subtests compared to RHS patients (see Table 11). A reason for this may be the general verbal requirements needed for an appropriate accomplishment of the subtests. In fact, lesion mapping studies support that patients must, at a minimum, comprehend verbally given instruction for each subtest in the predecessors of the WAIS-IV (Gläscher et al., 2009). Given that some of the LHS patients in this sample were marked by language deficits due to stroke, the performance across subtests could have been depressed because of a misunderstanding of task instructions. To overcome this issue, the current research only included patients who had no severe problems in comprehending speech. This was confirmed by logopaedic records. In addition, LHS patients with deficits in language abilities received particularly simple and slow test instructions, were given enough time to accomplish tasks with verbal demands, and were encouraged to use other modes of communication. Despite these precautions, the results of the current research suggest a possible negative contribution of language deficits on subtest performance of the WAIS-IV. Hence, supplementary analyses were conducted in order to further investigate this preliminary finding. The additional results demonstrate that the group of LHS⁺ patients performed significantly worse on subtests with verbal demands in comparison to LHS⁻ and RHS patients. Considering this, the effect of LHS on WAIS-IV subtests may not be primary due to deficits of cognitive requirements for the individual subtests but a result of language problems. Therefore, it is important for clinicians to be cautious regarding the interpretation of WAIS-IV results in LHS patients with language impairment. Future studies should more thoroughly investigate the verbal requirements of WAIS-IV subtests and the specific effect of language impairment post-stroke.

General intelligence of RHS and LHS patients

Regarding general intelligence in RHS and LHS patients, the FSIQ was found equally attenuated in both groups. This finding, while preliminary, suggests that RHS and LHS patients experience a similar loss of general intellectual functioning even though their cognitive profiles are characterised by a largely different amount of subtest scatter. An important implication of this is that the interpretation of the FSIQ should be warranted with caution. Particularly in LHS patients

because of their prominent deficits in working memory, processing speed abilities ($d = 0.84 - 1.14$), and their right dominant upper-limb hemiparesis, which might obscure the performance of subtests with motor demands.

A reasonable approach to tackle the issue of subtest scatter and to overcome the deficits related to LHS was to perform supplementary analyses involving the FSIQ < GAI discrepancy. These indicate that RHS and LHS patients do not differ in their GAI score. In both groups the pattern of FSIQ < GAI was significant in up to 61% of the cases. A discrepancy below the 10th percentile of the normative sample for a particular ability level occurred in 10% to 22% of the patients. This pattern should be considered as rare in the general population. From these results it can be inferred that the index of general intelligence in form of the FSIQ may be misleading for patients with a stroke. At the same time it cannot be concluded that the GAI is a better alternative since it does not reflect the heterogeneity within the cognitive profiles of RHS and LHS patients. Similar conclusions have been postulated in the literature where a direct relationship between general intelligence and brain damage has been neglected. It has been argued that by portraying these deficits in a single score would in fact result in a loss of data and therefore to unreliable estimates of cognitive functioning (Lezak et al., 2004). Considering this, it is therefore likely that the FSIQ and the GAI alone cannot depict these deficits and obscure the selective weaknesses found in the specific subtests. As a result, the general concept of intelligence may only be applied to a limited extend neuropsychological assessments of stroke patients in rehabilitation settings. Since the claim above was based on earlier Wechsler editions, there is still need to replicate previous findings on the association between general intelligence and brain damage with the WAIS-IV. This is particularly important against the backdrop of the substantial structural changes in the WAIS-IV which evoke a more dynamic and wide-ranging construct of intelligence. That is, in comparison to its predecessors, the FSIQ in the WAIS-IV reflects a different composition of cognitive functioning and is therefore likely to have a different sensitivity to neuropsychological impairment caused by neurological damage (Loring & Bauer, 2010).

Concluding on the above discussion, the following facts may be retained. First, the present investigation offers insights into the WAIS-IV cognitive pattern and intellectual functioning of first-ever unilateral, ischemic stroke patients in rehabilitation settings. Second, the results give preliminary indications that the WAIS-IV is to some extent sensitive to RHS and LHS stroke. Third, the cognitive deficits found are not conclusive and ultimate. Rather, they give a first impression about the cognitive state of being in rehabilitated stroke patients, providing an indication

for further and more specific neuropsychological testing. Several implications for clinical care can be drawn from the present research.

Clinical implications of the present research

Given that cognitive post-stroke sequelae are constantly changing during recovery, the present investigation contributes to the knowledge that stroke patients in rehabilitation suffer from severe and wide ranging cognitive dysfunctions. In practice, this information does not serve as a diagnostic marker but lends additional support to the understanding of the patients' cognitive status. Additionally, it provides means to quantify behaviour abnormalities that are associated with the stroke damage incurred in the area of the brain. In turn, the functional potential and rehabilitation success may be predicted. Therefore, the present results indirectly emphasize the previous claim that patients should be re-examined in their neuropsychological functioning at admission to rehabilitation (Heruti et al., 2002).

Whilst such an assessment at admission to rehabilitation has been claimed necessary, an appropriate assessment tool has not been agreed upon (Barker-Collo & Feigin, 2006; Gottesman & Hillis, 2010). A large variety of tools used to measure cognitive functioning post-stroke have been identified (see Lees, Fearon, Harrison, Broomfield & Quinn, 2012, for a review). In this context, some assert full assessment batteries while other favour the administration of screenings and flexible batteries (e.g. McDonnell, Bryan, Smith & Esterman, 2011; Pulsipher et al., 2013; Wilde, 2010). In practice, the latter approach is much more common because of limited time, effort, and costs. This method can accentuate deficits commonly found in stroke patients but fails to discover possible strengths. Conversely, a full battery of tests offers valuable information about both, strengths and weaknesses and generates a coherent picture of current abilities. This allows the establishment of a holistic view and realistic representation of a patient: how the stroke affects functional activities and remaining available resources. It is therefore likely to conclude that a full battery approach generates more applicable clinical judgements for the prognostic value of domain-specific cognitive functions. To this end, the current research reported core battery findings¹⁶ of the WAIS-IV. Other investigations involving patients with traumatic brain injury have provided additional support for an appropriate application of the WAIS-IV in neurological populations (Donders & Strong, 2014). The current results, however, should be considered as preliminary since this is the first study to report full WAIS-IV battery findings in patients with unilateral ischemic stroke.

¹⁶ The core battery constituted the supplementation of the subtest 'Block Design' with 'Picture Completion'.

There is more research needed to examine how the WAIS-IV differs from its predecessors with respect to sensitivity and specificity to stroke and other neurological conditions. Given the evidence of neuropsychological impairment post-stroke, the cognitive status at rehabilitation admission has been claimed necessary to identify. The extent to what this information aids clinical decision making and treatment planning is addressed in the following section.

The value of neuropsychological assessments in rehabilitation

Post-stroke neuropsychological impairments have been associated with higher mortality and health care costs (Claesson et al., 2005; Tatemichi, Paik, et al., 1994), functional recovery, rehabilitation success (Barker-Collo, Feigin, Parag, et al., 2010; Heruti et al., 2002; Nys, van Zandvoort, de Kort, van der Worp, et al., 2005; Zinn et al., 2004), and the patient's capability to comprehend therapeutic instructions and to actively participate in rehabilitation (Zinn et al., 2004). Therefore, neuropsychological assessments have been found useful for appropriate rehabilitation planning and the prediction of outpatient functional skills (Bennett, 2001). Taking this into account, it has been claimed that a higher cognitive status on admission to rehabilitation would lead to an increased participation in intervention and better functional outcomes (Heruti et al., 2002; Skidmore et al., 2010). Therefore, one of the main goals for intervention during rehabilitation should be to assess cognitive functioning early. In fact, an accumulating body of evidence emphasizes that the major burdens of stroke can be reduced and functional outcomes can be increased with the help of neuropsychological information (see Barker-Collo & Feigin, 2006, for a review). Thus, it can be hypothesised that the application of neuropsychological tests may bring about many advantages and offer valuable information for professionals in treatment planning and on functional outcomes.

The first advantage of neuropsychological assessment is to portray the patients' initial cognitive status. To illustrate, stroke patients with attention deficits are limited in their engagement in therapy as they are unable to cope with tasks that require doing several things at a time. Moreover, deficits in attention impair other higher order cognitive functions (Lezak et al., 2004), which could lead to a failure of learning and thus to a limited recovery in motor functions (Robertson et al., 1997). Furthermore, behavioural strategies trained during therapy may not be appropriately implemented due to memory and processing speed difficulties. This knowledge is especially useful during rehabilitation as it provides further perspectives how to adjust intervention to these problems, for example, by exerting less complex tasks.

Once the acute phase has been passed, a second advantage is the possibility to determine

the need for therapy. Since the recovery process of cognitive function between acute clinic and rehabilitation is most intensive (see section 2.2.2), it is especially important to re-examine the cognitive status in order to establish a basis for therapy planning. As argued above, a full test battery offers the possibility to thoroughly depict both strengths and weaknesses. For therapy planning, information about weaknesses can be used to predict potential areas of difficulty whilst information about strengths can be utilised as cognitive resources that should get reinforced.

A third central benefit is the generation of valuable information for professionals in outpatient care (Bennett, 2001). Since there is a progressively earlier release from rehabilitation settings compared to the past, patients with cognitive impairment are particularly dependent on on-going therapy as well as qualified and well-structured outpatients programs (Bennett, 2001; Zinn et al., 2004). A thorough neuropsychological evaluation may aid to estimate the patients' further needs and capability to function normally in their daily routine.

A last advantage of neuropsychological assessments denotes the increase of the awareness of strengths and weaknesses of the patients themselves. Challenged by cognitive sequelae, patients with a stroke have to take a high degree of mental effort to accomplish tasks. These daily struggles often result in feelings to never reach sense of normality. In turn, this can challenge their self-perception and can promote frustration and feelings of helplessness, anger, guilt, irritability, and agitation (Thompson & Ryan, 2009). In order to prevent or reduce these feelings, stroke patients often generate maladaptive strategies (i.e. less active, reluctant to leave home, refusal of social contacts; see Donnellan, Hevey, Hickey & O'Neill, 2006, for a review) and are at a risk to develop PSD (see Ayerbe et al., 2013, for a review). Information about neuropsychological functioning can be helpful to the extent that they depict those problems, which for the patients' remain hidden and invisible. The weaknesses that lead to daily challenges can be openly addressed and discussed in corroboration with test results. Treatment programs tailored to these limitations should then let the patients experience that they regain control over them. Most importantly, a support in recognising residual skills should not be neglected. Such psycho-educative approach, in fact, may facilitate rehabilitation engagement and consequent success.

Considering the aforementioned, it is important that, in addition to physical and occupational theory, cognitive rehabilitation should be given special attention in post-acute rehabilitation of stroke. However, until today, traditional treatment approaches place more emphasis on behavioural modification. In contrast, cognitive rehabilitation is often neglected and not routinely provided (see Barker-Collo & Feigin, 2006, for a review). Compared to 93% of the patients with

stroke who have physical goals contained in their treatment plan, only 55% were identified with cognitive-related aims. It was claimed that the main reason would likely be the under-diagnosed cognitive dysfunctions through screenings (Zinn et al., 2004). Again, this highlights the importance of a thorough neuropsychological assessment, ideally at the point of admission to rehabilitation. Notwithstanding these benefits, it is important to take into account individual factors limiting the interpretation of the current research. The next section addresses these shortcomings, which for the interpretation of the current research as well as of other neuropsychological tests need to be considered.

Specific limitations and directions for future research and clinical practice

A first issue that arises is the influence of secondary stroke sequelae on performance. The variables considered in the present research include language deficits, hemiparesis, neglect, hemianopsia, and PSD. The results implicate the possibility that language deficits additionally undermine performance in LHS patients. To prevent false interpretation in clinical practice, it is advised to initially screen for language impairments and to consult logopaedic information. In corroboration with this data, the subtests must still be considered with caution. The occurrence of a hemiparesis with an affected use of the non-dominant hand could negatively affect subtests with motor demands (e.g. SS, CD). Although, the current research and findings of others (Wilde, 2010) provide implications that hemiparesis is not an issue for performance on these tests, clinicians should still be cautious in their interpretation. It would be helpful to obtain additional sources of the WAIS-IV, such as behavioural observations of subtest pattern and the overall performance throughout assessment. This qualitative information heightens the likelihood to detect variations in performance. For the influence of neglect and hemianopsia on subtest performance with visual requirements (e.g. MR, CD) more research is needed to analyse their specific effects on performance. In the past, research associated neglect with increased cognitive impairment (Lindén et al., 2005) and suggested that it complicates the interpretation of test results (Wilde, 2010). Preliminary conclusions can be drawn that neglect and hemianopsia should be taken into account when interpreting WAIS-IV visuo-spatial subtest performance. With respect to PSD, the present research illustrates decreased performance of patients with PSD on subtests with visuo-spatial and processing speed requirements (MR, SS, VP). Although the effects were primarily found for those subtests previously identified as sensitive to RHS, they did not differ for the side of the stroke. The results are also consistent with recent research indicating an association between PSD and enhanced general cognitive impairment (see Robinson & Spalletta, 2010, for a review). Concluding

on these results, there should be increasing interest of the adverse influence of PSD on neuropsychological performance. Since cognitive dysfunctions, in turn, have been shown to negatively influence the recovery process and subsequent functional outcomes post-stroke, PSD should be accurately diagnosed and monitored with respect to these aspects as well. In future research, there should be more studies conducted to evaluate and verify the effect of PSD on WAIS-IV performance or other neuropsychological measurements.

A second problem with the application of the WAIS-IV denotes the issue of ecological validity. Although many assessment instruments measuring neuropsychological functioning are sought to be ecologically valid there is only scarce evidence for this (Goldstein, L. H. & McNeil, 2004). Therefore, drawing conclusions on everyday functioning (e.g. driving a car) from test results alone should be regarded with caution. Some support for the ecological validity of the WAIS-IV has been published recently, demonstrating moderate to strong relationships between the FSIQ, GAI, and WMI with functional abilities (Drozdick & Cullum, 2011). Perhaps, this finding provides a first implication for the importance of these composites on everyday functioning. For patients in rehabilitation facilities it may indicate that the WAIS-IV may in fact be helpful to understand the impact of cognitive deficits on daily functioning.

