Beyond Endophenotypes:
Direct and Indirect Effects of Sustained Attention and Behavioral Inhibition on Attention Deficit Hyperactivity Disorder

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List of Abbreviations

ACC	Anterior cingulate cortex
ADHD	Attention Deficit Hyperactivity Disorder
ANCOVA	Analysis of covariance
ANDRA2A	α2A receptor gene
BART	Balloon Analogue Risk Task
CBCL	Child Behavior Checklist
CCT	Columbia Card Task
CD	Conduct Disorder
CFT 20-R	Culture Fair Test 20-R
CHT	Choline transporter gene
CI	Confidence interval
CPT	Continuous Performance Task
DAT1	Dopamine transporter gene
DBH	Dopamine beta hydroxylase gene
DDC	Dopa decarboxylase gene
DMN	Default mode network
DRD2	Dopamine D2 receptor gene
DRD4	Dopamine D2 receptor gene
DSM-V	Diagnostic and Statistical Manual of Mental Disorders, 5th edition
DTI	Diffusion tensor imaging
EEG	Electroencephalogram
ERN	Error related negativity
ERP	Event related potential
FA	Fractional anisotropy
FBB-ADHS	German ADHD Rating Scale
fMRI	Functional magnetic resonance imaging
GDT	Game of Dice Task
GIDeCA	Gene-by-Environment Interactions on Decision Making in Children with ADHD
GLM	General linear model
ICD-10	International Classification of Diseases, 10th edition
IFG	Inferior frontal gyrus
IGT	Iowa Gambling Task

ISI	Interstimulus interval
LC	Locus coeruleus
MAO-A	Monoamine oxidase-A gene
NST	No Six Task
NET1	Norepinephrine transporter gene
ODD	Oppositional defiant disorder
PCS	Post-conflict slowing
PCE	Post-conflict error reduction
PES	Post-error slowing
PFC	Prefrontal cortex
pre-SMA	pre-Supplementary motor area
RTV	Response time variability
SAN	Sustained attention network
SDQ	Strength and Difficulties Questionnaire
SES	Socioeconomic status
SMA	Supplementary motor area
sMRI	Structural magnetic resonance imaging
SSRT	Stop signal reaction time
TPH2	Tryptophan hydroxylase-2

Zusammenfassung (German Abstract)

Aktuelle Theorien, welche die Ätiologie der Aufmerksamkeitsdefizit-/Hperaktivitätsstörung (ADHS) erklären, fokussieren auf kognitive und motivationale Defizite, neuronale Veränderungen oder genetische Prädispositionen. Diese unterschiedlichen Erklärungsmodelle werden in dieser Arbeit in ein Rahmenkonzept integriert. Das Rahmenkonzept basiert auf traditionellen Endophenotypenansätzen und erweitert diese, indem nicht nur *direkte* Effekte von kognitiven und motivationalen Funktionen auf ADHS Symptome berücksichtigt werden, sondern auch *indirekte* Effekte von kognitiven und motivationalen Funktionen über höhere psychologische Funktionen. Sowohl andauernde Aufmerksamkeit als auch Verhaltensinhibition sagen riskantes Entscheidungsverhalten vorher und riskantes Entscheidungsverhalten wiederum hängt mit Impulsivität und ADHS Symptomen zusammen. Daher wird in dieser Arbeit untersucht, inwiefern indirekte Effekte über riskantes Entscheidungsverhalten direkte Zusammenhänge zwischen andauernder Aufmerksamkeit, Verhaltensinhibition und ADHS Symptomen erklären können. *Methode.* Insgesamt erfüllten 95 Kinder (71 Jungen; 40 Kinder mit einer ADHS Diagnose) im Alter von 7;4 und 13;8 Jahren die Einschlusskriterien in die Studie. Die Kinder bearbeiteten drei computerisierte kognitive Aufgaben zur Messung andauernder Aufmerksamkeit und Verhaltensinhibition sowie drei Aufgaben zur Messung riskanten Entscheidungsverhaltens. *Ergebnisse.* ADHS Symptome konnten signifikant durch Defizite in andauernder Aufmerksamkeit erklärt werden, $F(1, 67) = 20.10$, $p < .001$, $\eta_p^2 = .231$. Dieser Effekt wurde partiell durch riskantes Entscheidungsverhalten mediiert, $b = 0.19$, 90% KI: [0.01, 0.41]. Ein weiterer indirekter Effekt von Verhaltensinhibition über riskantes Entscheidungsverhalten auf ADHS Symptome wurde beobachtet, $b = 8.57$, 99% KI: [1.38, 21.20]. Kinder mit Schwierigkeiten in Daueraufmerksamkeit oder Verhaltensinhibition trafen häufiger riskante Entscheidungen und wiesen daher stärkere ADHS Symptome auf. *Schlussfolgerung.* Theorien, welche die Ätiologie der ADHS erklären, sollten Defizite in andauernder Aufmerksamkeit als ein Kerndefizit von Kindern mit ADHS aufnehmen. Außerdem sollten die Theorien neben direkten Effekten kognitiver oder motivationaler Defizite auf ADHS Symptome auch indirekte Effekte kognitiver oder motivationaler Defizite über höhere psychologische Funktionen berücksichtigen.

Abstract

Current theoretical accounts investigating the etiology of Attention Deficit Hyperactivity Disorder (ADHD) rely on cognitive or motivational deficits, neuronal impairments, and genetic predispositions. Relying on the endophenotype concept, this thesis provides a theoretical framework to integrate these lines of research. The framework moves beyond a mere endophenotype approach as both *direct* effects of cognitive and motivational functions on ADHD symptoms and *indirect* effects of cognitive or motivational functions on ADHD symptoms through higher-order psychological functions are considered. Both, sustained attention and behavioral inhibition, are related to risky decision-making and risky decision-making in turn is associated with impulsivity and ADHD symptoms. Hence, this thesis addresses the question whether indirect effects through risky decision-making can account for relations between sustained attention, behavioral inhibition, and ADHD symptoms.

Method. Ninety-five children (71 males; 40 children with a diagnosed ADHD) aged between 7;4 and 13;8 years met inclusion criteria for the current study. Children worked on three computerized cognitive tasks measuring sustained attention and behavioral inhibition as well as three behavioral tasks measuring risky decision-making. ***Results.*** A sustained attention composite score significantly predicted ADHD symptoms, $F(1, 67) = 20.10$, $p < .001$, $\eta_p^2 = .231$. The effect was partially mediated by risky decision-making, $b = 0.19$, 90% CI: [0.01, 0.41], and an additional indirect of behavioral inhibition on ADHD symptoms through risky decision-making was observed, $b = 8.57$, 99% CI: [1.38, 21.20]. Children who had difficulties in sustained attention or behavioral inhibition made a higher number of risky decisions and in turn had more or stringer ADHD symptoms. ***Conclusion.*** Theoretical accounts investigating the etiology of ADHD should consider sustained attention as a core deficit of ADHD and incorporate indirect effects through higher-order psychological functions.

The Blind Men and the Elephant

by John Godfrey Saxe

It was six men of Indostan
To learning much inclined,
Who went to see the Elephant
(Though all of them were blind),
That each by observation
Might satisfy his mind.

The First approached the Elephant,
And happening to fall
Against his broad and sturdy side,
At once began to bawl:
"God bless me! but the Elephant
Is very like a WALL!"

The Second, feeling of the tusk,
Cried, "Ho, what have we here,
So very round and smooth and sharp?
To me 'tis mighty clear
This wonder of an Elephant
Is very like a SPEAR!"

The Third approached the animal,
And happening to take
The squirming trunk within his hands,
Thus boldly up and spake:
"I see," quoth he, "the Elephant
Is very like a SNAKE!"

The Fourth reached out an eager hand,
And felt about the knee
"What most this wondrous beast is like
Is mighty plain," quoth he:
"'Tis clear enough the Elephant
Is very like a TREE!"

The Fifth, who chanced to touch the ear,
Said: "E'en the blindest man
Can tell what this resembles most;
Deny the fact who can,
This marvel of an Elephant
Is very like a FAN!"

The Sixth no sooner had begun
About the beast to grope,
Than seizing on the swinging tail
That fell within his scope,
"I see," quoth he, "the Elephant
Is very like a ROPE!"

And so these men of Indostan
Disputed loud and long,
Each in his own opinion
Exceeding stiff and strong,
Though each was partly in the right,
And all were in the wrong.

Introduction

The parable of the blind men and the elephant is well known in Indian cultures as for instance the Hindu culture, the Buddhist culture, and the Jain Muslim culture. In Western society it became especially famous by the poem of John Godfrey Saxe (Gardner, 1992). In the parable a group of blind men did not have any previous knowledge about an elephant. Thus, they touched an elephant in order to gain a mental representation of what an elephant is like. However, each of them touched only one and a different part of the elephant, so that they were unable to reach consensus whether an elephant resembles a wall, a spear, a snake, a tree, a fan, or a rope. This picture of people being unable to see a whole multidimensional phenomenon due to having experienced only one single dimension of the phenomenon has recently been transferred to science when Steger (2003) compared the blind men from the Indian parable to different economic scholars and their view on the primary domain of globalization. While each scholar correctly identified and analyzed an important dimension of globalization, they failed to incorporate their knowledge into a larger multidimensional framework, thus losing sight of the interconnected whole phenomenon (Steger, 2003).

In a similar way, a comparison can be drawn between the blind men from the Indian parable and the scientific situation within the field of Attention Deficit Hyperactivity Disorder (ADHD). Over the last decade researchers from different scientific disciplines, as for instance, medicine, psychology, neuroscience, and genetics investigated the etiology and pathogenesis of ADHD. Comparable to the parable, the identified psychological, neuronal, genetic, and social-environmental factors that referred or contributed to ADHD have only been weakly related to each other and have not been incorporated into a larger framework. However, a framework that incorporates these factors might be especially useful to investigate the etiology and pathogenesis of a psychiatric disorder like ADHD because psychiatric disorders do not represent homogeneous syndromes (Hyman, 2010; Miller, 2010). In contrast, diagnostic criteria of psychiatric disorders are polythetic, meaning that diagnoses can be derived from different combinations of specific criteria listed under the disorder (Diagnostic and Statistical Manual of Mental Disorders, DSM-V; American Psychiatric Association, APA, 2013; Hyman, 2010). Hence, researchers on ADHD widely agree on viewing ADHD as a heterogeneous disorder with regard to behavioral symptoms, neuropsychological impairments, and genetic predispositions (Fair, Bathula, Nikolas, & Nigg, 2012; Sonuga- Barke, Bitsakou, & Thopmson, 2010; Wåhlstedt, Thorell, & Bohlin, 2009).

Therefore, the major aims of this thesis are (1) to develop a research framework for ADHD that incorporates psychological, neuronal, genetic and environmental factors to deal

with these different forms of heterogeneity and (2) to test specific predictions that can be derived from such a framework. The first chapter provides the reader with an overview of ADHD and the main neuropsychological theories (Johnson, Wiersema, & Kuntsi, 2009). A special emphasis is put on recent multiple pathway models of ADHD (de Zeeuw, van Dijk, van Belle, & Durston, 2012; Sonuga-Barke et al., 2010) as these models acknowledge the heterogeneity of ADHD and offer the opportunity to relate psychological, neuronal, genetic, and social-environmental factors.

In contrast to recent models of ADHD that do not consider an attentional deficit (de Zeeuw et al., 2012; Sergeant, 2000; Sonuga-Barke et al., 2010), theoretical accounts that emphasize on sustained attention and attentional deficits in ADHD are included, as recent evidence suggested a core deficit of sustained attention in children with ADHD (Tsal, Shalev, & Merovach, 2005). Finally, this chapter introduces the reader to the concept of endophenotypes (Gottesman & Gould, 2003) on which the current thesis relies to combine the different lines of research. Based on multiple pathway models and the endophenotype concept introduced in Chapter 1, Chapter 2 two offers a research framework of ADHD that (1) summarizes the current state of knowledge about psychological, neuronal, genetic, and social-environmental factors related to ADHD, and (2) connects the different lines of research. The research framework moves beyond a mere endophenotype approach by considering higher-order cognitive constructs as for instance risky decision-making that might mediate associations between endophenotypes and ADHD. Chapter 3 describes the current study that aimed at testing predictions derived from the outlined framework with a special regard to sustained attention and the role of higher-order cognitive functions. Results from the current study are reported in Chapter 4, while Chapter 5 concludes with a discussion of the study findings in relation to the outlined research framework of ADHD.

1. Theoretical Background

This chapter gives a brief overview of ADHD (1.1.) and the main neuropsychological theories of the disorder: the executive dysfunction theory (Barkley, 1997; 1.1.1.), the delay aversion theory (Sonuga-Barke, Taylor, Sembi, & Smith, 1992; 1.1.2.), the state regulation theory (Sergeant, 2000; 2005; 1.1.3.), the notion of impaired temporal processing (Toplak, Dockstader, & Tannock, 2006; 1.1.4.), and finally the emphasize on a deficit in attentional processes (Swanson et al., 1998; Tsal et al., 2005; 1.1.5.). This first part of the theoretical section closes with the description of multiple pathway models of ADHD that combine some of the other theories (de Zeeuw et al., 2012; Sonuga-Barke et al., 2010; 1.1.6.). In the second part of this section the reader is introduced to the concept of endophenotypes (Gottesman & Gould, 2003; 1.2.), a well-known concept in clinical psychopathology (Insel & Collins, 2003; see Miller & Rockstroh, 2013 for a review) that has already been applied to the relation of response inhibition and ADHD (Crosbie, Pérusse, Barr, & Schachar, 2008; Durston, de Zeeuw, & Staal, 2009).

1.1. Attention Deficit Hyperactivity Disorder

The first reference to an ADHD like disorder can be traced back to Melchior Adam Weikard's textbook *Der Philosophische Arzt* published in 1775 (Barkley & Peters, 2012). The textbook contained a chapter on *attentio volubilis* (volatile attention) in which Weikard described an inattentive person as someone, who only hears half of everything, who is impatient, reckless, and imprudent (Barkely & Peters, 2012). This early description of *attentio volubilis* corresponds strongly to the core symptoms inattention, hyperactivity, and impulsivity of ADHD as defined by the DSM-V or the International Classification of Diseases (ICD-10; World Health Organization, 2009). According to both classification systems children with ADHD often fail to give close attention to details or make careless mistakes, often run about or climb in situations where it is inappropriate, or have difficulties waiting their turn. To obtain a diagnosis, the symptoms have to be present prior to the age of twelve (DSM-V; prior to the age of six; ICD-10), have persisted for at least six months and have a negative impact on social or academic activities. Following these criteria, estimations of the prevalence of ADHD in children and adolescents based on standardized procedures in representative samples of the community revealed worldwide prevalence rates of five to seven percent (Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007; Polanczyk, Willcutt, Salum,

Kieling, & Rohde, 2014; Willcutt, 2012). However, ADHD is a very heterogeneous disorder and not every child displays symptoms in all three categories. Therefore, the DSM-V (2013) distinguishes between three presentations of ADHD: a predominantly inattentive presentation, a predominantly hyperactive/impulsive presentation and a combined presentation (Dalsgaard, 2013). Yet, heterogeneity does not only manifest on a behavioral level, but also on a genetic level (Franke, Neale, & Faraone, 2009) as well as on a level of psychological constructs (Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005) meaning that the disorder might be derived by different etiologies.

With heritability rates between 60% and 90% derived from twin studies (Waldman & Gizer, 2006) genetics are assumed to play a major role in the etiology of ADHD (Franke et al., 2009). However, a large number of candidate gene studies reported only small effect sizes and often could not be replicated (Faraone, Doyle, Mick, & Biederman, 2001) leading to the assumption that ADHD is a multi-genetic disorder (Banaschewski, Becker, Scherag, Franke, & Coghill, 2010; Faraone et al., 2005; Gizer, Ficks, & Waldman, 2009; but see Wallis, Russel, & Muenke, 2008). Various reasons have been proposed to explain this difference between large heritability estimates on the one hand and small effects of single genes on the other hand. These reasons include large effects of rare genetic variants that are poorly detectable, low power to detect gene-gene interactions, and an inadequate accounting for shared environmental effects between relatives (Eichler et al., 2010; Manolio et al., 2009). Besides approaches that address these pure genetic reasons, two different approaches have been used to investigate the relation of genetics and ADHD and genetics and psychiatric disorders in general: gene-environment interactions and endophenotypes (Caspi & Moffitt, 2006).

The approach of gene-environment interactions is based on two observations: First, in addition to genetic factors, environmental factors have an impact on ADHD as well (Banarjee, Middleton, & Faraone, 2007), and second, there is heterogeneity in children's response to these environmental factors (Nigg, Nikolas, & Burt, 2010; Moffitt, Caspi, & Rutter, 2005). It is further assumed that this heterogeneity in responding to environmental factors is due to differential susceptibility to these environmental factors depending on the child's genotype (Dick, 2011; see Nigg et al., 2010 for a review).