A third problem concerns the levels of premorbid cognitive functioning, which are the levels of cognitive abilities prior to stroke onset. In theory, they are considerably important to determine the true cognitive status of a patient. This is because the clinical significance of a result can vary depending on the premorbid levels of functioning. As such, an average performance can be a sign of a deficit because the premorbid level was high. Conversely, a decreased performance may not be indicative of a deficit because the premorbid level was low. Unfortunately, it is nearly impossible to gain objective data on premorbid functioning as the majority of patients have not been subject to neuropsychological testing prior to stroke or there is no access to such information. Therefore, most of prior research as well as the current could not be adjusted for premorbid functioning. In order to obtain some control on pre-existing cognitive deficits the present research included patients with a first-ever stroke, thereby controlling for pre-existing cognitive deficits due to stroke. In future studies and clinical practice a viable option to estimate premorbid levels may be to apply specific tests with minimal demands on current cognitive functioning, for example the National Adult Reading Test (NART; Nelson & Willison, 1991). Though, since the NART requires the reading of words it may be misleading after LHS. Due to an obvious disadvantage for some patients, other viable options to estimate premorbid functioning are the use of

demographic variables (e.g. years of education, occupation; Crawford, 2004).

A fourth and more general issue concerns the low adherence rates to standardised cognitive assessments compared to physical evaluation methods in clinical practice. Reasons for this are the length of assessment durations or the difficulty of implementation into clinical practice (Bland et al., 2013). The key to an adequate assessment is to find an appropriate balance between an over- and under-assessment. Many clinicians who are not familiar with neuropsychological testing often suppose that the application of many single tests will offer better results. However, it would be counterproductive to reach for one single subtests after the other because the findings of the previous one were inconclusive (Goldstein, L. H. & McNeil, 2004). This would only lead to unnecessary testing time and stress. It therefore appears that an initial battery-approach has the advantage to produce a useful and a thorough picture of cognitive functioning at once. Even though it is time consuming it can facilitate specific follow-up testing and therefore a more targeted use of neuropsychological tests. For older stroke patients or those who cannot endure an hour of testing, an additional option would be an abbreviated test form. Only recently, a seven-subtest short-form of the WAIS-IV has been published (Meyers, Zellinger, Kockler, Wagner & Miller, 2013). In the past, a similar version of the WAIS-R has been found useful for patients with brain damage (Callahan, Schopp & Johnstone, 1997).

A last fundamental problem is that there is a lack of a consensus for appropriate tests measuring neuropsychological functions post-stroke (Gottesman & Hillis, 2010). Overall, 300 different assessment tools used to evaluate neuropsychological functioning post-stroke throughout 408 studies have been identified (see Lees et al., 2012, for a review). Considering this disagreement among researchers, it is not surprising that practice guidelines for clinical care are undecided on this matter. Even though they comprise broad recommendation for the use of assessment tools they do not provide advice for or against them. Most importantly, they miss recommendations regarding neuropsychological assessment batteries entirely (for a complete list please refer to Duncan et al., 2005). As a consequence, there is a failure of a routine use of neuropsychological tests in clinical practice. Yet, it is precisely the use of standardised neuropsychological assessment tools encompassing all major aspects of cognitive functioning post-stroke what has frequently been requested in the literature (Gottesman & Hillis, 2010). A routine use of these tools can improve rehabilitation planning and may facilitate successful recovery.

Concluding remarks

The conclusive suggestion to emerge from the present research is that the WAIS-IV offers a way to provide a reliable insight into the cognitive status of patients with unilateral, ischemic stroke in inpatients rehabilitation facilities. In combination with prior research, several implications derived from the results should be retained. First, it appears that the WAIS-IV is sensitive to some lateralised deficit. Largely consistent with visuo-spatial hemispheric and language involvement, the respective subtests were found to differentiate between LHS and RHS stroke. It is important to remember that these results are not a verification of a unilateral cerebral dysfunction. Rather, they give an indication of a functional impairment that is often associated with the right or the left hemisphere. Second, subtests with visuo-spatial and language requirements can be useful in documenting deficits often observed in RHS and LHS patients. However, caution regarding secondary stroke sequelae in the interpretation of the results must be warranted. Third, the application of the full WAIS-IV battery in clinical practice provides many advantages. The subtest scores can provide valuable information about a variety of cognitive domains and about strengths and weaknesses. Fourth, the WAIS-IV can provide a fundamental basis to accomplish other and more specific neuropsychological tests. Together, they have considerable importance for clinical judgement and treatment planning. With this foundation, the patients may profit from a psycho-educative approach, which can lead to increased therapeutic motivation and treatment success as well as to a decreased risk to develop a PSD. For subsequent therapeutic interventions to be successful there is the need to utilize information for cognitive weaknesses and strengths to inform treatment plan. Thus, the conceptual premise is that neuropsychological functioning post-stroke should not be neglected in clinical practice. Specific aims to recover from or cope with cognitive dysfunctions should be generated in treatment plans. These will eventually assist the patients to learn and implement compensations for their cognitive deficits.

6.3 General limitations

In addition to the specific shortcomings, which have been mentioned during the discussion of the results, there are other, more general limitations that still need to be emphasised. These limitations refer to the data collection and the resulting sample as well as to the methods used and the corresponding research design. A first limitation with respect to sample and data collection denotes the non-randomization of the two samples. A non-probability sample was chosen for adults with ADHD as the sampling procedure was restricted to convenience. So, the adults were

recruited via letter or public advertisement. In terms of stroke, the adults were recruited from predefined rehabilitation facilities based on the criteria of inclusion. They were personally informed about the present research and asked for participation. A response bias may have resulted from both of these recruitment strategies, which could have led to a biased constellation of the samples. The much lower dropout rate suggests that the bias, if existent, is certainly smaller in the stroke group. Moreover, compared to other studies, the demographic characteristics of the adults with ADHD or stroke were similar (see section 4.1.1). As a result, the two samples appear representative on the basis of observable variables.

A second limitation regarding sample and data collection is the issue of control variables due to missing diagnostic information regarding stroke. The present research included a homogeneous sample of patients with unilateral, ischemic stroke due to MCA occlusion. The sample was selected based on the information of the clinical reports at the rehabilitation facilities. In the majority of the cases, the clinical reports did not contain information of neuroimaging data, including stroke distribution and size. Therefore, the strokes could not be verified and additional control information regarding distribution (e.g. frontal, parietal, temporal, occipital) could not be considered in the present research. Thus, the main weakness of the research involving stroke patients was the paucity of neurological information regarding stroke.

A third limitation must be acknowledged that denotes the test administration in the stroke sample. Given that stroke is a complex neurological condition, multiple parameters interactively influence cognitive functioning. Encompassing all factors of stroke sequelae (e.g. language deficit) and personal variables (e.g. premorbid functioning) simultaneously is difficult. Nevertheless, the present research still attempted to consider those variables in the interpretation of the results which have continuously been associated with depressed post-stroke neuropsychological functioning (see section 2.2.2). In this regard, the deficits of language functions after LHS concern further emphasis. The present research received information regarding deficits in language from logopaedic reports. A major limitation is that these reports did not contain information regarding specific diagnoses of language impairment. In order to still consider the implication of language on test performance, the current research systematically clustered patients with LHS based on logopaedic information (see section 4.1.3). More reliable methods, such as the National Institute of Health Stroke Scale (NIHSS) scale were not available. This is a weakness of the present research and limits the conclusions drawn on the influence of language impairment on test performance.

A fourth limitation of sample and data collection pertains to the size of the samples. A priori power analyses defined the sample sizes for adults with ADHD or stroke as sufficient for medium effects. However, the size of the samples can still be too small to detect significant effects with small effect sizes. This is especially true for sub-group inferential statistics.

A final limitation concerning the method and the research design denotes the recruitment of cross-sectional data, which prevents to determine causality and the natural history of cognitive functions in both samples. Furthermore, it is impossible to apply random treatment with respect to ADHD or stroke research. Instead, a randomly matched control group was used respectively to compare the results for ADHD or stroke patients.

7 Main conclusion

The present research has extended the knowledge on intellectual and cognitive functioning in adult ADHD and in patients with unilateral, ischemic stroke in inpatients rehabilitation by means of the WAIS-IV. The empirical findings provide an initial understanding for the interpretation of the composites, indices, and subtests in a psychiatric neuro-developmental and neurological sample. The present data serves to regard the WAIS-IV as a fundamental part in neuropsychological assessments to provide a broad indication of the current cognitive status in adults with ADHD or stroke. It sets an initial stage for further neuropsychological testing, diagnostic corroboration, and treatment planning in both clinical psychiatric settings and in neurological domains. By interpreting the WAIS-IV performance in these domains, it is important to remain cautious regarding general intellectual functioning. Reliable and relevant information may be attained through deemphasizing the FSIQ and by highlighting the subtest scatter and index discrepancies. Therefore, the quintessence of the present research is to stop seeing the scale as a rigid approach to identify mere cognitive and intellectual deficits. Instead, it is advised to regard the WAIS-IV as tool, which considers intelligence as an interactive and dynamic construct. This is to say that the indices and each subtest by itself assess interacting and complex cognitive traits. Comprehending these abilities together is critical to understand their clinical and behavioural correlates and in turn the expression as well as the development of general intelligence over the course of a disorder.

References

- Ackermann, H. & Schönle, P.-W. (2012). Multiprofessionelle neurologische Rehabilitation. Leitlinien für die Diagnostik und Therapie in der Neurologie. Deutsche Gesellschaft für Neurologie. In H. C. Diener & C. Weimar (Eds.), *Leitlinien für Diagnostik und Therapie in der Neurologie. Herausgegeben von der Kommission „Leitlinien“ der Deutschen Gesellschaft für Neurologie* (5th ed., pp. 1-9). Stuttgart: Thieme Verlag.
- Adler, L. A., Shaw, D., Sitt, D., Maya, E. & Ippolito, M. (2009). Issues in the diagnosis and treatment of adult ADHD by primary care physicians. *Primary Psychiatry, 16*, 57-63.
- Advokat, C. (2010). What are the cognitive effects of stimulant medications? Emphasis on adults with Attention-Deficit/Hyperactivity Disorder (ADHD). *Neuroscience & Biobehavioral Reviews, 34*, 1256-1266.
- Alderson, R. M., Kasper, L. J., Hudec, K. L. & Patros, C. H. G. (2013). Attention-Deficit/Hyperactivity Disorder (ADHD) and working memory in adults: a meta-analytic review. *Neuropsychology, 27*, 287-302.
- American Academy of Child and Adolescent Psychiatry (2007). Practice parameter for the assessment and treatment of children and adolescents with Attention-Deficit/Hyperactivity Disorder. *Journal of the American Academy of Child and Adolescent Psychiatry 46*, 894-921.
- American Heart Association (2010). Heart disease and stroke statistics-2010 update: a report from the American Heart Association. *Circulation, 121*, e46-e215.
- American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders*. Washington, DC: Author.
- American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders (Text revision)* (4th). Washington, DC: Author.
- American Psychiatric Association (2013). *Diagnostic and statistical manual of mental disorders* (5th). Arlington, VA: American Psychiatric Publishing.
- Antshel, K. M., Faraone, S. V., Maglione, K., Doyle, A., Fried, R., Seidman, L. J. et al. (2008). Is adult Attention Deficit Hyperactivity Disorder a valid diagnosis in the presence of high IQ? *Psychological Medicine, 24*, 1-11.
- Antshel, K. M., Faraone, S. V., Maglione, K., Doyle, A. E., Fried, R., Seidman, L. J. et al. (2010). Executive functioning in high-IQ adults with ADHD. *Psychological Medicine, 40*, 1909-1918.
- Arbeitsgruppe Bildungsberichterstattung. (2012). *Bildung in Deutschland 2012 - Ein indikatorengestützter Bericht mit einer Analyse zur kulturellen Bildung im Lebenslauf*. Bielefeld: Bertelsmann Verlag.
- Aron, A. R., Dowson, J. H., Sahakian, B. J. & Robbins, T. W. (2003). Methylphenidate improves response inhibition in adults with Attention-Deficit/Hyperactivity Disorder. *Biological Psychiatry, 54*, 1465-1468.
- Asherson, P. (2005). Clinical assessment and treatment of Attention Deficit Hyperactivity Disorder in adults. *Expert Review of Neurotherapeutics, 5*, 525-539.
- Aström, M., Adolfsson, R. & Asplund, K. (1993). Major depression in stroke patients. A 3-year longitudinal study. *Stroke, 24*, 976-982.

- Aycicegi-Dinn, A., Dervent-Ozbek, S., Yazgan, Y., Bicer, D. & Dinn, W. M. (2011). Neurocognitive correlates of adult Attention-Deficit/Hyperactivity Disorder in a Turkish sample. *Attention Deficit and Hyperactivity Disorders, 3*, 41-52.
- Ayerbe, L., Ayis, S., Wolfe, S. D. A. & Rudd, A. G. (2013). Natural history, predictors and outcomes of depression after stroke: systematic review and meta-analysis. *British Journal of Psychiatry 202*, 14-21.
- Baddeley, A. D. (1986). *Working Memory*. Oxford, UK: Oxford University Press.
- Baddeley, A. D. & Hitch, G. J. (1974). Working memory. In G. H. Bower (Ed.), *The psychology of learning and motivation: advances in research and theory* (Bd. 8, pp. 47-90). New York, NY: Academic Press.
- Ballard, C., Rowan, E., Stephens, S., Kalaria, R. & Kenny, R. A. (2003). Prospective follow-up study between 3 and 15 months after stroke: improvements and decline in cognitive function among dementia-free stroke survivors >75 years of age. *Stroke, 34*, 2440-2444.
- Ballard, C., Stephens, S., McLaren, A., Wesnes, K., Kenny, R. A., Burton, E. et al. (2002). Neuropsychological deficits in older stroke patients. *Annals of the New York Academy of Sciences, 977*, 179-182.
- 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*, 637-644.
- Barker-Collo, S. & Feigin, V. (2006). The impact of neuropsychological deficits on functional stroke outcomes. *Neuropsychology Review, 16*, 53-64.
- Barker-Collo, S., Feigin, V., Lawes, C., Senior, H. & Parag, V. (2010). Natural history of attention deficits and their influence on functional recovery from acute stages to 6 months after stroke. *Neuroepidemiology, 35*, 255-262.
- Barker-Collo, S., Feigin, V. L., Parag, V., Lawes, C. M. M. & Senior, H. (2010). Auckland Stroke Outcomes Study: part 2: cognition and functional outcomes 5 years poststroke. *Neurology, 75*, 1608-1616.
- Barker-Collo, S., Starkey, N., Lawes, C. M. M., Feigin, V., Senior, H. & Parag, V. (2012). Neuropsychological profiles of 5-year ischemic stroke survivors by oxfordshire stroke classification and hemisphere of lesion. *Stroke, 43*, 50-55.
- Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: constructing a unifying theory of ADHD. *Psychological Bulletin, 11*, 65-94.
- Barkley, R. A. (1999). Response inhibition in Attention-Deficit Hyperactivity Disorder. *Mental Retardation and Developmental Disabilities, 5*, 177-184.
- Barkley, R. A., Fischer, M., Smallish, L. & Fletcher, K. (2002). The persistence of Attention-Deficit/Hyperactivity Disorder into young adulthood as a function of reporting source and definition of disorder. *Journal of Abnormal Psychology, 111*, 279-289.
- Barkley, R. A., Murphy, K. R., Dupaul, G. J. & Bush, T. (2002). Driving in young adults with Attention Deficit Hyperactivity Disorder: knowledge, performance, adverse outcomes, and the role of executive functioning. *Journal of the International Neuropsychological Society, 8*, 655-672.
- Barkley, R. A., Murphy, K. R. & Fischer, M. (2008). *ADHD in adults: what the science says*. New York, NY: Guilford.
- Barrilleaux, K. & Advokat, C. (2009). Attribution and self-evaluation of Continuous Performance Test task performance in medicated and unmedicated adults with ADHD. *Journal of Attention Disorders, 12*, 291-298.