The endophenotype approach, however, replaces ADHD or ADHD symptoms with intermediate phenotypes, called 'endophenotypes' (see Section 1.2.) that are thought to have a simpler genetic underpinning than the disorder itself, so that it might be easier to identify genes associated with endophenotypes than genes associated with ADHD (Franke et al., 2009;

Gottesman & Gould, 2003). Psychological constructs (e.g., behavioral inhibition or sustained attention; see Sections 2.1. and 2.2.) as derived from psychological theories of ADHD (see Sections 1.1.1. – 1.1.6.) might constitute potential endophenotypes (Crosbie et al., 2008; Doyle et al., 2008; Rommelse, Altink, Martin, Buschgens, Faraone et al., 2008). However, large heterogeneity has also been reported for psychological constructs. For instance, only about 20%-50% of the children with ADHD exhibit deficits in behavioral inhibition (see Section 2.1.), or sustained attention (see Section 2.2.) respectively (de Zeeuw et al., 2012; Nigg et al, 2005; Sonuga-Barke et al., 2010). Nevertheless, this heterogeneity is much smaller than the genetic heterogeneity with single genes explaining only up to 5% of the variance in ADHD (Faraone et al., 2005). Therefore, the following sections (Sections 1.1.1. – 1.1.6.) introduce the main theories of ADHD from which different psychological endophenotypes can be derived.

1.1.1. The Executive Dysfunction Theory

The executive dysfunction theory tries to explain ADHD symptoms for children with predominantly hyperactive or impulsive symptoms, but not for children with predominantly inattentive symptoms (Barkley, 1997). The theory assumes a hierarchical structure with behavioral inhibition (see Section 2.1.) as the core deficit in ADHD. Behavioral inhibition is thought to provide higher-order executive functions (e.g., working memory, language, affect regulation) with the necessary temporal delay to respond in accordance with internal goals. Thus, deficient behavioral inhibition in ADHD leads to impairments in these higher-order executive functions that in turn lead to ADHD symptoms as for instance a decreased motor control, impaired goal directed responses, or diminished feedback sensitivity (Barkley, 1997). An essential component of the executive dysfunction theory is the relation of behavioral inhibition to neural circuits within the prefrontal cortex (PFC; Fuster, 2002, 2008). Relying on lesion studies revealing that lesions in the PFC sometimes are accompanied with symptoms of hyperactivity, impulsivity, and inattention (see Fuster, 2008 for an overview), the theory claims that deficits in behavioral inhibition can be fully explained by structural and functional abnormalities within the PFC (see Section 2.1.2.).

Despite supporting evidence for an executive function deficit in ADHD and a deficit in behavioral inhibition in particular (see Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005 for a meta-analysis), the theory faces several problems (Johnson et al., 2009). First, the theory does not account for all symptoms of ADHD (Barkley, 1997; Willcutt et al., 2005) and only

20% to 50% of children with ADHD exhibit deficits in behavioral inhibition (Nigg et al., 2005; Sonuga-Barke et al., 2010). Second, the terms executive function and behavioral inhibition are ill defined (Jurado & Rosselli, 2007; see Section 2.1. for the definition used in this thesis) and several measurement problems exist (Miyake, Friedman, Emerson, Witzki, & Howerter, 2000). Arguably, the most severe problem is the task inpurity problem. Neuropsychological tests often do not only tap at behavioral inhibition or other single executive functions, but instead include systematic variance attributable to task-unrelated processes and different cognitive functions (Miyake et al., 2000). These problems make it difficult to produce testable hypothesis with regard to behavioral inhibition as the real core deficit of ADHD and the relation of behavioral inhibition and neural circuits within the PFC in particular (Johnson et al., 2009).

Regardless of these difficulties, the executive dysfunction theory stimulated research about ADHD on a behavioral, neuronal, and genetic level (see Sections 2.1.1.- 2.1.3.) and meta-analyses confirmed an inhibitory deficit in ADHD both on a behavioral (Willcutt et al., 2005) and neuronal level (Hart, Radua, Nakao, Mataix-Cols, & Rubia, 2013). Thus, behavioral inhibition might be one key deficit in ADHD.

1.1.2. The Delay Aversion Theory

In contrast to other theories of ADHD that focus on cognitive deficits (see Section 1.1.1., Section 1.1.3. and Section 1.1.4), the delay aversion theory focuses more on motivational and emotional processes in ADHD. According to this theory, children with ADHD experience a negative emotional reaction to the experience of delay, which is behaviorally manifested in attempts to avoid the delay situation or to reduce the sensation of delay (Sonuga-Barke, 2005). The impulsive, hyperactive, and inattentive symptoms of ADHD therefore represent functional expressions of delay aversion (Sonuga-Barke, 2002) with cognitive deficits in executive functions arising as secondary effects (Sonuga-Barke, Williams, Hall, & Saxton, 1996).

Two motivational mechanisms are supposed to underlie delay aversion: First, a shortened delay-of-reinforcement gradient as defined by a higher discounting rate of the value of future objects (Sagvolden, Johansen, Aase, & Russel, 2005) and second, the motivation to avoid or reduce delay (Bitsakou, Psychogiou, Thompson, & Sonuga-Barke, 2009; Sonuga-Barke, 2002). The shortened delay-of-reinforcement gradient leads to repeated failures in delay situations, that are consequently associated with negative emotions. Hence, the

avoidance of delay situations prevents from experiencing negative emotions, so that by means of negative reinforcement the avoidance of delay situations is increased (Sonuga-Barke, 2002, 2003).

Despite supporting evidence that delay aversion is involved in ADHD (see Section 2.2.; Sonuga-Barke, Sergeant, Nigg, & Willcutt, 2008 for a review), the theory faces several problems. First, only about 30% of children with ADHD exhibit delay aversion, indicating that delay aversion does not necessarily lead to ADHD (Sonuga-Barke et al., 2010). Second, even for children exhibiting delay aversion it does not explain the whole range of ADHD symptoms (Marco et al., 2009). Furthermore, although ADHD symptoms are thought to arise as a response to delay, no clear rule exists to predict situations in which symptoms of hyperactivity will and situations in which symptoms of inattention or impulsivity will occur, making it difficult to falsify the theory (Johnson et al., 2009).

Regardless of these difficulties, it is widely acknowledged that motivational deficits, including delay aversion, are one key deficit in ADHD (Marco et al., 2009; Sagvolden et al., 2005; Sergeant, 2005; Sonuga-Barke, 2002).

1.1.3. Impaired Temporal Processing

While the executive dysfunction theory and the delay aversion theory argue for cognitive or motivational deficits as the core deficits in ADHD, the account of impaired temporal processing (Toplak et al., 2006) assumes that the core problem of children with ADHD is a deficit in temporal processing caused by structural and functional abnormalities in neural circuits involving the cerebellum. Deficits in temporal processing are assumed to result in problems in motor timing and action control, finally leading to symptoms of ADHD (Toplak et al., 2006). This theoretical account received strong support from neuroscience, as the cerebellum has been identified as a key structure for temporal processing (Ivry & Spencer, 2004), motor control (Manto et al., 2012), and executive functions (Koziol et al., 2014; Koziol, Budding, & Chidekel, 2012).

However, despite supporting evidence for deficits in temporal processing (Toplak et al., 2006) as well as functional and structural abnormalities within the cerebellum in children with ADHD (Noreika, Falter, & Rubia, 2013), the account faces several problems. First, no formal formulation of the account exists, making it difficult to state precise predictions that could be tested and falsified. Second, only about 20% to 50% of children with ADHD exhibit deficits in temporal processing, demonstrating that deficits in temporal processing do not

necessarily lead to ADHD (de Zeeuw et al., 2012; Sonuga-Barke et al., 2010).

Regardless of these difficulties, impaired temporal processing as one key deficit in ADHD became increasingly popular in the recent years and has been incorporated into theories of ADHD (de Zeeuw et al., 2012; Sonuga-Barke et al., 2010; see Section 1.1.6.).

1.1.4. The State Regulation Theory

The state regulation theory (Sergeant, 2000, 2005) is based on Sanders' cognitive energetic model (1983). The model states that task efficiency depends on elementary cognitive processes (e.g., stimulus preprocessing, feature extraction, response choice, motor adjustment) and the energy that is provided to perform these processes. Whether the cognitive processes can be efficiently performed is related to the arousal and activation levels of the participant. Arousal can be defined as time-locked phasic physiological responses to input stimuli, whereas activation refers to tonic physiological readiness to respond (Pribram & McGuiness, 1975). Arousal and activation are controlled by effort either activating or inhibiting the two systems in order to meet the task demands, while the effort system is under the control of an evaluation unit that monitors the current state of the arousal and activation systems (Sanders, 1983). The state regulation theory assumes that children with ADHD have difficulties in maintaining an optimal level of activation, possibly due to not allocating enough effort. Therefore, performance deficits in behavioral inhibition and executive function tasks do not represent a core deficit of ADHD but rather a deficit in adjusting energetic states in relation to environmental demands (Sergeant 2000, 2005). Consistent with the Yerkes-Dodson law (Yerkes & Dodson, 1908), the relation between energetic states and behavioral inhibition should follow an u-shaped curve, where children with ADHD exhibit the largest performance deficits under conditions of low and high activation, but not under conditions of medium activation (Sergeant, 2005).

Despite supporting evidence for an energetic dysfunction in children with ADHD (Metin, Roeyers, Wiersema, van der Meere, & Sonuga-Barke, 2012), the theory faces several problems. First, there are no direct measures for the energetic pools activation, arousal, and effort, and conclusions have mainly been derived from mere behavioral performance. Second, the model claims that differences between children with and without ADHD should be absent or at least minimal when children are in an optimal energetic state. However, specifying optimal energetic states should be context dependent and might vary between children, making it difficult to falsify the theory (Johnson et al., 2009).

Regardless of these difficulties, the state regulation theory has brought back the environment into research on ADHD, highlighting the importance to analyze environmental demands in relation to cognitive dysfunctions. In addition, the state theory offers an cognitive-energetic explanation for behavioral deficits and thus, represents an alternative to theoretical accounts that primarily focus on deficits in cognitive performance, in particular on deficits in behavioral inhibition or sustained attention.

1.1.5. The Model of Impaired Attention Systems

Early theories of ADHD emphasized problems of sustained attention as a core deficit in ADHD (Douglas, 1972; Douglas & Peters, 1979). However, no formal theory was stated and subsequent theories explained deficits in sustained attention in terms of a general understimulation (Zentall & Zentall, 1983), a consequence of a an inhibitory deficit (Barkley, 1997), or non-optimal energetic states (Sergeant, 2000). Only recently, did attentional accounts of ADHD reemerge.

According to the Petersen-Posner perspective on attention systems (Petersen & Posner, 2012; Posner & Petersen, 1990), several attention systems can be distinguished by their neuronal networks, namely (1) an alerting attention system that prepares and sustains attention in order to process signals, (2) an orienting attention system that prioritized the processing of sensory input by preselecting the modlity or location of the expected stimulus, and (3) an executive attention system that detects signals for a focal and conscious processing (Petersen & Posner, 2012; Posner & Petersen, 1990). Following this distinction, the attention model for ADHD links different symptoms of ADHD to the three attention systems (Swanson et al., 1998). Within this model, symptoms of sustained attention (e.g., ‘difficulties in sustaining attention’ or ‘avoiding sustained effort’) are related to the alerting attention system, while symptoms of selective and orienting attention (e.g., ‘the child is distracted by irrelevant stimuli’ or ‘fails to give close attention to details’) are related to the orienting attention system, and symptoms of impulsivity (e.g., ‘blurts out answers’ or ‘interrupts or intrudes’) are related to the executive attention system (Swanson et al., 1998). The model has been confirmed by empirical results (Tsal et al., 2005) indicating that children with ADHD were impaired in all three attentional functions. However, a deficit in sustained attention is the most pronounced deficit in children with ADHD, characterizing 85% of the children (Tsal et al., 2005).

Despite the supporting evidence for an impairment in attention systems in children with ADHD, particularly in sustained attention, the account faces several problems. First, only few studies tested all three functions (Johnson et al., 2008; Mullane, Corkum, Klein, McLaughlin, & Lawrence, 2011; Tsal et al., 2005) and inconsistend results have been reported, although sustained attention was always impaired in children with ADHD. However, not all children with ADHD are impaired in all attention systems, especially not in the orienting system (Johnson et al., 2008; Mullane et al., 2011; Tsal et al., 2005) and finally, the account does not exclude other mechanims that explain deficits in the behavioral performance in children with ADHD during attentional tasks. Thus, behavioral deficits might not indicate attentional deficits per se but rather a general underarousal (Zentall & Zentall, 1983) or non-optimal energetic states (Sergeant, 2000).

1.1.6. Multiple Pathway Models

Since the aforementioned theories are not able to explain the heterogeneity in ADHD and usually only hold for 20% to 50% of the children with ADHD, researchers of ADHD tried to combine different theoretical approaches into unified models. Sonuga-Barke (2002) first incorporated the executive dysfunction theory into his own theory of delay aversion. According to the dual pathway model, a deficit in behavioral inhibition, as proposed in the executive dysfunction theory, constitutes one pathway to ADHD symptoms, while delay aversion constitutes another pathway. Thus, impairment in one pathway would be sufficient for a child to exhibit ADHD symptoms. To add biological plausibility to these two pathways, the model proposes different neural circuits being involved: prefrontal areas for behavioral inhibition and meso-limbic reward circuits for delay aversion (Sonuga-Barke, 2002). However, in a recent study only about 50% of the children with ADHD exhibited deficits in behavioral inhibition or delay aversion (Sonuga-Barke et al., 2010).

Recent results from an exploratory factor analysis suggested that temporal processing might represent an additional pathway independent from behavioral inhibition and delay aversion (Sonuga-Barke et al., 2010). This extended triple pathway model received further support from neuroscientific research, claiming that the pathways could be distinguished by different neuronal circuits (Durston, van Belle, & de Zeeuw, 2011). Still, the three pathways are unable to explain all the heterogeneity in ADHD (de Zeeuw et al., 2012; Sonuga-Barke et al., 2010) and another exploratory factor analysis concluded that sustained attention might represent an additional forth pathway (de Zeeuw et al., 2012). Yet, further pathways might be

obtained when additional psychological constructs, for instance working memory, are considered (Coghill, Seth, & Matthews, 2014).

However, while the number of potential pathways is unclear, neurobiological evidence has been reported for behavioral inhibition, delay aversion, temporal processing, and sustained attention making these pathways biological plausible.

1.2. Endophenotypes

Endophenotypes have been conceptualized as intermediate phenotypes that causally connect genetic liabilities to psychiatric disorders (Cannon & Keller, 2006; Gottesman & Gould, 2003; Miller & Rockstroh, 2013 for an extensive review). Based on this conceptualization, endophenotypes can be neurophysiological, biochemical, endocrinal, neuroanatomical, cognitive, or neuropsychological (Gottesman & Gould, 2003). Thus, endophenotypes do not need to be biological themselves as long as they have a biological basis (Cannon & Keller, 2006; Kendler & Neale, 2010). However, in contrast to more general terms like “biomarkers”, “biological substrates” or “latent phenotypes”, endophenotypes need to be heritable (Kendler & Neale, 2010; Sabb et al., 2009). As this genetic underpinning of endophenotypes is assumed to be simpler than the genetic underpinning of traits and psychiatric disorders, endophenotypes mediating genetic effects on psychiatric symptoms are generally thought to be especially helpful to clarify genetic contributions to the development of complex psychological traits and the etiology of disorders (Insel & Collins, 2003; but see Flint & Munafo, 2007).

Criteria for endophenotypes vary between studies and specific definitions, but generally share several elements (Crosbie et al., 2008; Doyle et al., 2005; Kendler & Neale, 2010; Miller & Rockstroh, 2013). Notably, endophenotypes should (a) be correlated with the disorder of interest, (b) be reliable and measured by tools with sound psychometric properties, (c) be biologically related to the disorder of interest, (d) be heritable so that the endophenotypic variation in the population is at least partly caused by a genetic variation between individuals, and (e) be found in non-affected family members at a higher rate than in the general population (Crosbie et al., 2008; Doyle et al., 2005; Hasler, Drevets, Gould, Gottesman, & Manji, 2006; Kendler & Neale, 2010).

Recently, it has been suggested that behavioral inhibition, delay aversion, temporal processing, and sustained attention might constitute potential endophenotypes for ADHD (Bidwell, Willcutt, DeFries, & Pennington, 2007; Crosbie et al., 2008; Doyle et al., 2008;

Durston et al., 2009; Kuntsi, Andreou, Ma, Börger, & van der Meere, 2005; Rommelse, Altink, Martin, Buschgens, Faraone et al., 2008; Rommelse, Altink, Oosterlaan et al., 2008). However, only for behavioral inhibition, all criteria for endophenotypes have been examined (Crosbie et al., 2008; Durston et al., 2009; Kuntsi et al., 2005), leaving it unclear whether delay aversion, temporal processing, and sustained attention can really be regarded as endophenotypes. For instance, with regard to delay aversion, heritability has mainly been revealed for a shortened delay-of-reinforcement gradient (Anokhin, Golosheykin, Grant, & Heath, 2011; Kawamura et al., 2013; Paloyelis, Asherson, Mehta, Faraone, & Kuntsi, 2010) but not for the motivation to avoid or reduce delay. In similar, with regard to temporal processing, only few studies investigated associations with genetics at all (Rommelse, Oosterlaan, Buitelaar, Faraone, & Sergeant, 2007; Sysoeva, Tonevitsky, & Wackermann, 2010; Wiener, Lohoff, & Coslett, 2011). Finally, Section 2.2. presents an examination of the criteria for endophenotypes with regard to sustained attention.