- Beck, S. J., Hanson, C. A., Puffenberger, S. S., Benninger, K. L. & Benniger, W. B. (2010). A controlled trial of working memory training for children and adolescents with ADHD. *Journal of Clinical Child and Adolescent Psychology, 39*, 825-836.
- Bekker, E. M., Overtom, C. C., Kenemans, J. L., Kooij, J. J. S., De Noord, I., Buitelaar, J. K. et al. (2005). Stopping and changing in adults with ADHD. *Psychological Medicine, 35*, 807-816.
- Bennett, T. L. (2001). Neuropsychological evaluation in rehabilitation planning and evaluation of functional skills. *Archives of Clinical Neuropsychology, 16*, 237-253.
- Benson, N., Hulac, D. M. & Kranzler, J. H. (2010). Independent examination of the Wechsler Adult Intelligence Scale - Fourth Edition (WAIS-IV): what does the WAIS-IV measure? *Psychological Assessment, 22*, 121-130.
- Berg, A., Palomäki, H., Lehtihalmes, M., Lönnqvist, J. & Kaste, M. (2003). Poststroke depression: an 18-months follow-up. *Stroke, 34*, 138-143.
- Bhagal, S. K., Teasell, R., Foley, N. & Speechley, M. (2004). Lesion location and poststroke depression: systematic review of the methodological limitations in the literature. *Stroke, 35*, 794-802.
- Biederman, J., Faraone, S. V., Monuteaux, M. C., Bober, M. & Cadogen, E. (2004). Gender effects on Attention-Deficit/Hyperactivity Disorder in adults, revisited. *Biological Psychiatry, 55*, 692-700.
- Biederman, J., Faraone, S. V., Spencer, T. J., Wilens, T., Norman, D., Lapey, K. et al. (1993). Patterns of psychiatric comorbidity, cognition, and psychosocial functioning in adults with Attention Deficit Hyperactivity Disorder. *American Journal of Psychiatry, 150*, 1792-1798.
- Biederman, J., Mick, E. & Faraone, S. V. (2000). Age-dependent decline of symptoms of Attention Deficit Hyperactivity Disorder: impact of remission definition and symptom type. *American Journal of Psychiatry, 157*, 816-818.
- Biederman, J., Petty, C. R., Clarke, A., Lomedico, A. & Faraone, S. V. (2011). Predictors of persistent ADHD: an 11-year follow-up study. *Journal of Psychiatric Research, 45*, 150-155.
- Biederman, J., Petty, C. R., Evans, M., Small, J. & Faraone, S. V. (2010). How persistent is ADHD? A controlled 10-year follow-up study of boys with ADHD. *Psychiatry Research, 177*, 299-304.
- Biederman, J., Petty, C. R., Fried, R., Black, S., Faneuil, A., Doyle, A. E. et al. (2008). Discordance between psychometric testing and questionnaire-based definitions of executive function deficits in individuals with ADHD. *Journal of Attention Disorders, 12*, 92-102.
- Biederman, J., Petty, C. R., Fried, R., Fontanella, J., Doyle, A. E., Seidman, L. J. et al. (2006). Impact of psychometrically defined deficits of executive functioning in adults with Attention Deficit Hyperactivity Disorder. *American Journal of Psychiatry, 163*, 1730-1738.
- Biederman, J., Petty, C. R., Fried, R., Kaiser, R., Dolan, C. R., Schoenfeld, S. et al. (2008). Educational and occupational underattainment in adults with Attention-Deficit/Hyperactivity Disorder: a controlled study. *Journal of Clinical Psychiatry, 69*, 1217-1222.
- Biederman, J., Petty, C. R., Monuteaux, M. C., Fried, R., Byrne, D., Mirto, T. et al. (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*, 409-417.
- Bland, M. D., Sturmoski, A., Whitson, M., Harris, H., Connor, L. T., Fucetola, R. et al. (2013). Clinician adherence to a standardized assessment battery across settings and disciplines in a poststroke rehabilitation population. *Archives of Physical Medicine and Rehabilitation, 94*, 1048-1053.e1.

- Boonstra, A. M., Kooij, J. J. S., Oosterlaan, J., Sergeant, J. A. & Buitelaar, J. K. (2010). To act or not to act, that's the problem: primarily inhibition difficulties in adult ADHD. *Neuropsychology, 24*, 209-221.
- Boonstra, A. M., Oosterlaan, J., Sergeant, J. A. & Buitelaar, J. K. (2005). Executive functioning in adult ADHD: a meta-analytic review. *Psychological Medicine, 35*, 1097-1108.
- Bornstein, R. A. & Matarazzo, J. D. (1982). Wechsler VIQ versus PIQ differences in cerebral dysfunction: a literature review with emphasis on sex differences. *Journal of Clinical Neuropsychology, 4*, 319-334.
- Bortz, J. & Döring, N. (2006). *Forschungsmethoden und Evaluation für Human- und Sozialwissenschaftler* (4th). Heidelberg: Springer.
- Bradac, G. B. (2011). *Cerebral angiography: normal anatomy and vascular pathology*. New York, NY: Springer.
- 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*, 1-14.
- Broca, P. (1863). Localization of cerebral functions. Location of articulate language. *Bulletin of the Society of Anthropology, 4*, 200-203.
- Broca, P. (1865). On the location of the faculty of articulate language in the left hemisphere of the brain. *Bulletin of the Society of Anthropology, 6*, 377-393.
- Brodmann, K. (1909). *Vergleichende Lokalisationslehre der Großhirnrinde in ihren Prinzipien dargestellt auf Grund des Zellenbaues*. Leipzig: Johann Ambrosius Barth Verlag.
- Brown, T. E. (1996). *Brown Attention-Deficit Disorder Scales*. San Antonio, TX: The Psychological Corporation.
- Bundesarbeitsgemeinschaft für Rehabilitation (1995). *Empfehlungen zur Neurologischen Rehabilitation von Patienten mit schweren und schwersten Hirnschädigungen in den Phasen B und C*. Frankfurt/Main: BAR Publikation.
- Burns, N. R., Nettelbeck, T. & McPherson, J. (2009). Attention and intelligence. *Journal of Individual Differences, 30*, 44-57.
- Buxbaum, L. J., Ferraro, M. K., Veramonti, T., Farne, A., Whyte, J., Ladavas, E. et al. (2004). Hemispatial neglect: subtypes, neuroanatomy, and disability. *Neurology, 62*, 122-127.
- Callahan, C. D., Schopp, L. & Johnstone, B. (1997). Clinical utility of a seven subtest WAIS-R short form in the neuropsychological assessment of traumatic brain injury. *Archives of Clinical Neuropsychology, 12*, 133-138.
- Canivez, G. L. & Watkins, M. W. (2012). Investigation of the factor structure of the Wechsler Adult Intelligence Scale - Fourth Edition (WAIS-IV): exploratory and higher order factor analyses. *Psychological Assessment, 22*, 827-836.
- Carroll, J. B. (1993). *Human cognitive abilities: a survey of factor-analytic studies*. Cambridge, UK: Cambridge University Press.
- Carson, A. J., MacHale, S., Allen, K., Lawrie, S. M., Dennis, M., House, A. et al. (2000). Depression after stroke and lesion location: a systematic review. *The Lancet, 356*, 122-126.
- Castellanos, F. X., Sonuga-Barke, E. J. S., Milham, M. P. & Tannock, R. (2006). Characterizing cognition in ADHD: beyond executive dysfunction. *Trends in Cognitive Sciences, 10*, 117-123.
- Castellanos, F. X. & Tannock, R. (2002). Neuroscience of Attention-Deficit/Hyperactivity Disorder: the search for endophenotypes. *Nature Reviews Neuroscience, 3*, 617-628.
- Chaytor, N., Schmitter-Edgecombe, M. & Burr, R. (2006). Improving the ecological validity of executive functioning assessment. *Archives of Clinical Neuropsychology, 21*, 217-227.

- Chemerinski, E. & Robinson, R. G. (2000). The neuropsychiatry of stroke. *Psychosomatics*, *41*, 5-14.
- Cherry, E. C. (1953). Some experiments on the recognition of speech with one and two ears. *Journal of the Acoustical Society of America*, *25*, 975-979.
- Claesson, L., Linden, T., Skoog, I. & Blomstrand, C. (2005). Cognitive impairment after stroke - impact on activities of daily living and costs of care for elderly people. The Göteborg 70+ Stroke Study. *Cerebrovascular Diseases*, *19*, 102-109.
- Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd). Hillsdale, NJ: Lawrence Earlbaum Associates.
- Cohen, R. A. (1993). *The neuropsychology of attention*. New York, NY: Plenum Press.
- Conners, C. K., Erhardt, D. & Sparrow, E. (1999). *Conners' adult ADHD rating scales (CAARS)*. North Tonawanda, NY: Multi-Health Systems.
- Cowan, N. (1988). Evolving conceptions of memory storage, selective attention, and their mutual constraints within the human information-processing system. *Psychological Bulletin*, *104*, 163-191.
- Crawford, J. D. (1991). The relationship between tests of sustained attention and fluid intelligence. *Personality and Individual Differences*, *12*, 599-611.
- Crawford, J. D. (2004). Psychometric foundations of neuropsychological assessment. In L. H. Goldstein & J. E. McNeil (Eds.), *Clinical neuropsychology: a practical guide to assessment and management for clinicians* (pp. 121-140). Chichester, UK: Wiley.
- Culpepper, L. & Mattingly, G. (2010). Challenges in identifying and managing Attention-Deficit/Hyperactivity Disorder in adults in the primary care setting: a review of the literature. *Primary Care Companion to the Journal of Clinical Psychiatry*, *12*, 794-795.
- D'Esposito, M., Aguirre, G. K., Zarahn, E., Ballard, D., Shin, R. K. & Lease, J. (1998). Functional MRI studies of spatial and nonspatial working memory. *Cognitive Brain Research*, *7*, 1-13.
- Damasio, H., Grabowski, T. J., Tranel, D., Hichwa, R. D. & Damasio, A. R. (1996). A neural basis for lexical retrieval. *Nature*, *380*, 499-505.
- Davies, R. D., Jones, D. M. & Taylor, A. (1984). Selective and sustained attention tasks: individual and group differences. In R. Parasuraman & R. D. Davies (Eds.), *Varieties of attention* (pp. 395-447). Orlando, FL: Academic Press.
- de Graaf, R., Kessler, R. C., Fayyad, J., ten Have, M., Alonso, J., Angermeyer, M. et al. (2008). The prevalence and effects of adult Attention-Deficit/Hyperactivity Disorder (ADHD) on the performance of workers: results from the WHO World Mental Health Survey Initiative. *Occupational and Environmental Medicine*, *65*, 835-842.
- del Campo, N., Chamberlain, S. R., Sahakian, B. J. & Robbins, T. W. (2011). The roles of dopamine and noradrenaline in the pathophysiology and treatment of Attention-Deficit/Hyperactivity Disorder. *Biological Psychiatry*, *69*, e145-e157.
- Deutsche Gesellschaft für Kinder- und Jugendpsychiatrie und Psychotherapie (2007). *Deutsche Gesellschaft für Kinder- und Jugendpsychiatrie und Psychotherapie. Leitlinien zur Diagnostik und Therapie von psychischen Störungen im Säuglings-, Kindes- und Jugendalter*(3rd). Cologne: Deutscher Ärzte Verlag.
- Dinn, W. M., Robbins, N. C. & Harris, C. L. (2001). Adult Attention-Deficit/Hyperactivity Disorder: neuropsychological correlates and clinical presentation. *Brain and Cognition*, *46*, 114-121.
- Dohmen, C., Garlip, G., Sitzer, M., Siebler, M., Malevani, J., Kessler, K. R. et al. (2006). Post-stroke-depression. *Fortschritte der Neurologie Psychiatrie*, *74*, 257-262.

- Donders, J. & Strong, C.-A. H. (2014). Clinical utility of the Wechsler Adult Intelligence Scale - Fourth Edition after traumatic brain injury. *Assessment*. Retrieved October 1, 2014, from <http://asm.sagepub.com/content/early/2014/04/20/1073191114530776.long>
- Donnellan, C., Hevey, D., Hickey, A. & O'Neill, D. (2006). Defining and quantifying coping strategies after stroke: a review. *Journal of Neurology, Neurosurgery & Psychiatry*, *77*, 1208-1218.
- Doshi, J. A., Hodgkins, P., Kahle, J., Sikirica, V., Cangelosi, M. J., Setyawan, J. et al. (2012). Economic impact of childhood and adult Attention-Deficit/Hyperactivity Disorder in the United States. *Journal of the American Academy of Child & Adolescent Psychiatry*, *51*, 990-1002.e2.
- Doyle, A. E., Biederman, J., Seidman, L. J., Weber, W. & Faraone, S. V. (2000). Diagnostic efficiency of neuropsychological test scores for discriminating boys with and without Attention Deficit-Hyperactivity Disorder. *Journal of Consulting and Clinical Psychology*, *68*, 477-488.
- Drozdzick, L. W. & Cullum, C. M. (2011). Expanding the ecological validity of WAIS-IV and WMS-IV with the Texas Functional Living Scale. *Assessment*, *18*, 141-155.
- Duncan, P. W., Wallace, D., Lai, S. M., Johnson, D., Embretson, S. & Laster, L. J. (1999). The Stroke Impact Scale Version 2.0: evaluation of reliability, validity, and sensitivity to change. *Stroke*, *30*, 2131-2140.
- Duncan, P. W., Zorowitz, R., Bates, B., Choi, J. Y., Glasberg, J. J., Graham, G. D. et al. (2005). Management of adult stroke rehabilitation care: a clinical practice guideline. *Stroke*, *36*, 100-143.
- Durston, S., Pol, H. E., Schnack, H. G., Buitelaar, J. K., Steenhuis, M. P., Minderaa, R. B. et al. (2004). Magnetic resonance imaging of boys with Attention-Deficit/Hyperactivity Disorder and their unaffected siblings. *Child & Adolescent Social Work Journal*, *21*, 332-340.
- Ebert, D., Krause, J. & Roth-Sackenheim, C. (2003). ADHS im Erwachsenenalter - Leitlinien auf der Basis eines Expertenkonsensus mit Unterstützung der DGPPN. *Nervenarzt*, *74*, 939-946.
- Engle, R. W. (2002). Working memory capacity as executive attention. *Current Directions in Psychological Science*, *11*, 19-23.
- Engle, R. W., Tuholski, S. W., Laughlin, J. E. & Conway, A. R. A. (1999). Working memory, short-term memory, and general fluid intelligence: a latent-variable approach. *Journal of Experimental Psychology: General*, *128*, 309-331.
- Epstein, J. N., Conners, C. K., Sitarenios, G. & Erhardt, D. (1998). Continuous Performance Test results of adults with Attention Deficit Hyperactivity Disorder. *The Clinical Neuropsychologist*, *12*, 155-168.
- Epstein, J. N., Johnson, D. E. & Conners, C. K. (2001). *CAADID. The Conner's Adult ADHD Diagnostic Interview for DSM-IV*. North Tonawanda, NY: MHS Inc.
- Epstein, J. N., Johnson, D. E., Varia, I. M. & Conners, C. K. (2001). Neuropsychological assessment of response inhibition in adults with ADHD. *Journal of Clinical and Experimental Neuropsychology*, *23*, 362-371.
- 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*, 159-165.
- Faraone, S. V., Biederman, J., Spencer, T., Wilens, T., Seidman, L. J., Mick, E. et al. (2000). Attention-Deficit/Hyperactivity Disorder in adults: an overview. *Society of Biological Psychiatry*, *48*, 9-20.

- Faraone, S. V. & Doyle, A. E. (2001). The nature and heritability of Attention-Deficit/Hyperactivity Disorder. *Child and Adolescent Psychiatric Clinics of North America*, *10*, 299-316.
- Faraone, S. V., Perlis, R. H., Doyle, A. E., Smoller, J. W., Goralnick, J. J., Holmgren, M. A. et al. (2005). Molecular genetics of Attention-Deficit/Hyperactivity Disorder. *Biological Psychiatry*, *57*, 1313-1323.
- Feigin, V. L., Lawes, C. M., Bennett, D. A., Barker-Collo, S. L. & Parag, V. (2009). Worldwide stroke incidence and early case fatality reported in 56 population-based studies: a systematic review. *Lancet Neurology*, *8*, 355-369.
- Fischer, M., Barkley, R. A., Smallish, L. & Fletcher, K. (2005). Executive functioning in hyperactive children as young adults: attention, inhibition, response perseveration, and the impact of comorbidity. *Developmental Neuropsychology*, *27*, 107-133.
- Fischer, M., Barkley, R. A., Smallish, L. & Fletcher, K. (2007). Hyperactive children as young adults: driving abilities, safe driving behavior, and adverse driving outcomes. *Accident Analysis and Prevention*, *39*, 94-105.
- Flanagan, D. P., Genshaft, J. L. & Harrison, P. L. (1997). *Contemporary intellectual assessment*. New York, NY: Guilford Press.
- Flanagan, D. P., Genshaft, J. L. & Harrison, P. L. (2012). *Contemporary intellectual assessment* (3rd ed.). New York, NY: Guilford Press.
- Fodor, J. A. (1983). *The modularity of mind*. Cambridge, MA: MIT Press.
- Folstein, M. F., Folstein, S. E. & McHugh, P. R. (1975). Mini-Mental-State: A practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research*, *12*, 189-198.
- 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*, 543-555.
- Fuermaier, A. B. M., Tucha, L., Koerts, J., Aschenbrenner, S., Westermann, C., Weisbrod, M. et al. (2013). Complex prospective memory in adults with Attention Deficit Hyperactivity Disorder. *Plos One*, *8*, e58338.
- Gadow, K., Sprafkin, J. & Weiss, M. D. (1999). *Adult symptom inventory*. New York, NY: Checkmate Plus.
- Gall, F. J. & Spurzheim, G. (1810-1819). *Anatomie et physiologie du systeme nerveux en general et du cerveau en particulier* [Sketch of the new anatomy and physiology of the brain and nervous system of Drs. Gall and Spurzheim, considered as comprehending a complete system of zoonomy with observations on its tendency to the improvement of education]. Paris: Schoell.
- Gerritsen, M. J. J., Berg, I. J., Deelman, B. G., Visser-Keizer, A. C. & Jong, B. M. (2003). Speed of information processing after unilateral stroke. *Journal of Clinical and Experimental Neuropsychology*, *25*, 1-13.
- Ghanizadeh, A. (2013). Agreement between Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, and the proposed DSM-V Attention Deficit Hyperactivity Disorder diagnostic criteria: an exploratory study. *Comprehensive Psychiatry*, *54*, 7-10.
- Gitelman, D. R. (2003). Attention and its disorders: imaging in clinical neuroscience. *British Medical Bulletin*, *65*, 21-34.
- Gjervan, B., Torgersen, T., Nordahl, H. M. & Rasmussen, K. (2012). Functional impairment and occupational outcome in adults with ADHD. *Journal of Attention Disorders*, *16*, 544-552.
- Gläscher, J., Tranel, D., Paul, L. K., Rudrauf, D., Rorden, C., Hornaday, A. et al. (2009). Lesion mapping of cognitive abilities linked to intelligence. *Neuron*, *61*, 681-691.

- Glosser, G. & Goodglass, H. (1990). Disorders in executive control functions among aphasic and other brain-damaged patients. *Journal of Clinical and Experimental Neuropsychology*, *12*, 485-501.
- Golden, C. J. (1978). *Stroop Color and Word Test: Manual for clinical and experimental uses*. Chicago, IL: Stoelting.
- Goldstein, L. B., Adams, R., Alberts, M. J., Appel, L. J., Brass, L. M., Bushnell, C. D. et al. (2006). Primary prevention of ischemic stroke: a guideline from the American Heart Association/American Stroke Association Stroke Council: cosponsored by the Atherosclerotic Peripheral Vascular Disease Interdisciplinary Working Group; Cardiovascular Nursing Council; Clinical Cardiology Council; Nutrition, Physical Activity, and Metabolism Council; and the Quality of Care and Outcomes Research Interdisciplinary Working Group: the American Academy of Neurology affirms the value of this guideline. *Stroke*, *37*, 1583-1633.
- Goldstein, L. H. & McNeil, J. E. (2004). General introduction: What is the relevance of neuropsychology for clinical psychology practice? In L. H. Goldstein & J. E. McNeil (Eds.), *Clinical neuropsychology: a practical guide to assessment and management for clinicians* (pp. 3-20). Chichester, UK: Wiley.
- Goodwin, E., Gudjonsson, G. H., Sigurdsson, J. F. & Young, S. (2011). The impact of ADHD symptoms on intelligence test achievement and speed of performance. *Personality and Individual Differences*, *50*, 1273-1277.
- Görtz-Dorten, A. & Döpfner, M. (2014). Assessment of ADHD. *Pharmakon*, *2*, 37-42.
- Gottesman, R. F. & Hillis, A. E. (2010). Predictors and assessment of cognitive dysfunction resulting from ischaemic stroke. *The Lancet Neurology*, *9*, 895-905.
- Gottfredson, L. S. (1997). Why g matters: the complexity of everyday life. *Intelligence*, *24*, 79-132.
- Gropper, R. J., Gotlieb, H., Kronitz, R. & Tannock, R. (2014). Working memory training in college students with ADHD or LD. *Journal of Attention Disorders*, *18*, 331-345.
- Hacke, W. (2010). *Neurologie* (13th). Heidelberg: Springer.
- Hackett, M. L., Yapa, C., Parag, V. & Anderson, C. S. (2005). Frequency of depression after stroke: a systematic review of observational studies. *Stroke*, *36*, 1330-1340.
- Halleland, H. B., Haavik, J. & Lundervold, A. J. (2012). Set-shifting in adults with ADHD. *Journal of the International Neuropsychological Society*, *18*, 728-737.
- Harrison, A. G., DeLisle, M. M. & Parker, K. C. H. (2008). An investigation of the General Abilities Index in a group of diagnostically mixed patients. *Journal of Psychoeducational Assessment*, *26*, 247-259.
- Hartman, D. E. (2009). Wechsler Adult Intelligence Scale IV (WAIS IV): return of the gold standard. *Applied Neuropsychology*, *16*, 85-87.
- Harvey, E., Danforth, J. S., McKee, T. E., Ulaszek, W. R. & Friedman, J. L. (2003). Parenting of children with Attention-Deficit/Hyperactivity Disorder (ADHD): the role of parental ADHD symptomatology. *Journal of Attention Disorders*, *7*, 31-42.
- Hautzinger, M., Kühner, C. & Keller, F. (2006). *Beck-Depressions-Inventar (BDI-2)* (2nd). Frankfurt: Pearson.
- Hernandez-Cardenache, R. & Johnson-Greene, D. (2013). Rehabilitation in stroke. In C. A. Nogle, R. S. Dean & M. T. Barisa (Eds.), *Neuropsychological rehabilitation* (pp. 161-184). New York, NY: Springer Press.
- Heruti, R. J., Lusky, A., Dankner, R., Ring, H., Dolgopiat, M., Barell, V. et al. (2002). Rehabilitation outcome of elderly patients after a first stroke: effect of cognitive status at admission on the functional outcome. *Archives of Physical Medicine and Rehabilitation*, *83*, 742-749.

- Hervey, A. S., Epstein, J. & Curry, J. F. (2004). Neuropsychology of adults with Attention-Deficit/Hyperactivity Disorder: a meta-analytic review. *Neuropsychology, 18*, 485-503.
- Heuschmann, P. U., Busse, O., Wagner, M., Endres, M., Villringer, A., Röther, J. et al. (2010). Schlaganfallhäufigkeit und Versorgung von Schlaganfallpatienten in Deutschland. *Aktuelle Neurologie, 37*, 333-340.
- Holdnack, J. A., Moberg, P. J., Arnold, S. E., Gur, R. C. & Gur, R. E. (1995). Speed of processing and verbal learning deficits in adults diagnosed with Attention Deficit Disorder. *Neuropsychiatry, Neuropsychology & Behavioral Neurology, 8*, 282-292.
- Holmes, J., Gathercole, S. E. & Dunning, D. L. (2009). Adaptive training leads to sustained enhancement of poor working memory in children. *Developmental Science, 12*, F9-F15.
- Holst, Y. & Thorell, L. B. (2013). Neuropsychological functioning in adults with ADHD and adults with other psychiatric disorders: the issue of specificity. *Journal of Attention Disorders*. Retrieved October 1, 2014, from <http://jad.sagepub.com/content/early/2013/10/17/1087054713506264.long>
- Horn, J. L. & Cattell, R. B. (1966). Refinement and test of the theory of fluid and crystallized general intelligence. *Journal of Educational Psychology, 57*, 253-270.
- Horn, J. L. & Noll, J. (1997). Human cognitive capabilities: Gf-Gc theory. In D. P. Flanagan, J. L. Genshaft & P. L. Harrison (Eds.), *Contemporary intellectual assessment: theories, tests and issues* (pp. 53-91). New York, NY: Guilford Press.
- Hostenbach, J., Mulder, T., van Limbeek, J., Donders, R. & Schoonderwaldt, H. (1998). Cognitive decline following stroke: a comprehensive study of cognitive decline following stroke. *Journal of Clinical and Experimental Neuropsychology, 20*, 503-517.
- Huang-Pollock, C. L. & Nigg, J. T. (2003). Searching for the attention deficit in Attention Deficit Hyperactivity Disorder: the case of visuospatial orienting. *Clinical Psychology Review, 23*, 801-830.
- Hugdahl, K. (2000). Lateralization of cognitive processes in the brain. *Acta Psychologica, 105*, 211-235.
- Hyndman, D. & Ashburn, A. (2003). People with stroke living in the community: attention deficits, balance, ADL ability and falls. *Disability and Rehabilitation, 25*, 817-822.
- Hyndman, D., Pickering, R. M. & Ashburn, A. (2008). The influence of attention deficits on functional recovery post stroke during the first 12 months after discharge from hospital. *Journal of Neurology, Neurosurgery, and Psychiatry, 79*, 656-663.
- International Labour Organization (2008). *Resolution concerning updating the International Standard Classification of Occupations (ISCO)*. Geneva: International Labour Organization.
- Iverson, G. L., Mendrek, A. & Adams, R. L. (2004). The persistent belief that VIQ-PIQ splits suggest lateralized brain damage. *Applied Neuropsychology, 11*, 85-90.
- Jaillard, A., Grand, S., Le Bas, J. F. & Hommel, M. (2010). Predictive cognitive dysfunction in nondemented patients early after stroke. *Cerebrovascular Diseases, 29*, 415-423.
- Jaillard, A., Naegele, B., Trabucco-Miguel, S., LeBas, J. F. & Hommel, M. (2009). Hidden dysfunctioning in subacute stroke. *Stroke, 40*, 2473-2479.
- Jiang, Y. & Johnston, C. (2012). The relationship between ADHD symptoms and competence as reported by both self and others. *Journal of Attention Disorders, 16*, 418-426.
- Johnson, D. E. & Conners, C. K. (2002). The assessment process: conditions and comorbidities. In S. Goldstein & A. T. Ellison (Eds.), *Clinician's guide to adult ADHD: assessment and intervention* (pp. 71-83). San Diego, CA: Academic Press.