2. A Research Framework for ADHD

Heterogeneity in ADHD is displayed on a symptom level as well as on the level of psychological constructs, neuronal associations, genetic liabilities, and social-environmental factors (Fair et al., 2012; Franke et al., 2009; Sonuga-Barke et al., 2010; Wåhlstedt et al., 2009). Hence, a framework that incorporates these factors might be especially useful to investigate the etiology and pathogenesis. Whereas theories or models of ADHD try to explain how and why different psychological, neuronal, genetic, and social-environmental factors are related ADHD including causal links and scientific laws, this chapter presents a research framework for the study of heterogeneity in ADHD, that connects different lines of research and thereby aims to guide researchers in planning studies, choosing measures, and selecting research questions (Imenda, 2014; McCloskey, 1991; Wacker, 1998). The center of the presented framework is formed by psychological constructs that have been derived from well-defined psychological theories and have been included in recent multiple pathway models: behavioral inhibition, delay aversion, temporal processing, and sustained attention (de Zeeuw et al., 2012; Sonuga-Barke et al., 2010; see Sections 1.1.1. – 1.1.6.). Referring to the endophenotypes approach, genetic and neuroscientific factors will be connected to these psychological constructs, thereby evaluating their validity as endophenotypes for ADHD.

Relying on previous work by Crosbie and colleagues (2008), Figure 1 provides a graphical illustration for the proposed framework. The first layer constitutes a genetic layer that containing various genes, which are involved in the multigenetic development of ADHD (Banaschewski et al., 2010; Faraone et al., 2005; Gizer et al., 2009). However, genes do not have a direct impact on behavior (Robinson, Fernald, & Clayton, 2008). Instead, genes code for proteins, indicating how much of a protein is produced, as well as when and where in the brain a protein is expressed (Landis & Insel, 2008). Small differences in this protein synthesis can result in large differences in brain functions. Hence, genetic influences on ADHD cannot be understood without understanding how genes influence neuronal circuits (Landis & Insel, 2008). The second layer of the framework therefore consists of different neuronal structures and neuronal functions that in turn map on psychological constructs in the third layer: behavioral inhibition, delay aversion, temporal processing, and sustained attention. In accordance with multiple pathway models (de Zeeuw et al., 2012; Sonuga-Barke et al., 2010; see Section 1.1.5.), these psychological constructs are related to symptoms of ADHD (impulsivity, hyperactivity, and inattention) in the fourth layer.

However, according to the DSM-V (APA, 2013) the different symptoms of ADHD still represent a variety of different behaviors, as for instance, impulsivity includes behaviors as “blurts out answers” or “interrupts or intrudes” while inattention includes behaviors as “avoiding sustained effort” or “fails to give close attention to details”. Thus, the fourth layer of this framework is further divided into a higher-level that represents the ADHD symptom dimensions and a lower-level that represents different higher-order psychological functions or complex behaviors which can be considered as quantitative facets of the symptom dimensions. For instance, risky decision-making is widely regarded as a facet for impulsivity, albeit other facets of impulsivity exist (see Section 2.3.). However, as a facet of impulsivity, risky decision-making and other higher-order psychological functions are more homogeneous and less complex constructs than the complete symptom dimension. In addition, endophenotypes might not be related to all facets of the symptom alike. Therefore, associations between endophenotypes (e.g., behavioral inhibition or sustained attention) and specific facets of symptom dimensions (e.g., risky decision-making) should be higher than associations between endeophenotypes and the symptom dimensions in general.

Finally, ADHD is not a purely genetic or biological disorder and environmental factors can have an influence on each layer of the framework (Coghill, Nigg, Rothenberger, Sonuga-Barke, & Tannock, 2005). For instance, environmental factors can affect gene expression (Robinson et al., 2008), neuronal development (Lawson, Duda, Avants, Wu, & Farah, 2013), or cognitive performance (Farah et al., 2006). Similar to the research on stress (Pearlin, Menaghan, Lieberman, & Mullan, 1981), the framework distinguishes between chronically, long-term, situation unspecific environmental factors, and acute, short-term, situation specific environmental factors. While situation unspecific environmental factors might contribute more to the development of neuronal structures and cognitive functions, situation specific environmental factors might determine whether these neuro-cognitive deficits matter in a specific situation (Bäckman & Dixon, 1992).

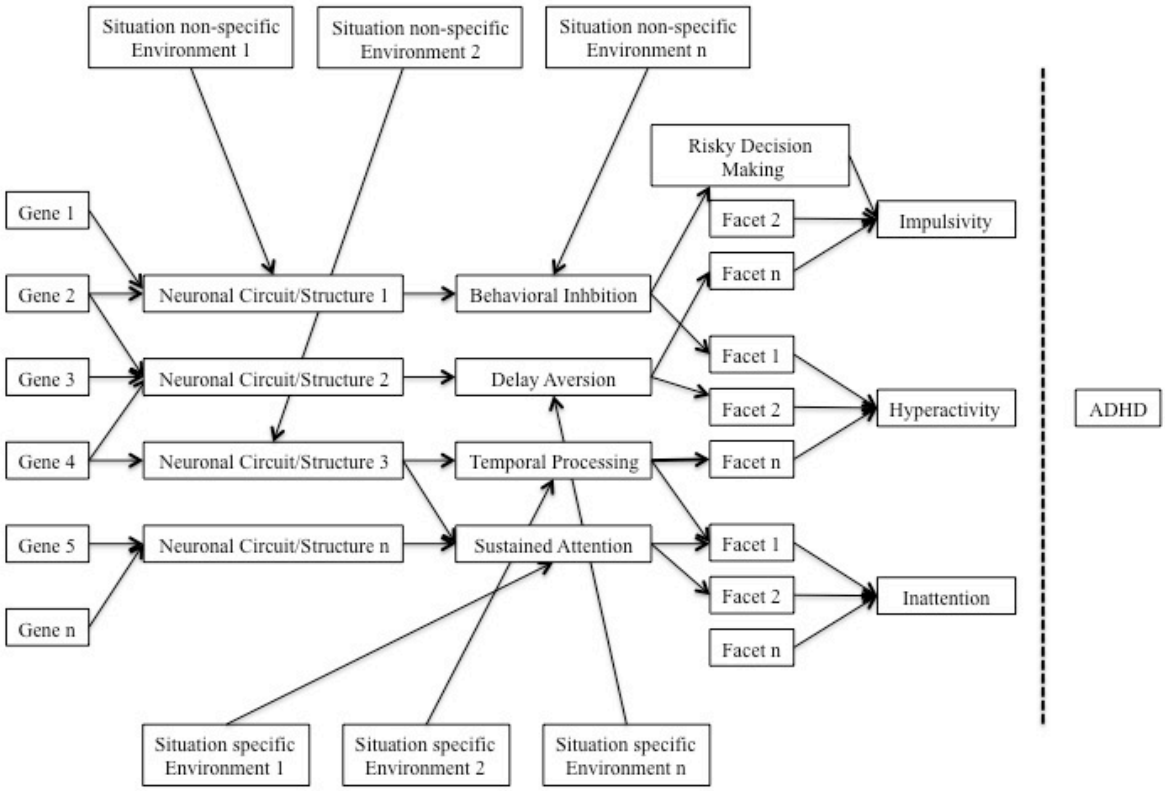

Figure 1. Proposed research framework to study ADHD. Multiple genes affect different neuronal circuits that in turn influence psychological functions. Psychological functions can be related to specific facets of the ADHD symptoms impulsivity, hyperactivity, and inattention. When the severity of these symptoms exceeds a threshold (dashed line), ADHD can be diagnosed. Environmental factors affect each stage of the model. Situation non-specific environmental factors primarily influence neuronal circuits and long-term developments of psychological functions whereas situation specific factors primarily influence whether deficits in psychological functions lead to symptoms of ADHD in specific situations. For a better readability the figure does not include connections within the layers (e.g., connections between neuronal circuits or psychological functions) although such connections are possible and likely.

The proposed framework is able to handle heterogeneity within ADHD as heterogeneity can emanate from individual differences on each layer or from different environmental demands. In addition, relying on the endophenotypes approach, neuroscience and genetics are incorporated into psychological theories of ADHD. In the following sections I will demonstrate how research from psychology, neuroscience, and genetics can be related within the proposed framework by exemplarily reviewing behavioral, neuroscientific, genetic, and environmental associations of behavioral inhibition and sustained attention with ADHD.