- Kaesberg, S., Fink, G. R. & Kalbe, E. (2013). Neuropsychologische Frühdiagnostik nach einem Schlaganfall - ein Überblick im deutschsprachigen Raum verfügbarer Instrumente und Vorstellung eines neuen Screening - Verfahrens. *Fortschritte der Neurologie und Psychiatrie*, *81*, 482-492.
- Kane, M. J., Bleckley, M. K., Conway, A. R. A. & Engle, R. W. (2001). A controlled-attention view of working-memory capacity. *Journal of Experimental Psychology: General*, *130*, 169-183.
- Kaplan, E., Goodglass, H. & Weintraub, S. (1983). *Boston Naming Test*. Philadelphia, PA: Lea & Febiger.
- Kaufman, A. S. & Kaufman, N. L. (1994). *K-SNAP: Kaufman short neuropsychological assessment procedure*. Bloomington, MN: Pearson Assessments.
- Kerkhoff, G. (2001). Spatial hemineglect in humans. *Progress in Neurobiology*, *63*, 1-27.
- Kessler, R. C., Adler, L. A., Ames, M., Barkley, R. A., Birnbaum, H., Greenberg, P. et al. (2005). The prevalence and effects of adult Attention Deficit/Hyperactivity Disorder on work performance in a nationally representative sample of workers. *Journal of Occupational and Environmental Medicine*, *47*, 565-572.
- Kessler, R. C., Adler, L. A., Ames, M., Demler, O., Faraone, S. V., Hiripi, E. et al. (2005). The World Health Organization adult ADHD self-report scale (ASRS): a short screening scale for use in the general population. *Psychological Medicine*, *35*, 245-256.
- Kessler, R. C., Adler, L. A., Barkley, R. A., Biederman, J., Conners, K., Demler, O. et al. (2006). The prevalence and correlates of adult ADHD in the United States: results from the National Comorbidity Survey Replication. *American Journal of Psychiatry*, *163*, 716-723.
- Kessler, R. C., Green, J., Adler, L. A., Barkley, R., Chatterji, S., Faraone, S. V. et al. (2010). Structure and diagnosis of adult Attention Deficit/Hyperactivity Disorder: analysis of expanded symptom criteria from the adult ADHD clinical diagnostic scale. *Archives of General Psychiatry*, *67*, 1168-1178.
- Kessler, R. C., Lane, M., Stang, P. E., Van Brunt, D. L. & Trott, G. E. (2008). The prevalence and workplace costs of adult Attention Deficit Hyperactivity Disorder in a large manufacturing firm. *Psychological Medicine*, *21*, 1-11.
- Kiernan, R. J., Mueller, J., Langston, J. W. & Van Dyke, C. (1987). The Neurobehavioral Cognitive Status Examination: a brief but quantitative approach to cognitive assessment. *Annals of Internal Medicine*, *107*, 481-485.
- Klingberg, T., Forssberg, H. & Westerberg, H. (2002). Training of working memory in children with ADHD. *Journal of Clinical and Experimental Neuropsychology*, *24*, 781-791.
- Knouse, L. E. & Safren, S. A. (2010). Current status of cognitive behavioral therapy for adult Attention-Deficit Hyperactivity Disorder. *Psychiatric Clinics of North America*, *33*, 497-509.
- Kofler, M. J., Rapport, M. D., Sarver, D. E., Raiker, J. S., Orban, S. A., Friedman, L. M. et al. (2013). Reaction time variability in ADHD: a meta-analytic review of 319 studies. *Clinical Psychology Review*, *33*, 795-811.
- Kohen, R., Cain, K. C., Mitchell, P. H., Becker, K., Buzaitis, A., Millard, S. P. et al. (2008). Association of serotonin transporter gene polymorphisms with poststroke depression. *Archives of General Psychiatry*, *65*, 1296-1302.
- Konrad, A., Dielentheis, T. F., El Masri, D., Bayerl, M., Fehr, C., Gesierich, T. et al. (2010). Disturbed structural connectivity is related to inattention and impulsivity in adult Attention Deficit Hyperactivity Disorder. *European Journal of Neuroscience*, *31*, 912-919.
- Kooij, J. J. S. (2013). *Adult ADHD: Diagnostic assessment and treatment* (3rd). London, UK: Springer.

- Kooij, J. J. S., Bejerot, S., Blackwell, A., Caci, H., Casas-Brugue, M., Carpentier, P. J. et al. (2010). European consensus statement on diagnosis and treatment of adult ADHD: the European Network Adult ADHD. *BMC Psychiatry*, *10*, 1-24.
- Kooij, J. J. S., Boonstra, A. M., Willemsen-Swinkels, S. H. N., Bekker, E. M., Noord, I. D. & Buitelaar, J. K. (2008). Reliability, validity, and utility of instruments for self-report and informant report regarding symptoms of Attention-Deficit/Hyperactivity Disorder (ADHD) in adult patients. *Journal of Attention Disorders*, *11*, 445-458.
- Kumral, E., Topcuoglu, M. A. & Onal, M. Z. (2009). Anterior circulation syndromes. In M. Fisher (Ed.), *Handbook of Clinical Neurology Volume 94, Stroke Part III: investigation and management* (pp. 485-536). Edinburgh, UK: Elsevier.
- Kuncel, N. R., Hezlett, S. A. & Ones, D. S. (2004). Academic performance, career potential, creativity, and job performance: can one construct predict them all? *Journal of Personality and Social Psychology*, *86*, 148-161.
- Kurscheidt, J. C., Peiler, P., Behnken, A., Abel, S., Pedersen, A., Suslow, T. et al. (2008). Acute effects of methylphenidate on neuropsychological parameters in adults with ADHD: possible relevance for therapy. *Journal of Neural Transmission*, *115*, 357-362.
- Lara, C., Fayyad, J., de Graaf, R., Kessler, R. C., Aguilar-Gaxiola, S., Angermeyer, M. et al. (2009). Childhood predictors of adult Attention-Deficit/Hyperactivity Disorder: results from the World Health Organization World Mental Health Survey Initiative. *Biological Psychiatry*, *65*, 46-54.
- Laures-Gore, J., Marshall, R. S. & Verner, E. (2010). Performance of individuals with left hemisphere stroke and aphasia and individuals with right brain damage on forward and backward digit span tasks. *Aphasiology*, *25*, 43-56.
- Lee, K. H., Choi, Y. Y. & Gray, J. R. (2007). What about the neural basis of crystallized intelligence? *Behavioral and Brain Sciences*, *30*, 159-161.
- Lees, R., Fearon, P., Harrison, J. K., Broomfield, N. M. & Quinn, T. J. (2012). Cognitive and mood assessment in stroke research: focused review of contemporary studies. *Stroke*, *43*, 1678-1680.
- Leśniak, M., Bak, T., Czepiel, W., Seniów, J. & Członkowska, A. (2008). Frequency and prognostic value of cognitive disorders in stroke patients. *Dementia and Geriatric Cognitive Disorders*, *26*, 356-363.
- Levy, F., Hay, D. A., McStephen, M., Wood, C. & Waldman, I. (1997). Attention-Deficit Hyperactivity Disorder: a category or a continuum? Genetic analysis of a large-scale twin study. *Journal of the American Academy of Child and Adolescent Psychiatry*, *36*, 737-744.
- Lewin, J. S., Friedman, L., Wu, D., Miller, D. A., Thompson, L. A., Klein, S. K. et al. (1996). Cortical localization of human sustained attention: detection with functional MR using a visual vigilance paradigm. *Journal of Computer Assisted Tomography*, *20*, 695-701.
- Lezak, M. D. (1988). Neuropsychological tests and assessment techniques. In F. Boller & J. Grafman (Eds.), *Handbook of neuropsychology* (pp. 47-68). Amsterdam: Elsevier.
- Lezak, M. D., Howieson, D. B. & Loring, D. W. (2004). *Neuropsychological assessment* (4th). New York, NY: Oxford University Press.
- Lichtenberger, E. O. & Kaufman, A. S. (2009a). Clinical Application II: Age and intelligence across the adult life span. In E. O. Lichtenberger & A. S. Kaufman (Eds.), *Essentials of WAIS-IV assessment* (2nd ed., pp. 245-298). Hoboken, NJ: Wiley.
- Lichtenberger, E. O. & Kaufman, A. S. (2009b). *Essentials of WAIS-IV assessment* (2nd). Hoboken, NJ: Wiley.

- Lichtenberger, E. O. & Kaufman, A. S. (2009c). How to interpret the WAIS-IV: conceptual and clinical foundations. In E. O. Lichtenberger & A. S. Kaufman (Eds.), *Essentials of WAIS-IV assessment* (2nd ed., pp. 119-215). Hoboken, NJ: Wiley.
- Lincoln, N. B., Nicholl, C. R., Flannaghan, T., Leonard, M. & Van der Gucht, E. (2003). The validity of questionnaire measures for assessing depression after stroke. *Clinical Rehabilitation*, *17*, 840-846.
- Lindén, T., Samuelsson, H., Skoog, I. & Blomstrand, C. (2005). Visual neglect and cognitive impairment in elderly patients late after stroke. *Acta Neurologica Scandinavica*, *111*, 163-168.
- Loring, D. W. & Bauer, R. M. (2010). Testing the limits: cautions and concerns regarding the new Wechsler IQ and Memory scales. *Neurology*, *74*, 685-690.
- Lovejoy, D. W., Ball, J. D., Keats, M., Stutts, M. L., Spain, E. H., Janda, L. et al. (1999). Neuropsychological performance of adults with Attention Deficit Hyperactivity Disorder (ADHD): diagnostic classification estimates for measures of frontal lobe/executive functioning. *Journal of the International Neuropsychological Society*, *5*, 222-233.
- Lubow, R. E., Kaplan, O. & Manor, I. (2014). Latent inhibition in ADHD adults on and off medication: a preliminary study. *Journal of Attention Disorders*, *18*, 625-631.
- Makris, N., Biederman, J., Valera, E. M., Bush, G., Kaiser, J., Kennedy, D. N. et al. (2007). Cortical thinning of the attention and executive function networks in adults with Attention-Deficit/Hyperactivity Disorder. *Cerebral Cortex*, *17*, 1364-1375.
- Mannuzza, S., Klein, R. G. & Moulton, J. L. (2008). Lifetime criminality among boys with Attention Deficit Hyperactivity Disorder: a prospective follow-up study into adulthood using official arrest records. *Psychiatry Research*, *160*, 237-246.
- Marchetta, N. D. J., Hurks, P. P. M., De Sonneville, L. M. J., Krabbendam, L. & Jolles, J. (2008). Sustained and focused attention deficits in adult ADHD. *Journal of Attention Disorders*, *11*, 664-676.
- Marchetta, N. D. J., Hurks, P. P. M., Krabbendam, L. & Jolles, J. (2008). Interference control, working memory, concept shifting, and verbal fluency in adults with Attention-Deficit/Hyperactivity Disorder (ADHD). *Neuropsychology*, *22*, 74-84.
- McCann, B. S. & Roy-Byrne, P. (2004). Screening and diagnostic utility of self-report Attention Deficit Hyperactivity Disorder scales in adults. *Comprehensive Psychiatry*, *45*, 175-183.
- McCloskey, G., Hartz, E. S. & Scipioni, K. (2009). A neuropsychological approach to interpretation of the WAIS-IV and the use of the WAIS-IV in learning disability assessment. In E. O. Lichtenberger & A. S. Kaufman (Eds.), *Essentials of WAIS-IV assessment* (2nd ed., pp. 216-253). Hoboken, NJ: Wiley.
- McDonnell, M. N., Bryan, J., Smith, A. E. & Esterman, A. J. (2011). Assessing cognitive impairment following stroke. *Journal of Clinical and Experimental Neuropsychology*, *33*, 945-953.
- McDowd, J. M., Filion, D. L., Pohl, P. S., Richards, L. G. & Stiers, W. (2003). Attentional abilities and functional outcomes following stroke. *The Journals of Gerontology: Psychological Sciences*, *58B*, 45-53.
- McGough, J. J., Smalley, S. L., McCracken, J. T., May Yang, M. S., Del'Homme, M., Lynn, D. E. et al. (2005). Psychiatric comorbidity in adult Attention-Deficit/Hyperactivity Disorders: findings from multiplex families. *American Journal of Psychiatry*, *162*, 1621-1627.
- McGrew, K. S. & Flanagan, D. P. (1998). *The intelligence test desk reference (ITDR): Gf-Gc cross battery assessment*. Boston, MA: Allyn & Bacon.

- McLean, A., Dowson, J., Toone, B., Young, S., Bazanis, E., Robbins, T. et al. (2004). Characteristic neurocognitive profile associated with Attention-Deficit/Hyperactivity Disorder. *Psychological Medicine, 34*, 681-692.
- Mesulam, M.-M. (1985). Attention, confusional states and neglect. In M.-M. Mesulam (Ed.), *Principles of behavioral neurology* (pp. 125-168). Philadelphia, PA: FA Davis.
- Mesulam, M.-M. (2000). Attentional networks, confusional states, and neglect syndromes. In M.-M. Mesulam (Ed.), *Principles of behavioral and cognitive neurology* (2nd ed., pp. 174-256). New York, NY: Oxford University Press.
- Meyer, G. J., Finn, S. E., Eyde, L. D., Kay, G. G., Moreland, K. L., Dies, R. R. et al. (2001). Psychological testing and psychological assessment: a review of evidence and issues. *American Psychologist, 56*, 128-165.
- Meyers, J. E., Zellinger, M. M., Kockler, T., Wagner, M. & Miller, R. M. (2013). A validated seven-subtest short form for the WAIS-IV. *Applied Neuropsychology: Adult, 20*, 249-256.
- Mick, E., Biederman, J., Prince, J., Fischer, M. J. & Faraone, S. V. (2002). Impact of low birth weight on Attention-Deficit/Hyperactivity Disorder. *Journal of Developmental & Behavioral Pediatrics, 23*, 16-22.
- Miller, E. L., Murray, L., Richards, L. G., Zorowitz, R. D., Bakas, T., Clark, P. et al. (2010). Comprehensive overview of nursing and interdisciplinary rehabilitation care of the stroke patient: a scientific statement from the American Heart Association. *Stroke, 41*, 2402-2448.
- Mirsky, A. F., Anthony, B. J., Duncan, C. C., Ahearn, M. & Kellam, S. G. (1991). Analysis of the elements of attention: a neuropsychological approach. *Neuropsychology Review, 2*, 109-145.
- Mongia, M. & Hechtman, L. (2012). Cognitive Behavior Therapy for adults with Attention-Deficit/Hyperactivity Disorder: a review of recent randomized controlled trials. *Current Psychiatry Reports, 14*, 561-567.
- Montour-Proulx, I., Braun, C. M. J., Daigneault, S., Rouleau, I., Kuehn, S. & Bégin, J. (2004). Predictors of intellectual function after a unilateral cortical lesion: study of 635 patients from infancy to adulthood. *Journal of Child Neurology, 19*, 935-943.
- Mrakotsky, C. (2007). Konzepte der Entwicklungsneuropsychologie. In L. Kaufmann, H.-C. Nuerk, K. Konrad & K. Willmes (Eds.), *Kognitive Entwicklungsneuropsychologie* (pp. 25-44). Göttingen: Hogrefe.
- Müller, B. W., Gimbel, K., Keller-Pließnig, A., Sartory, G., Gastpar, M. & Davids, E. (2007). Neuropsychological assessment of adult patients with Attention-Deficit/Hyperactivity Disorder. *European Archives of Psychiatry and Clinical Neuroscience, 257*, 112-119.
- Müri, R. M., Bühler, R., Heinemann, D., Mosimann, U. P., Felblinger, J., Schlaepfer, T. E. et al. (2002). Hemispheric asymmetry in visuospatial attention assessed with transcranial magnetic stimulation. *Experimental Brain Research, 143*, 426-430.
- Murphy, K. R., Barkley, R. A. & Bush, T. (2001). Executive functioning and olfactory identification in young adults with Attention Deficit-Hyperactivity Disorder. *Neuropsychology, 15*, 211-220.
- Nasreddine, Z. S., Phillips, N. A., Bédirian, V., Charbonneau, S., Whitehead, V., Collin, I. et al. (2005). The Montreal Cognitive Assessment, MoCA: a brief screening tool for mild cognitive impairment. *Journal of the American Geriatrics Society, 53*, 695-699.
- National Institute of Neurological Disorders and Stroke (n.d.). *National Institute of Health Stroke Scale (NIHSS)*. Retrieved October 1, 2014, from http://www.ninds.nih.gov/doctors/NIH_Stroke_Scale_Booklet.pdf