In line with the criteria for endophenotypes (Crosbie et al., 2008; Doyle et al., 2005; Hasler et al., 2006; Kendler & Neale, 2010; see Section 1.2.), I will give a definition of behavioral inhibition and sustained attention, describe the measures by which they are assessed, report the reliability of these measures, and review behavioral evidences for a correlation of behavioral inhibition and sustained attention with ADHD. Subsequently, to underline the biological plausibility of behavioral inhibition and sustained attention as endophenotypes, I will report their neural underpinnings and their neural associations with ADHD. Third, I will examine genetic associations of behavioral inhibition and sustained attention in relation to ADHD. Finally, to account for additional environmental influences, I will review how situation non-specific and situation specific environmental factors are related to each of the two psychological constructs and ADHD (see Sections 2.1. – 2.4.). Moving beyond endophenotypes I will finally review evidence for enhanced risky decision-making in ADHD, as risky decision-making is considered a lower-level facet of impulsivity (Whiteside & Lynam, 2001), thereby emphasizing the notion of smaller well-defined, quantitative behavioral outcomes instead of a categorical diagnosis (Cuthbert, 2005; Hyman, 2010; Morris & Cuthbert, 2012; see Coghill & Sonuga-Barke, 2012 for a discussion in ADHD).

2.1. Behavioral Inhibition

Behavioral inhibition can be defined as the ability to inhibit or stop a dominant response to an event in order to avoid inappropriate or unwanted responding (Renison, Ponsford, Testa, Richardson, & Brownfield, 2012). However, behavioral inhibition is not a homogeneous construct as the definition already distinguishes two different inhibitory processes: inhibiting a response *before* it is initialized and stopping an *ongoing* response. Indeed, theoretical approaches (Barkley, 1997) and latent variables approaches (Friedman & Miyake, 2004; Huizinga, Dolan, & van der Molen, 2006), investigating the nature of behavioral inhibition, identified even more different inhibitory processes, namely (a)

withholding of responses, (b) inhibition of already initialized responses, (c) interference control, and (d) resistance to distraction or disruption by competing events. These inhibitory processes differ in their timing of motor control processes on the one hand (Chambers, Garavan, & Bellgrove, 2009), and attentional control processes on the other hand (Awh, Belopolsky, & Theeuwes, 2012). Both, withholding of responses and inhibition of already initialized responses, refer to motor control, although their timing is different. While withholding of responses requires a person to inhibit a response before its execution has started, inhibition of an already initialized response requires a person to terminate a response after it has started, but before it has finished (Barkley, 1997). In contrast, interference control and resistance to distraction refer to attentional control processes. Both processes deal with distractors that have to be inhibited in order to perform a task. However, in interference control tasks the distractors are part of the task and activate conflicting responses (Botvinick, Braver, Barch, Carter, & Cohen, 2001), whereas in resistance to distraction tasks the distractors can be any kind of external distraction or interruption. Consequently, a large number of different tasks have been used to measure resistance to distraction. Distractors include words presented in the unattended channel of a dichotic listening task (Prior, Sanson, Freethy, & Geffen, 1985), comic figures in a computerized letter search task (Forster & Lavie, 2008), or movie excerpts while working on a math task (Gawrilow, Gollwitzer, & Oettingen, 2011, Study 2). However, as these tasks are very different and have only been rarely used in ADHD, the following review on behavioral inhibition only includes withholding of responses, inhibition of already initialized responses, and interference control.

However, cognitive theories (e.g., Botvinick et al., 2001) argue that behavioral measures of behavioral inhibition are confounded by tow underlying processes. To successfully inhibit a response (e.g., to adapt to response conflict), it is a prerequisite to first identify that one is in a situation in which inhibition is required (e.g., conflict-monitoring). Only afterwards can a behavior be inhibited. Therefore, the relation of behavioral inhibition and conflict-monitoring is discussed.

2.1.1. Behavioral Performance

This section reports on the behavioral performance of children with and without ADHD on several measures of behavioral inhibition and conflict-monitoring that can be derived from cognitige tasks. An overview of the tasks and measures reviewed in this section, and whether high or low values of the measure are associated with a deficit in behavioral inhibition or conflict-monitoring is displayed in Table 1.

Withholding of responses. A common paradigm to measure withholding of responses is the Go/No-Go task (Donders, 1868/1969; Verbruggen & Logan, 2008a). Within the Go/No-Go task participants are presented with a series of stimuli and instructed to respond whenever a Go stimulus is presented, but to withhold their response if a No-Go stimulus is presented (e.g., to press a response key every time a picture of an animal is presented but not to press the response key if the animal on the picture is a goose). The mapping of stimuli onto Go and No-Go responses is arbitrary and explained to participants in the beginning of the experiment. In the Go/No-Go task, behavioral inhibition, as defined by withholding of responses, is assessed by the probability of responding on No-Go trials also referred to as the proportion of inhibition errors a participant made throughout the experiment (Rauch, Gold, & Schmitt, 2012; Verbruggen & Logan, 2008a). Recently, the factorial structure, construct validity, and test-retest reliability of a parametric Go/No-Go task have been estimated, revealing that the amount of inhibition errors loads on a different factor than the attention related aspects of the task (e.g., the percentage of correct hits to targets), albeit showing only a modest test-retest reliability (r = .63; Langenecker, Zubieta, Young, Akil, & Nielson, 2007; Votruba & Langenecker, 2013). However, the differentiation of inhibition errors from other cognitive aspects of the task, like response times or response time variability (RTV), is in line with other factor analytical results, indicating the construct validity of the measure (De Zeeuw, Weusten et al., 2012).

As proposed, several studies observed that children with ADHD commit more inhibition errors than unaffected children (de Zeeuw et al., 2012; Gawrilow & Gollwitzer, 2008; Rauch et al., 2012; but see Smith, Taylor, Brammer, Toone, & Rubia, 2006). This deficit in withholding of responses in children with ADHD was maintained even after manipulations of motivation (rewarding fast answers by winning points and punishing inhibition errors by loosing points) or working memory load had been applyed. Manipulations of motivation or working memory load did not affect children with and without ADHD differentially (Wodka et al., 2007).

Inhibition of already initialized responses. A common paradigm to measure the inhibition of already initialized responses is the Stop-Signal task (Lappin & Eriksen, 1966; Logan & Cowan, 1984). In a Stop-Signal task participants usually perform a visual choice reaction time task (e.g., press the left response key for the letter A and the right response key for the letter B). On random occasions a stop signal (e.g., a tone) is presented, ordering the participants to inhibit their response on that trial. The tacit assumption of the task states that two independent processes determine whether the response on a stop trial is executed or not. If the response process related to the visual stimuli finishes before the stop process related to the stop signal, the response is executed. However, if the stop process finishes before the response process, the response is inhibited. Which of the two processes wins the race depends on the time interval between the onset of the visual stimulus and the presentation of the stop signal. If the stop signal occurs early enough, the response will be inhibited. But if the stop signal occurs too late, the response will be executed (Overtoom et al., 2002; Verbruggen & Logan, 2008a, 2008b). In the Stop-Signal task, behavioral inhibition, as defined by the inhibition of already initialized responses, is assessed by two indices: the probability of responding on stop signal trials and an estimate of the latency of the stop process (stop signal reaction time; SSRT; Verbruggen & Logan, 2008a, 2008b). Simulation studies (Band, van der Molen, & Logan, 2003) and split-half methods (Logan, Schachar, & Tannock, 1997; Williams, Ponesse, Schachar, Logan, & Tannock, 1999) showed a high reliability ($r > .83$) for the SSRT measure for participants older than eight years. However, if response time distributions are highly skewed, some commonly used methods for the estimation of the SSRT tend to overestimate the SSRT (Verbruggen, Chambers, & Logan, 2013).

As proposed, several studies observed a higher SSRT for children with ADHD compared to unaffected children (Dimoska, Johnstone, Barry, & Clarke, 2003; Geurts, Verté, Oosterlaan, Roeyers, & Sergeant, 2004; Overtoom et al., 2002; Pliszka, Borcherding, Spratley, Leon, & Irick, 1997; but see Kuntsi, Oosterlaan, & Stevenson, 2001). Indeed, according to meta-analyses a prolonged SSRT for children with ADHD was found in more than 80% of all studies using the Stop-Signal task (Willcutt et al., 2005), leading to a mean effect size of $d = .61$. An even higher mean effect size of $d = .79$ was found in adults showing that a deficient inhibition of already initialized responses continues in adults with ADHD (Lijffijt, Kenemans, Verbaten, & van Engeland, 2005).

Interference control. Common paradigms to measure interference control are the Stroop task (Stroop, 1935) and the Flanker task (Eriksen & Eriksen, 1974). In the Stroop task participants are presented with a series of color words printed in various ink colors. For each color word participants are asked to name the color of the ink rather than the color of the word. Within congruent trials the name of the color word and its ink color match (e.g., the word 'blue' written in blue), while in incongruent trials the name of the color word and its ink color do not match (e.g., the word 'blue' written in red). According to parallel distributed processing models, incongruent trials lead to interference by simultaneous activation of incompatible and competing representations explaining longer response times and more errors on incongruent than on congruent trials (Botvinick, Cohen, & Carter, 2004; Cohen, Dunbar, & McClelland, 1990; MacLeod, 1991). In the Stroop task, behavioral inhibition, as defined by interference control, can be assessed by several indices depending on different variants of the Stroop task. When a fixed time interval is used, interference scores are composed by the difference in the amount of words read in the congruent condition and the amount of words read in the incongruent condition. Accordingly, when a fixed amount of trials is used, interference scores are composed by the difference in the response times to congruent and incongruent word-ink pairs. For some variants of the Stroop task errors are analyzed as well, and interference scores for errors are composed in the same manner as interference scores for response times (Homack & Riccio, 2004). Other ways to assess interference control include ratio scores and analysis of covariance (Lansbergen, Kenemans, & van Engeland, 2007). Reliability for interference scores has not been demonstrated so far. However, the reliability of the amount of words read in the different conditions for parallel test forms of the Stroop is rather good (r = .82; Sacks, Clark, Pols, & Geffen, 1991; see Homack & Riccio, 2004 for further discussion).

As proposed, several studies observed a higher interference effect for individuals with ADHD compared to unaffected individuals in the Stroop task (Ikeda, Okuzumi, & Kokubun, 2013; MacLeod & Prior, 1996; Scheres et al., 2004; Seidman, Biederman, Valera, Monuteaux, Doyle, & Faraone, 2006; but see Goldberg et al., 2005). This deficit in interference control held after controlling for intelligence (Seidman, Biederman et al., 2006) but might disappear after controlling for general response speed (Soutschek et al., 2013). The effect size for interference control further depends on the method used to calculate the inference score with ratio scores showing the most consistent results (Lansbergen et al., 2007).

In the Flanker task, participants have to respond to a central target but to ignore flanking distractors. Traditionally, targets and flanking distractors have been arrows either pointing to the left or right direction. On congruent trials, both, the target and the flanking distractors, point in the same direction (e.g., >>>>>) whereas on incongruent trials the target and the flanking distractors point at opposite directions (e.g., >><>>). According to parallel distributed processing models, incongruent trials lead to interference by the simultaneous activation of incompatible and competing representations, explaining longer response times and more errors on incongruent than on congruent trials (Botvinick et al., 2004; Cohen, Servan-Schreiber, & McClelland, 1992). In the Flanker task, behavioral inhibition, as defined by interference control, can be assessed by the difference in response times on congruent and incongruent trials (Flanker interference effect for response times) and by the difference in error rates on congruent and incongruent trials (Flanker interference effect for errors), respectively. Although internal consistencies and test-retest reliabilities for response times on congruent and incongruent trials are high, the difference measure to assess interference control only shows a modest internal consistency ($\alpha = .68$) and a modest test-retest reliability ($r = .53$; Wöstmann et al., 2013). A reduced reliability is generally found in difference scores and constitutes an inherent problem of these measures (Edwards, 2001). However, the modest reliability values are in line with reliability assessments found for other neurocognitive measures, like processing speed, visual memory, or verbal memory (Schatz, 2010).

As proposed, several studies observed a higher interference effect for children with ADHD compared to unaffected children in the Flanker task (Crone, Jennings, & van der Molen, 2003; Konrad, Neufang, Hanisch, Fink, & Herpertz-Dahlmann, 2006; but see Booth, Carlson, & Tucker, 2007). Moreover, a recent review on interference control in the flanker task concluded a weaker interference control in children with ADHD compared to unaffected children, although only a trend was found in the majority of the studies (Mullane, Corkum, Klein, & McLaughlin, 2009).

Behavioral inhibition and conflict-monitoring theory. Research focusing on behavioral inhibition faces an important limitation: The sole focus relies on the exertion of inhibition but no importance is given to how inhibition itself is brought up (see Botvinick et al., 2001, 2004). How does a person know when a situation requires inhibiting a response? The conflict-monitoring theory (Botvinick et al., 2001, 2004) proposes a solution to this problem by postulating a conflict-monitoring unit. This unit detects when a response conflict is present, for instance, when in an incongruent Flanker trial both the target and the flanker stimuli activate different and contradictory responses. The conflict-monitoring unit measures

the conflict strength and subsequently signals the need for inhibition or other forms of cognitive control to units that regulate conflict adaptation, as for instance by exerting behavioral inhibition (Botvinick et al., 2001, 2004).

Thus, a behaviorally observed deficit in behavioral inhibition, for instance in interference control, can either be due to a deficit in conflict-monitoring (e.g., a deficit in identifying situations in which inhibition as needed) or a deficit in conflict adaptation (e.g., a deficit inhibiting a response). Behavioral markers indicating conflict-monitoring include a slowing down in response times after errors (post-error slowing, PES; Rabbitt, 1966), a slowing down in response times in trials after incongruent trials (post-conflict slowing, PCS; Verguts, Notebaert, Kunde, & Wühr, 2011), or a reduced interference effect after incongruent trials (sequential congruency effect; Gratton, Coles, & Donchin, 1992; Wendt, Luna-Rodriguez, & Jacobsen, 2012). Sequential congruency effects have mainly been observed for response times, but they also emerge for errors (Botvinick et al., 2001; Gratton et al., 1992; Wendt et al., 2012). For instance, in a Flanker task, participants respond faster and more accurate to incongruent stimuli, when the preceding trial was incongruent as well. In similar, they respond faster and more accurate to congruent trials, when the preceding trial was congruent as well. According to the conflict-monitoring theory, this effect is explained by incongruent trials causing a response conflict. This conflict is measured in the conflict-monitroring unit and forwarded to an attentional control unit. In the subsequent trial the attentional focus is narrowed to the center of the screen leading to a better processing of the central target stimulus and less processing of the Flanker stimuli. Thus, if the subsequent trial is incongruent the performance is less deteriorated by processing the incongruent Flankers, however, if the subsequent trial is congruent the performance is less enhanced by processing the congruent Flankers (Botvinick et al., 2001, 2004). However, no assessments of reliability have been reported for sequential congruency effects, PES or PCS.

Comparing participants with and without ADHD, several studies suggested a deficient conflict-monitoring in ADHD as evinced in a less post-error slowing (Gupta & Kar, 2009; Schachar et al., 2004; Wiersema, van der Meere, & Roeyers, 2005), while other studies failed to reproduce these results (e.g., Cao et al., 2013; Jonkman, van Melis, Kemner, & Markus, 2007; see Shiels & Hawk Jr., 2010 for an overview). However, as deficits in conflict-monitoring and deficits in behavioral inhibition have not been related in ADHD so far, it remains unclear, whether conflict-monitoring, behavioral inhibition or both processes are impaired in ADHD.

Table 1

Overview of Tasks and Performance Measures for Behavioral Inhibition, Conflict-Monitoring, and Sustained Attention

Psychological Function	Tasks	Measure of Performance	Indicator for Deficit[a]
Behavioral inhibition			
Withholding of responses	Go/No-Go	Number of inhibition errors	high
Inhibition of already initialized responses	Stop-Signal	Number of inhibition errors	high
		SSRT	large
Interference control	Flanker	Interference effect: errors	large
	Stroop	Interference effect: response times	large
Conflict-monitoring			
	Flanker/Stroop	Post-error slowing	small
		Post-conflict slowing	small
		Sequential congruency effect: RT	small
		Sequential congruency effect: errors	small
Sustained attention			
	CPT	Number of omission errors[b]	high
		Response time variability[b]	large
		Index of signal sensitivity d'[c]	low
		Drift rate v[c]	small

Note. [a] displays which values of the performance measure indicate a deficit on the psychological function; [b] can be derived from any cognitive task; [c] can be derived from any cognitive task with two response alternatives; CPT = Continuous Performance Task; RT = response time.

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Summary. Taken together, there is evidence for a deficit in behavioral inhibition in children with ADHD with regard to withholding of responses, inhibition of already initialized responses, and interference control. Reliabilities for measures from different cognitive tasks are modest or satisfactory. However, it is possible that the reported deficits in behavioral inhibition in children with ADHD might reflect deficits in conflict-monitoring rather than in behavioral inhibition per se.

2.1.2. Neuronal Correlates

The following section reviews neuronal correlates for behavioral inhibition that have been derived from electrophysiology and imaging, for instance, functional magnetic resonance imaging (fMRI), structural magnetic resonance imaging (sMRI), resting state fMRI and diffusion tensor imaging (DTI).