- Nelson, H. E. & Willison, J. R. (1991). *National Adult Reading Test (NART)*. Windsor, U.K.: NFER-Nelson.
- Ng, V. W. K., Eslinger, P. J., Williams, S. C. R., Brammer, M. J., Bullmore, E. T., Andrew, C. M. et al. (2000). Hemispheric preference in visuospatial processing: a complementary approach with fMRI and lesion studies. *Human Brain Mapping, 10*, 80-86.
- Ng, Y. S., Stein, J., Ning, M. & Black-Schaffer, R. M. (2007). Comparison of clinical characteristics and functional outcomes of ischemic stroke in different vascular territories. *Stroke, 38*, 2309-2314.
- Nigg, J. T. (2001). Is ADHD a disinhibitory disorder? *Psychological Bulletin, 127*, 571-598.
- Nigg, J. T., Butler, K. M., Huang-Pollock, C. L. & Henderson, J. M. (2002). Inhibitory processes in adults with persistent childhood onset ADHD. *Journal of Consulting and Clinical Psychology, 70*, 153-157.
- Nigg, J. T., Nikolas, M. & Burt, S. A. (2010). Measured gene-by-environment interaction in relation to Attention-Deficit/Hyperactivity Disorder. *Journal of the American Academy of Child & Adolescent Psychiatry, 49*, 863-873.
- Nigg, J. T., Stavro, G., Ettenhofer, M., Hambrick, D. Z., Miller, T. & Henderson, J. M. (2005). Executive functions and ADHD in adults: evidence for selective effects on ADHD symptom domains. *Journal of Abnormal Psychology, 114*, 706-717.
- Nigg, J. T., Willcutt, E. G., Doyle, A. E. & Sonuga-Barke, E. J. S. (2005). Causal heterogeneity in Attention-Deficit/Hyperactivity Disorder: do we need neuropsychologically impaired subtypes? *Biological Psychiatry, 57*, 1224-1223.
- Nijboer, T. C. W., Kollen, B. J. & Kwakkel, G. (2013). Time course of visuospatial neglect early after stroke: a longitudinal cohort study. *Cortex, 30*, 1-7.
- Norman, D. & Shallice, T. (1986). Attention to action: willed and automatic control of behavior. In R. Davidson, R. G. Schwartz & D. Shapiro (Eds.), *Consciousness and self-regulation: advances in research and theory* (pp. 1-18). New York, NY: Plenum Press.
- Nys, G. M. S., van Zandvoort, M. J. E., de Kort, P. L., Jansen, B. P. W., de Haan, E. H. & Kappelle, L. J. (2007). Cognitive disorders in acute stroke: prevalence and clinical determinants. *Cerebrovascular Diseases, 23*, 408-416.
- Nys, G. M. S., van Zandvoort, M. J. E., de Kort, P. L., van der Worp, H. B., Jansen, B. P., Algra, A. et al. (2005). The prognostic value of domain-specific cognitive abilities in acute first-ever stroke. *Neurology, 64*, 821-827.
- Nys, G. M. S., van Zandvoort, M. J. E., de Kort, P. L. M., Jansen, B. P. W., van der Worp, H. B., Kappelle, L. J. et al. (2005). Domain specific cognitive recovery after first-ever stroke: a follow up study of 111 cases. *Journal of the International Neuropsychological Society, 11*, 795-806.
- Ohlmeier, M. D., Peters, K., Te Wildt, B. T., Zedler, M., Ziegenbein, M., Wiese, B. et al. (2008). Comorbidity of alcohol and substance dependence with Attention-Deficit/Hyperactivity Disorder (ADHD). *Alcohol and alcoholism (Oxford, Oxfordshire), 43*, 300-304.
- Olesen, P. J., Westerberg, H. & Klingberg, T. (2004). Increased prefrontal and parietal activity after training of working memory. *Nature Neuroscience, 7*, 75-79.
- Orfei, M. D., Caltagirone, C. & Spalletta, G. (2009). The evaluation of anosognosia in stroke patients. *Cerebrovascular Diseases, 27*, 280-289.
- Paradiso, S., Anderson, B. M., Boles Ponto, L. L., Tranel, D. & Robinson, R. G. (2011). Altered neural activity and emotions following right Middle Cerebral Artery Stroke. *Journal of Stroke and Cerebrovascular Diseases, 20*, 94-104.
- Parasuraman, R. (1998). *The attentive brain*. Cambridge, MA: MIT Press.

- Pardo, J. V., Fox, P. T. & Raichle, M. E. (1991). Localization of a human system for sustained attention by positron emission tomography. *Nature*, *349*, 61-64.
- Parker, H. C., Ellison, A. T., Sherman, G., Frisch, M. E., Kay, C., Burton, S. et al. (2012). Improving the diagnosis, treatment, and follow-up of adult Attention Deficit/Hyperactivity Disorder (ADHD) patients in primary care utilizing a performance improvement continuing medical education (PI CME) activity. *CE Measure*, *6*, 3-12.
- Patel, M. D., Coshall, C., Rudd, A. G. & Wolfe, C. D. A. (2003). Natural history of cognitive impairment after stroke and factors associated with its recovery. *Clinical Rehabilitation*, *17*, 158-166.
- Pazvantoğlu, O., Aker, A. A., Karabekiroğlu, K., Akbaş, S., Sarısoy, G., Baykal, S. et al. (2012). Neuropsychological weaknesses in adult ADHD; cognitive functions as core deficit and roles of them in persistence to adulthood. *Journal of the International Neuropsychological Society*, *18*, 819-826.
- Pendlebury, S. T., Cuthbertson, F. C., Welch, S. J. V., Mehta, Z. & Rothwell, P. M. (2010). Underestimation of cognitive impairment by Mini-Mental State Examination versus the Montreal Cognitive Assessment in patients with transient ischemic attack and stroke: a population-based study. *Stroke*, *41*, 1290-1293.
- Pesok, A. (2013). Clinical dilemmas in the assessment and management of ADHD in adult: a psychiatrist's view from an urban hospital clinic. In C. B. H. Surman (Ed.), *Clinical assessment of ADHD in adults ADHD in adults: a practical guide to evaluation and management* (pp. 191-196). New York, NY: Springer.
- Pessoa, L. & Ungerleider, L. G. (2004). Top-down mechanisms for working memory and attentional processes. In M. S. Gazzaniga (Ed.), *The cognitive neurosciences* (3rd ed., pp. 919-930). Cambridge, UK: MIT Press.
- Petermann, F. (2012). *Wechsler Adult Intelligence Scale - Fourth Edition (WAIS-IV). German Version*. Frankfurt: Pearson Assessment.
- Philipose, L. E., Alphs, H., Prabhakaran, V. & Hillis, A. E. (2007). Testing conclusions from functional imaging of working memory with data from acute stroke. *Behavioral Neurology*, *18*, 37-43.
- Planton, M., Peiffer, S., Albucher, J. F., Barbeau, E. J., Tardy, J., Pastor, J. et al. (2012). Neuropsychological outcome after a first symptomatic ischaemic stroke with 'good recovery'. *European Journal of Neurology*, *19*, 212-219.
- Pohjasvaara, T., Leskelä, M., Vataja, R., Kalska, H., Ylikoski, R., Hietanen, M. et al. (2002). Post-stroke depression, executive dysfunction and functional outcome. *European Journal of Neurology*, *9*, 269-275.
- Posner, M. I. & Boies, S. J. (1971). Components of attention. *Psychological Review*, *78*, 391-408.
- Posner, M. I. & Petersen, S. E. (1990). The attention system of the human brain. *Annual Review of Neuroscience*, *13*, 25-42.
- Posner, M. I. & Rothbart, M. K. (2007). Research on attention networks as a model for the integration of psychological science. *Annual Review of Psychology*, *58*, 1-23.
- Pulsipher, D. T., Stricker, N. H., Sadek, J. R. & Haaland, K. Y. (2013). Clinical utility of the Neuropsychological Assessment Battery (NAB) after unilateral stroke. *The Clinical Neuropsychologist*, *27*, 1-22.
- Purdy, M. H. (2011). Executive functions: theory, assessment and treatment. In M. Kimbarow (Ed.), *Cognitive communication disorders* (pp. 77-90). San Diego, CA: Plural Publishing.
- Quinlan, D. M. & Brown, T. E. (2003). Assessment of short-term verbal memory impairments in adolescents and adults with ADHD. *Journal of Attention Disorders*, *6*, 143-152.

- Ramsay, J. R. (2010). *Nonmedication treatments for adult ADHD: evaluating impact on daily functioning and well-being*. Washington, DC: American Psychological Association.
- Rappoport, L. J., Friedman, S. L., Tzelepis, A. & Van Voorhis, A. (2002). Experienced emotion and affect recognition in adult Attention-Deficit Hyperactivity Disorder. *Neuropsychology, 16*, 102-110.
- Rappoport, L. J., Van Voorhis, A., Tzelepis, A. & Friedman, S. R. (2001). Executive functioning in adult Attention-Deficit Hyperactivity Disorder. *The Clinical Neuropsychologist, 15*, 479-491.
- Rashid, F. L., Morris, M. K. & Morris, R. G. (2001). Naming and verbal memory skills in adults with Attention Deficit Hyperactivity Disorder and Reading Disability. *Journal of Clinical Psychology, 57*, 829-838.
- Rasquin, S. M. C., Lodder, J., Ponds, R. W., Winkens, I., Jolles, J. & Verhey, F. R. (2004). Cognitive functioning after stroke: a one-year follow-up study. *Dementia and Geriatric Cognitive Disorders, 18*, 138-144.
- Reeves, M. J., Bushnell, C. D., Howard, G., Gargano, J. W., Duncan, P. W., Lynch, G. et al. (2008). Sex differences in stroke: epidemiology, clinical presentation, medical care, and outcomes. *The Lancet Neurology, 7*, 915-926.
- Reitan, R. M. (1992). *Trail Making Test: Manual for Administration and Scoring*. Tucson, AZ: Reitan Neuropsychology Laboratory.
- Retz-Junginger, P., Retz, W., Blocher, D., Weijers, H. G., Trott, G. E., Wender, P. H. et al. (2002). Wender Utah Rating Scale (WURS-k). Die deutsche Kurzform zur retrospektiven Erfassung des hyperkinetischen Syndroms bei Erwachsenen. *Nervenarzt, 73*, 830-838.
- Riccio, C. A., Wolfe, M. E., Romine, C., Davis, B. & Sullivan, J. R. (2004). The Tower of London and neuropsychological assessment of ADHD in adults. *Archives of Clinical Neuropsychology, 19*, 661-671.
- Ringman, J. M., Saver, J. L., Woolson, R. F., Clarke, W. R. & Adams, H. P. (2004). Frequency, risk factors, anatomy, and course of unilateral neglect in an acute stroke cohort. *Neurology, 63*, 468-474.
- Robertson, I., Ridgeway, V., Greenfield, E. & Parr, A. (1997). Motor recovery after stroke depends on intact sustained attention: a 2-year follow-up study. *Neuropsychology, 11*, 290-295.
- Robin, A. L. & Payson, E. (2002). The impact of ADHD on marriage. *The ADHD Report, 10*, 9-14.
- Robins, L. N., Helzer, J. E., Croughan, J. & Ratcliff, K. S. (1981). National institute of mental health diagnostic interview schedule. Its history, characteristic, and validity. *Archives of General Psychiatry, 38*, 381-389.
- Robinson, R. G. & Spalletta, G. (2010). Poststroke depression: a review. *The Canadian Journal of Psychiatry, 55*, 341-349.
- Rohlf, H., Jucksch, V., Gawrilow, C., Huss, M., Hein, J., Lehmkuhl, U. et al. (2012). Set shifting and working memory in adults with Attention-Deficit/Hyperactivity Disorder. *Journal of Neural Transmission, 119*, 95-106.
- Rösler, M., Retz-Junginger, P., Retz, W. & Stieglitz, R. D. (2008). *Homburger ADHS-Skalen für Erwachsene (HASE)*. Göttingen: Hogrefe.
- Rösler, M., Retz, W., Retz-Junginger, P., Stieglitz, R. D., Kessler, H., Reimherr, F. et al. (2008). ADHS-Diagnose bei Erwachsenen: Nach DSM-IV, ICD-10 und den UTAH-Kriterien. *Nervenarzt, 79*, 320-327.
- Rosvold, H. E., Mirsky, A. F., Sarason, I., Bronsome, E. D. & Beck, L. H. (1956). A continuous performance test of brain damage. *Journal of Consulting Psychology and Aging, 20*, 343-350.

- Russell, V. A. (2002). Hypodopaminergic and hypernoradrenergic activity in prefrontal cortex slices of an animal model for Attention-Deficit Hyperactivity Disorder - the spontaneously hypertensive rat. *Behavioural Brain Research, 130*, 191-196.
- Rutledge, K. J., van den Bos, W., McClure, S. M. & Schweitzer, J. B. (2012). Training cognition in ADHD: current findings, borrowed concepts, and future directions. *Neurotherapeutics, 9*, 542-558.
- Ryan, J. J., Bartels, J. M., Morris, J., Cluff, R. B. & Gontkovsky, S. T. (2009). WAIS-III VIQ-PIQ and VCI-POI discrepancies in lateralized cerebral damage. *International Journal of Neuroscience, 119*, 1198-1209.
- Schmidt-Atzert, L., Krumm, S. & Bühner, M. (2008). Aufmerksamkeitsdiagnostik. Ableitung eines Strukturmodells und systematische Einordnung von Tests. *Zeitschrift für Neuropsychologie, 19*, 59-82.
- Schmidt, S. & Petermann, F. (2009a). *ADHS-Screening für Erwachsene (ADHS-E)*. Frankfurt: Pearson Assessment.
- Schmidt, S. & Petermann, F. (2009b). Developmental psychopathology: Attention Deficit Hyperactivity Disorder. *BMC Psychiatry, 9*, 1-10.
- Schneider, W. J. & McGrew, K. S. (2012). The Cattell-Horn-Carroll model of intelligence. In D. P. Flanagan & P. L. Harrison (Eds.), *Contemporary intellectual assessment: theories, tests, and issues* (3rd ed., pp. 99-144). New York, NY: Guilford.
- Schneider, W. J. & Shiffrin, R. M. (1977). Controlled and automatic human information processing: 1: detecting, search, and attention. *Psychological Review, 84*, 1-66.
- Schoechlin, C. & Engel, R. R. (2005). Neuropsychological performance in adult Attention-Deficit Hyperactivity Disorder: meta-analysis of empirical data. *Archives of Clinical Neuropsychology, 20*, 727-744.
- Schouten, E. A., Schiemanck, S. K., Brand, N. & Post, M. W. M. (2009). Long-term deficits in episodic memory after ischemic stroke: evaluation and prediction of verbal and visual memory performance based on lesion characteristics. *Journal of Stroke and Cerebrovascular Diseases, 18*, 128-138.
- Schreiber, H. E., Javorsky, D. J., Robinson, J. E. & Stern, R. A. (1999). Rey-Osterrieth Complex Figure Performance in adults with Attention Deficit Hyperactivity Disorder: a validation study of the Boston Qualitative Scoring System. *The Clinical Neuropsychologist, 13*, 509-520.
- Schubert, F. & Lalouschek, W. (2006). Schlaganfall. In J. Lehrner, G. Pusswald, E. Fertl, I. Kryspin-Exner & W. Strubreither (Eds.), *Klinische Neuropsychologie* (pp. 303-314). Wien: Springer.
- Schwartz, G. E., Davidson, R. J. & Maer, F. (1975). Right hemisphere lateralization for emotion in the human brain: interactions with cognition. *Science, 190*, 286-288.
- Schwartz, J. A., Speed, N. M., Brunberg, J. A., Brewer, T. L., Brown, M. & Greden, J. F. (1993). Depression in stroke rehabilitation. *Biological Psychiatry, 33*, 694-699.
- Schweizer, K. (2005). An overview of research into the cognitive basis of intelligence. *Journal of Differential Psychology, 26*, 43-51.
- Schweizer, K. (2010). The relationship between attention and intelligence. In A. Gruszka, G. Matthews & B. Szymura (Eds.), *Handbook of individual differences in cognition. Attention, memory, and executive control* (pp. 247-262). New York, NY: Springer.
- Schweizer, K. & Moosbrugger, H. (2004). Attention and working memory as predictors of intelligence. *Intelligence, 32*, 329-347.
- Schweizer, K., Moosbrugger, H. & Goldhammer, F. (2005). The structure of the relationship between attention and intelligence. *Intelligence, 33*, 589-611.

- Schweizer, K., Zimmermann, P. & Koch, W. (2000). Sustained attention, intelligence, and the crucial role of perceptual processes. *Learning and Individual Differences*, *12*, 271-286.
- Seidman, L. J. (2006). Neuropsychological functioning in people with ADHD across the lifespan. *Clinical Psychology Review*, *26*, 466-485.
- Seidman, L. J., Biederman, J., Weber, W., Hatch, M. & Faraone, S. V. (1998). Neuropsychological function in adults with Attention-Deficit Hyperactivity Disorder. *Biological Psychiatry*, *44*, 260-268.
- Seidman, L. J., Doyle, A., Fried, R., Valera, E., Crum, K. & Matthews, L. (2004). Neuropsychological function in adults with Attention-Deficit/Hyperactivity Disorder. *Psychiatric Clinics of North America*, *27*, 261-282.
- Seidman, L. J., Valera, E. M., Makris, N., Monuteaux, M. C., Boriel, D., Kelkar, K. et al. (2006). Dorsolateral prefrontal and anterior cingulate cortex volumetric abnormalities in adults with Attention-Deficit/Hyperactivity Disorder identified by magnetic resonance imaging. *Biological Psychiatry*, *60*, 1071-1080.
- Seixas, M., Weiss, M. & Müller, U. (2012). Systematic review of national and international guidelines on Attention-Deficit Hyperactivity Disorder. *Journal of Psychopharmacology*, *26*, 753-765.
- Siebert, R. J., Walkey, F. H. & Turner-Stokes, L. (2009). An examination of the factor structure of the Beck Depression Inventory-II in a neurorehabilitation inpatient sample. *Journal of the International Neuropsychological Society*, *15*, 142-147.
- Silva, K. L., Guimarães-da-Silva, P. O., Grevet, E. H., Victor, M. M., Salgado, C. A. I., Vitola, E. S. et al. (2013). Cognitive deficits in adults with ADHD go beyond comorbidity effects. *Journal of Attention Disorders*, *17*, 483-488.
- Simon, V., Czobor, P., Bálint, S., Mészáros, Á. & Bitter, I. (2009). Prevalence and correlates of adult Attention-Deficit Hyperactivity Disorder: meta-analysis. *The British Journal of Psychiatry*, *194*, 204-211.
- Skidmore, E. R., Whyte, E. M., Holm, M. B., Becker, J. T., Butters, M. A., Dew, M. A. et al. (2010). Cognitive and affective predictors of rehabilitation participation after stroke. *Archives of Physical Medicine and Rehabilitation*, *91*, 203-207.
- Skodzik, T., Holling, H. & Pedersen, A. (2013). Long-term memory performance in adult ADHD: a meta-analysis. *Journal of Attention Disorders*. Retrieved October 1, 2014, from <http://jad.sagepub.com/content/early/2013/11/14/1087054713510561.full.pdf>
- Snaphaan, L. & de Leeuw, F.-E. (2007). Poststroke memory function in nondemented patients: a systematic review on frequency and neuroimaging correlates. *Stroke*, *38*, 198-203.
- Spalletta, G., Bossu, P., Ciaramella, A., Bria, P., Caltagirone, C. & Robinson, R. G. (2006). The etiology of poststroke depression: a review of the literature and a new hypothesis involving inflammatory cytokines. *Molecular Psychiatry*, *11*, 984-991.
- Spearman, C. (1904). General intelligence, objectively determined and measured. *American Journal of Psychology*, *15*, 201-293.
- Spikman, J. & van Zomeren, E. (2010). Assessment of attention. In J. Gurd, U. Kischka & J. Marshall (Eds.), *The handbook of clinical neuropsychology* (2nd ed., pp. 81-96). Oxford, UK: Oxford University Press.
- Sporns, O., Chialvo, D. R., Kaiser, M. & Hilgetag, C. C. (2004). Organization, development and function of complex brain networks. *Trends in Cognitive Sciences*, *8*, 418-425.
- SPSS Inc (2011). *SPSS Base 20.0 for Windows User's Guide*. Chicago, IL: SPSS Inc.
- Stankov, L. (1983). Attention and intelligence. *Journal of Educational Psychology*, *75*, 471-490.
- Stapleton, T., Ashburn, A. & Stack, E. (2001). A pilot study of attention deficits, balance control and falls in the subacute stage following stroke. *Clinical Rehabilitation*, *15*, 437-444.

- StataCorp (2013). *Stata Statistical Software: Release 13*. College Station, TX: StataCorp LP.
- Sternberg, R. J. (2009). *Cognitive Psychology* (5th). Belmont, CA: Wadsworth.
- Stieglitz, R. D., Nyberg, E. & Hofecker-Fallahpour, M. (2012). *ADHS im Erwachsenenalter - Fortschritte der Psychotherapie*. Göttingen: Hogrefe.
- Stricker, N. H., Tybur, J. M., Sadek, J. R. & Haaland, K. Y. (2010). Utility of the Neuropsychological Assessment Battery in detecting cognitive impairment after unilateral stroke. *Journal of the International Neuropsychological Society*, *16*, 813-821.
- Surman, C. B. H. (2013). Clinical assessment of ADHD in adults. In C. B. H. Surman (Ed.), *ADHD in adults: a practical guide to evaluation and management* (pp. 19-44). New York, NY: Springer.
- Tatemichi, T. K., Desmond, D. W., Stern, Y., Paik, M., Sano, M. & Bagiella, E. (1994). Cognitive impairment after stroke: frequency, patterns, and relationship to functional abilities *Journal of Neurology, Neurosurgery & Psychiatry*, *20*, 503-517.
- Tatemichi, T. K., Paik, M., Bagiella, E., Desmond, D. W., Pirro, M. & Hanzawa, L. K. (1994). Dementia after stroke is a predictor of long-term survival. *Stroke*, *25*, 1915-1919.
- Taylor, E., Döpfner, M., Sergeant, J., Asherson, P., Banaschewski, T., Buitelaar, J. et al. (2004). European clinical guidelines for hyperkinetic disorder - first upgrade. *European Child and Adolescent Psychiatry*, *13*, 1/7-1/30.
- 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*, 291-301.
- 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*, 3-16.
- Thilmann, A., Nachtmann, A. & Scharff, A. (2006). Neurologischer Reha-Score – Eine Skala zur Messung des Patientenzustandes und Aufwandes in der neurologischen Rehabilitation. *Nervenarzt*, *77*, 1456-1463.
- Thompson, H. S. & Ryan, A. (2009). The impact of stroke consequences on spousal relationships from the perspective of the person with stroke. *Journal of Clinical Nursing*, *18*, 1803-1811.
- Tompkins, C. A., Bloise, C. G., Timko, M. L. & Baumgaertner, A. (1994). Working memory and inference revision in brain-damaged and normally aging adults. *Journal of Speech and Hearing Research*, *37*, 896-912.
- Tompkins, C. A., Scharp, V. L., Meigh, K. M. & Fassbinder, W. (2008). Coarse coding and discourse comprehension in adults with right hemisphere brain damage. *Aphasiology*, *22*, 204-223.
- Torgersen, T., Gjervan, B. & Rasmussen, K. (2006). ADHD in adults: a study of clinical characteristics, impairment and comorbidity. *Nordic Journal of Psychiatry*, *60*, 38-43.
- Treisman, A. M. (1969). Strategies and models of selective attention. *Psychological Review*, *76*, 282-299.
- Tucha, O., Mecklinger, L., Laufkötter, R., Kaunzinger, I., Paul, G., Klein, H. et al. (2005). Clustering and switching on verbal and figural fluency functions in adults with Attention Deficit Hyperactivity Disorder. *Cognitive Neuropsychiatry*, *10*, 231-248.
- Turner, D. C., Blackwell, A. D., Dowson, J. H., McLean, A. & Sahakian, B. J. (2005). Neurocognitive effects of methylphenidate in adult Attention-Deficit/Hyperactivity Disorder. *Psychopharmacology*, *178*, 286-295.
- UK Adult ADHD Network (2013). *Handbook for Attention Deficit Hyperactivity Disorder in adults*. London, UK: Springer Healthcare.

- Valera, E. M., Faraone, S. V., Murray, K. E. & Seidman, L. J. (2007). Meta-analysis of structural imaging findings in Attention-Deficit/Hyperactivity Disorder. *Biological Psychiatry*, *61*, 1361-1369.
- Valko, L., Doehnert, M., Müller, U. C., Schneider, G., Albrecht, B., Drechsler, R. et al. (2009). Differences in neurophysiological markers of inhibitory and temporal processing deficits in children and adults with ADHD. *Journal of Psychophysiology*, *23*, 235-246.
- Valko, L., Schneider, G., Doehnert, M., Müller, U., Brandeis, D., Steinhausen, H.-C. et al. (2010). Time processing in children and adults with ADHD. *Journal of Neural Transmission*, *117*, 1213-1228.
- van den Heuvel, M. P. & Hulshoff Pol, H. E. (2010). Exploring the brain network: a review on resting-state fMRI functional connectivity. *European Neuropsychopharmacology*, *20*, 519-534.
- Van Zomeren, A. H. & Brouwer, W. H. (1994). *Clinical neuropsychology of attention*. New York, NY: Oxford University Press.
- Vataja, R., Pohjasvaara, T., Mäntylä, R., Ylikoski, R., Leppävuori, A., Leskelä, M. et al. (2003). MRI correlates of executive dysfunction in patients with ischaemic stroke. *European Journal of Neurology*, *10*, 625-631.
- Waite, R. & Ramsay, J. R. (2010). Adults with ADHD: who are we missing? *Issues in Mental Health Nursing*, *31*, 670-678.
- Waite, R., Vlam, R. C., Irrera-Newcomb, M. & Babcock, T. (2013). The diagnosis less traveled: NPs' role in recognizing adult ADHD. *Journal of the American Association of Nurse Practitioners*, *25*, 302-308.
- Walker, A. Y., Shores, A. E., Trollor, J. N., Lee, T. & Sachdev, P. S. (2000). Neuropsychological functioning of adults with Attention Deficit Hyperactivity Disorder. *Journal of Clinical and Experimental Neuropsychology*, *22*, 115-124.
- Ward, M. F., Wender, P. H. & Reimherr, F. W. (1993). The Wender Utah Rating Scale: An aid in the retrospective diagnosis of childhood Attention Deficit Hyperactivity Disorder. *American Journal of Psychiatry*, *150*, 885-890.
- Warrington, E. K., James, M. & Maciejewski, C. (1986). The WAIS as a lateralizing and localizing diagnostic instrument: a study of 656 patients with unilateral cerebral lesions. *Neuropsychologia*, *24*, 223-239.
- Wechsler, D. (1939). *The measurement of adult intelligence*. Baltimore, MD: Williams & Wilkins.
- Wechsler, D. (2008). *Wechsler Adult Intelligence Scale-IV*. San Antonio, TX: The Psychological Corporation.
- Wechsler, D. (2009a). *Wechsler Memory Scale - Fourth Edition. Manual* (4th). San Antonio, TX: Pearson Assessment.
- Wechsler, D. (2009b). *Wechsler Memory Scale: Manual* (4th). San Antonio, TX: Pearson Assessment.
- Wechsler, D., Coalson, D. L. & Raiford, S. E. (2008). *WAIS-IV technical and interpretive manual*. San Antonio, TX: Pearson.
- Weiss, L. G., Keith, T. Z., Zhu, J. & Chen, H. (2013). WAIS-IV and clinical validation of the four- and five-factor interpretative approaches. *Journal of Psychoeducational Assessment*, *31*, 94-113.
- Weiss, L. G., Saklofske, D. H., Coalson, D. & Raiford, S. E. (2010). Theoretical, empirical and clinical foundations of the WAIS-IV index scores. In L. G. Weiss, D. H. Saklofske, D. Coalson & S. E. Raiford (Eds.), *WAIS-IV clinical use and interpretation: scientist-practitioner perspectives* (pp. 61-94). San Diego, CA: Academic Press.

- Weiss, M. & Murray, C. D. (2003). Assessment and management of Attention-Deficit Hyperactivity Disorder in adults. *Canadian Medical Association Journal*, *168*, 715-722.
- Wiersema, R., van der Meere, J., Antrop, I. & Roeyers, H. (2006). State regulation in adult ADHD: an event-related potential study. *Journal of Clinical and Experimental Neuropsychology*, *28*, 1113-1126.
- Wilde, M. C. (2010). Lesion location and repeatable battery for the assessment of neuropsychological status performance in acute ischemic stroke. *The Clinical Neuropsychologist*, *24*, 57-69.
- 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*, 1336-1346.
- Willcutt, E. G., Pennington, B. F., Olson, R. K., Chhabildas, N. & Hulslander, J. (2005). Neuropsychological analyses of comorbidity between reading disability and Attention Deficit Hyperactivity Disorder: in search of the common deficit. *Developmental Neuropsychology*, *27*, 35-78.
- Woods, S. P., Lovejoy, D. W. & Ball, J. D. (2002). Neuropsychological characteristics of adults with ADHD: a comprehensive review of initial studies. *The Clinical Neuropsychologist*, *16*, 12-34.
- Woods, S. P., Lovejoy, D. W., Stutts, M. L., Ball, J. D. & Fals-Stewart, W. (2002). Comparative efficiency of a discrepancy analysis for the classification of Attention-Deficit/Hyperactivity Disorder in adults. *Archives of Clinical Neuropsychology*, *17*, 351-369.
- World Health Organisation (1992). *ICD-10 classifications of mental and behavioural disorder: Clinical descriptions and diagnostic guidelines*. Geneva: World Health Organisation.
- World Health Organisation (1978). *Cerebrovascular disorders: a clinical and research classification*. Geneva: World Health Organisation.
- Yang, L., Ji, N., Guan, L. L., Chen, Y., Qian, Q. J. & Wang, Y. F. (2007). Comorbidity of attention deficit hyperactivity disorder in different age group. *Journal of Peking University. Health Sciences*, *39*, 229-232.
- Yang, S., Hua, P., Shang, X., Hu, R., Mo, X. & Pan, X. (2013). Predictors of early post ischemic stroke apathy and depression: a cross-sectional study. *BMC Psychiatry*, *13*, 164.
- Young, S. & Gudjonsson, G. H. (2005). Neuropsychological correlates of the YAQ-S and YAQ-I self- and informant-reported ADHD symptomatology, emotional and social problems and delinquent behaviour. *British Journal of Clinical Psychology*, *44*, 47-57.
- Young, S., Morris, R., Toone, B. & Tyson, C. (2007). Planning ability in adults with Attention-Deficit/Hyperactivity Disorder. *Neuropsychology*, *21*, 581-589.
- Ziegler, E., Blocher, D., Bro, J. & Rösler, M. (2003). Erfassung von Symptomen aus dem Spektrum des Hyperkinetischen Syndroms bei Häftlingen einer Justizvollzugsanstalt. *Recht & Psychiatrie*, *21*, 17-21.
- Zillmer, E. A., Waechtler, C., Harris, B., Khan, F. & Fowler, P. C. (1992). The effects of unilateral and multifocal lesions on the WAIS-R: a factor analytic study of stroke patients. *Archives of Clinical Neuropsychology*, *7*, 29-40.
- Zimmermann, P. & Fimm, B. (2009). *Testbatterie zur Aufmerksamkeitsprüfung (TAP)*. Herzogenrath: Psychologische Testsysteme.
- Zinn, S., Bosworth, H. B., Hoenig, H. M. & Swartzwelder, H. S. (2007). Executive function deficits in acute stroke. *Archives of Physical Medicine and Rehabilitation*, *88*, 173-180.

- Zinn, S., Dudley, T. K., Bosworth, H. B., Hoenig, H. M., Duncan, P. W. & Horner, R. D. (2004). The effect of poststroke cognitive impairment on rehabilitation process and functional outcome. *Archives of Physical Medicine and Rehabilitation*, *85*, 1084-1090.
- Zucker, M., Morris, M. K., Ingram, S. M., Morris, R. D. & Bakeman, R. (2002). Concordance of self- and informant ratings of adults' current and childhood Attention-Deficit/Hyperactivity Disorder symptoms. *Psychological Assessment*, *14*, 379-389.
- Zwaan, M., Größ, B., Müller, A., Graap, H., Martin, A., Glaesmer, H. et al. (2012). The estimated prevalence and correlates of adult ADHD in a German community sample. *European Archives of Psychiatry and Clinical Neuroscience*, *262*, 79-86.

APPENDICES

APPENDIX A - Contents of the WAIS-IV

Table 1. WAIS-IV subtests, CHC broad abilities, and cognitive capacities needed for effective performance

	Description	CHC ^a broad abilities	Primary cognitive capacities ^b	Secondary cognitive capacities ^c	
Core subtests	BD^d	Re-creation of specific visual designs with building blocks <u>Designed to measure:</u> nonverbal concept formation and reasoning, organization, visual-motor coordination	G _v ^q	reasoning with nonverbal/visual stimuli	visual acuity; planning abilities; WM and attention; visualization and visual perception/discrimination; motor coordination; visuo-motor/motor/visual processing speed
	SI^e	Finding the similarity between two words <u>Designed to measure:</u> verbal concept formation, verbal reasoning, lexical knowledge, induction	G _c ^r , G _f ^s	retrieval of verbal content and knowledge; reasoning with auditory verbal content	auditory acuity/discrimination; working memory and attention; expressive and receptive language
	DS^f	Recalling a sequence of digits; forwards, backwards, and in sequence <u>Designed to measure:</u> WM, memory span, cognitive flexibility, mental alertness, attention, mental manipulation, visuospatial imaging	G _{sm} ^t	initial registration of auditory verbal information; working memory for verbal information; mental manipulation	auditory acuity/discrimination; attention; auditory/mental processing speed; retrieval of verbal information from long-term storage; expressive language; sequencing/organization
	MR^g	Selecting a response option to complete a matrix or series of objects <u>Designed to measure:</u> general sequential reasoning, spatial ability, perceptual organization	G _f ^s	reasoning with nonverbal, visual stimuli	visual acuity; working memory and attention; visualization and visual; perception/discrimination; visual processing speed
	VC^h	Defining words <u>Designed to measure:</u> lexical knowledge, verbal concept formation	G _c ^u	retrieval of semantic knowledge from long-term memory	auditory acuity/discrimination; attention; expressive and receptive language

Table 1. (continued)

Core subtests	ARⁱ	Solving series of arithmetic problems mentally <u>Designed to measure:</u> WM, numeral reasoning, mental manipulation, concentration, attention, mental alertness	Gf [§] , Gsm ^t , Gq ^v	initial registration of auditory verbal information; verbal working memory; mental manipulation	auditory acuity/discrimination; attention; auditory/mental processing speed; retrieval of verbal information from long-term storage; math calculation/problem-solving skills; expressive language; sequencing/organization
	SSI	Searching and recognizing whether a target appears in a group of symbols <u>Designed to measure:</u> visual motor coordination, cognitive flexibility, attention, concentration, psychomotor speed, short-term visual memory	Gs ^w	motor/visual/visuo-motor processing speed	visual acuity; working memory and attention; visualization and visual perception/discrimination; graphomotor capacity; language representation
	VP^k	Restructuring various puzzles with three pieces out of five response options <u>Designed to measure:</u> nonverbal reasoning, analyze and synthesize abstract visual stimuli, spatial visualization	Gv ^q	visual perceptual processing; reasoning with nonverbal/visual stimuli	visual acuity; working memory and attention; visualization and visual perception/discrimination; visual processing speed
	IN^l	Answering various questions of general knowledge topics <u>Designed to measure:</u> general knowledge	Gc ^u	retrieval of semantic knowledge from long-term storage	auditory acuity; auditory processing speed; working memory and attention; expressive and receptive language
	CD^m	Copying symbols to their paired numbers <u>Designed to measure:</u> visual motor coordination, visual scanning, cognitive flexibility, attention, concentration, psychomotor speed, short-term visual memory, learning	Gs ^w	motor/visual/visuo-motor processing speed	visual acuity; working memory and attention; visualization and visual perception/discrimination; graphomotor capacity; language representation; multitasking/organization

Table 1. (continued)

Supplemental subtests	LNSⁿ	Recalling numbers and letters in ascending order <u>Designed to measure:</u> WM, sequential processing, mental manipulation, attention, concentration, memory span, cognitive flexibility	Gsm ^t	initial registration of auditory verbal information; verbal working memory; mental manipulation	auditory acuity/discrimination; attention; auditory/mental processing speed; retrieval of verbal information from long-term storage; expressive language; sequencing/organization
	CA^o	Marking target shapes in a structured arrangement of shapes <u>Designed to measure:</u> visual selective attention, vigilance, perceptual speed, visual-motor ability	Gs ^w	motor/visual/visuo-motor processing speed	visual acuity; working memory and attention; visualization and visual perception/discrimination; grapho-motor capacity; language representation; multitasking/organization
	PC^p	Identification of the missing part in a picture <u>Designed to measure:</u> visual perception, organization, concentration, visual recognition	Gv ^q , Gc ^u	reasoning with nonverbal/visual stimuli	visual acuity; working memory and attention; visualization and visual perception/discrimination; visual processing speed; language representation of visual stimuli

Source. (Flanagan et al., 2012; Wechsler et al., 2008).

Note. Secondary capacities include also executive functions, which are not included in this table because they are intricately involved in the performance of all subtests to the extent that they cue and direct the use of the other cognitive capacities. It is important to note that executive functions are mostly “minimized through the use of explicit directions, teaching examples, and example responses” in the WAIS-IV (Lichtenberger & Kaufman, 2009b, Appendix B.2, p.5).

^aCattell-Horn-Carroll, ^bmost often required and essential for effective performance of subtests, ^cprerequisites or basic abilities to perform the subtests, ^dBlock Design, ^eSimilarities, ^fDigit Span, ^gMatrix Reasoning, ^hVocabulary, ⁱArithmetic, ^jSymbol Search, ^kVisual Puzzles, ^lInformation, ^mCoding, ⁿLetter-Number Sequencing, ^oCancellation, ^pPicture Completion, ^qVisual Processing, ^rCrystallized Intelligence, ^sFluid Reasoning, ^tShort-Term Memory, ^vQuantitative Knowledge, ^wProcessing Speed.

APPENDIX B - Study I

Theiling, J., & Petermann, F. (2014). Neuropsychological Profiles on the WAIS-IV of ADHD adults. *Journal of Attention Disorders*. Retrieved October 10, 2014, from: <http://jad.sagepub.com/content/early/2014/01/21/1087054713518241.abstract>

APPENDIX C - Study II

Theiling, J., Petermann, F., & Daseking, M. (2013). WAIS-IV profiles in first-ever unilateral ischemic stroke patients. *Zeitschrift für Neuropsychologie*, *24*, 239-252.

APPENDIX D - Study III

Theiling, J., Petermann, F., & Daseking, M. (2013). Zusammenhang zwischen selbsteingeschätzter ADHS-Symptomatik und der Leistungsfähigkeit in der WAIS-IV. *Gesundheitswesen*, 75, 768-744.

APPENDIX E - Statement of the author's contribution to each publication

The current doctoral thesis was conducted by M.Sc. Johanna Theiling according to § 6 subparagraph 5 of the formal requirements for doctoral candidates at the University of Bremen. The thesis is based on three empirical studies, which were published by national and internationally peer-reviewed journals. Different working steps were required to accomplish this empirical research. The contribution to all publications includes the steps of conception and design, the literature research, data collection, preparation, analyses, and interpretation, as well as the drafting and revisions of the manuscripts. Table E2 depicts the extent of the contribution of Johanna Theiling to each study in the specific working steps. Whether Johanna Theiling contributed solely to the working step is indicated as *fully*, whether Johanna Theiling made the most important contribution is indicated as *mostly* or whether Johanna Theiling and at least one further contributor shared an equivalent amount of contribution is indicated as *partly*. Apart from this, Johanna Theiling was in a steady and consistent cooperation with the other contributors throughout the present research. Their ideas and suggestions had always a great impact.

Table 2. The contribution to the empirical studies

	Empirical studies		
	Study I ^a	Study II ^b	Study III ^c
Conception	Fully	Fully	Partly
Literature research	Fully	Fully	Mostly
Data collection	Mostly	Mostly	Mostly
Data preparation	Fully	Fully	Fully
Data analysis	Fully	Fully	Partly
Result interpretation	Fully	Fully	Partly
Drafting of manuscript	Fully	Mostly	Partly
Manuscript revision	Mostly	Mostly	Mostly

^aNeuropsychological Profiles on the WAIS-IV of ADHD adults - *Journal of Attention Disorders*, ^bWAIS-IV profiles in first-ever unilateral ischemic stroke patients - *Zeitschrift für Neuropsychologie*, ^cZusammenhang zwischen selbsteingeschätzter ADHS-Symptomatik und der Leistungsfähigkeit in der WAIS-IV - *Gesundheitswesen*.

M.Sc. Johanna Theiling and the other contributors, Prof. Dr. Franz Petermann, and PD Dr. Monika Daseking, herewith certify that the statement of the author's contribution to each publication made by Johanna Theiling is accurate and that permission is granted for these studies to be included in her doctoral thesis.

Bremen, 01.10.2014

.....
Prof. Dr. Franz Petermann

.....
PD Dr. Monika Daseking

.....
M.Sc. Johanna Theiling

APPENDIX F - Declaration of originality

In accordance with § 6 subparagraph 5 of the formal requirements for doctoral candidates at the University of Bremen, I hereby declare that the present dissertation represents my own original work except where specifically acknowledged. Wherever contributions of others are involved, every effort is made to indicate this clearly, with due reference to the literature. I further declare that this dissertation does not contain any material which has previously been accepted and is not currently considered for the award of any other university degree in my name. In addition, I certify that no part of the present thesis will, in the future, be used in a submission in my name, for any other degree in any university without the prior approval of the University of Bremen.

Bremen, 01.10.2014

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M.Sc. Johanna Theiling