Electrophysiology. Electrophysiological studies on Go/No-Go and Stop-Signal tasks identified two event related potential (ERP) markers for No-Go, compared to Go trials, and for Stop trials, compared to Go trials, respectively: the frontal-midline N200, a negative shift approximately 200-300 ms after stimulus onset (N200) and the anterior P300, a positive shift that peaks between 300 and 600 ms after stimulus onset, together referred to as the N2/P3 complex (Bokura, Yamaguchi, & Kobayashi, 2001; van Boxtel, van der Molen, Jennings, & Brunia, 2001; see Huster, Enriquez-Geppert, Lavallee, Falkenstein, & Herrmann, 2013 for a review). Both, the N200 and the P300, have generally been related to behavioral inhibition (e.g., van Boxel et al., 2001; Wild-Wall, Oades, Schmidt-Wessels, Christiansen, & Falkenstein, 2009). However, as the P300 is larger after successful inhibition trials, it might rather be an indicator of behavioral inhibition efficiency, than an indicator of behavioral inhibition per se (Liotti, Pliszka, Perez, Kothmann, & Woldorff, 2005). Finally, the N200 but not the P300 have been reported in tasks on interference control, for instance in the Flanker and in the Simon task (Huster et al., 2013; Nigbur, Ivanova, & Stürmer, 2011), indicating that withholding of responses, inhibition of already initialized responses and interference control share common and individual neural mechanisms.

In line with the behavioral results, several studies observed a reduction of the N200 amplitude in children with ADHD compared to unaffected children (Dimoska et al., 2003; Johnstone, Barry, Markovska, Dimoska, & Clarke, 2009; Liotti et al., 2005; Pliszka, Liotti, & Woldorff, 2000; Wild-Wall et al., 2009; but see Cao et al., 2013; Fallgatter et al., 2004) as well as a reduction of the P300 amplitude (Liotti et al., 2005; Paul et al., 2007; Paul-Jordanov,

Bechthold, & Gawrilow, 2010; Wild-Wall et al., 2009; but see Dimoska et al., 2003; Johnstone et al., 2009). However, as with the behavioral results, it has been argued that the N2/P3 complex does not reflect behavioral inhibition but rather conflict-monitoring (Donkers & van Boxtel, 2004; Smith, Smith, Provost, & Heathcote, 2010). This has been underscored both by a parallel distributed processing model of conflict-monitoring and empirical results revealing an enhanced N200 amplitude in incongruent trials and a negative ERP subsequent to errors (error related negativity, ERN) in a Flanker task (Yeung, Botvinick, & Cohen, 2004). Indeed, in addition to a reduced N200 several studies revealed a reduced ERN in children with ADHD compared to unaffected children (Albrecht et al., 2008; Liotti et al., 2005; but see Wiersema et al., 2005). These results were further confirmed by a recent meta-analysis displaying a reduced ERN of medium size (d = .50) in juvenile and adult ADHD patients (Geburek, Rist, Gediga, Stroux, & Pedersen, 2013). However, as with the behavioral data, there is still a debate on whether the N2/P3 complex reflects behavioral inhibition or conflict-monitoring.

Imaging. Within the human brain, the prefrontal cortex (PFC) has long been regarded as the key structure for cognitive control and especially for behavioral inhibition (Miller & Cohen, 2001). Studies using fMRI to measure neural correlates of Go/No-Go and Stop-Signal tasks revealed the involvement of fronto-basal ganglia circuits in behavioral inhibition (Aron 2011; Chambers et al., 2009). Accordingly, a fronto-basal ganglia model of behavioral inhibition was proposed (Chambers et al., 2009). In order to respond to Go stimuli responses are initialized in the supplementary motor area (SMA), the pre-supplementary motor area (pre-SMA) and other motor regions and sent to the subthalamic nucleus to suppress all other motor programs. Subsequently, the selected response is released and terminated via fronto-basal ganglia pathways. On a No-Go or Stop trial processing follows the identical steps until the Stop stimulus is processed. Then an additional reset signal is sent from the inferior frontal gyrus (IFG) to the subthalamic nucleus that cancels the initialized response (for details see Chambers et al., 2009). Following this model, most neuroimaging studies on behavioral inhibition focused either on the pre-SMA or the IFG as the neural source of inhibition (Aron 2011; Aron & Poldrack, 2005; Lenartowicz, Verbruggen, Logan, & Poldrack, 2011).

Ontological concepts of cognitive control assume that different tasks measuring behavioral inhibition should activate task specific neural networks as well as a common key neuronal network structure reflecting pure inhibition (Lenartowicz, Kalar, Congdon, & Poldrack, 2010). However, a meta-analysis, comparing neural network activity between Go/No-Go and Stop-Signal paradigms (Swick, Ashley, & Turken, 2011), did not support the

notion of the IFG being crucial for response inhibition. Instead, the authors found the anterior insula to reflect a general form of response inhibition. These findings are in line with other meta-analyses (Criaud & Boulinguez, 2013; Simmonds, Pekar, & Mostofsky, 2008), concluding that activation of the IFG might be task dependent and reflect attentional processes or working memory processes. Moreover, when neural activity in various behavioral inhibition tasks, including a Go/No-Go and a Flanker task, was directly compared (Wager et al., 2005) a different inhibitory network emerged which is composed of the anterior insula, the anterior prefrontal cortex, and the anterior cingulate cortex (ACC). However, especially the ACC has been regarded as the key structure of conflict-monitoring (Botvinick et al., 2004; Egner & Hirsch, 2005), leaving it unclear whether the neural structures involved in behavioral inhibition tasks are solely responsible for behavioral inhibition.

Regardless of their exact function during behavioral inhibition tasks, a meta-analysis (Dickstein, Bannon, Castellanos, & Milham, 2006) and several studies using fMRI revealed a reduced activity in the pre-SMA (Suskauer et al., 2008; Tamm, Menon, Ringel, & Reiss, 2004), the IFG (Booth et al., 2005; Ma et al., 2012; Rubia, Smith, Brammer, Toone, & Taylor, 2005), the caudate nucleus, putamen, or globus pallidus (Booth et al., 2005; Teicher et al., 2000), and the ACC (Mulder et al., 2008; Pliszka et al., 2006; Tamm et al., 2004) in children with ADHD compared to unaffected children.

Theses results have further been confirmed by studies investigating the neuronal structure in children with and without ADHD using sMRI: Children with ADHD displayed a reduction of volume and cortical thickness of the PFC, in particular of the IFG (Batty et al., 2010; Depue, Burgess, Bidwell, Willcutt, & Banich, 2010; Durston et al., 2004; Kates et al., 2002; Sowell et al., 2003), of the pre-SMA (Mostofsky, Cooper, Kates, Denckla, & Kaufmann, 2002), of basal ganglia areas including the caudate nucleus and globus pallidus (Aylward et al., 1996; Castellanos et al., 2002; Castellanos et al., 2003; Valera, Faraone, Murray, & Seidman, 2007), and of the ACC (Bledsoe, Semrud-Clikeman, & Pliszka, 2013; Seidman, Valera et al., 2006). However, the structural differences in the caudate nucleus and globus pallidus might disappear in adulthood (Castellanos et al., 2002).

In addition, children with ADHD showed less structural connectivity within the pre-SMA (Ashtari et al., 2005) and between frontal and striatal brain regions (de Zeeuw, Mandl, Hulshoff Pol, van Engeland, & Durston, 2012) as measured by fractional anisotropy (FA), a quantitative measure of the homogeneity of the white matter microstructural organization that was derived from DTI. Furthermore, FA in right prefrontal fibers correlated with reduced IFG activity in children with ADHD but not in unaffected children (Casey et al., 2007). These

results match results from functional brain network connectivity studies during tasks that revealed less connectivity in fronto-basal ganglia networks (Cubillo, Halari, Ecker, Giampietro, Taylor, & Rubia, 2010; Konrad & Eickhoff, 2010).

Taken together, there is evidence for a behavioral inhibition related impairment of fronto-basal ganglia circuits in children with ADHD from a functional, structural, and connectivity perspective.

2.1.3. Genetic Associations

Family-genetic studies on behavioral inhibition in children with ADHD revealed an estimated heritability of 24% for the SSRT (Rommelse, Arias-Vásquez et al., 2008). In families with at least one child with a diagnosed ADHD and at least one additional sibling, a significant sibling correlation was observed for the SSRT that was larger than the sibling correlation for ADHD symptoms (Rommelse, Altink, Martin, Buschgens, Buitelaar et al., 2008). In addition, children with ADHD and poor inhibitiory performance (indicated by the SSRT) were found four times more likely to have a first-degree relative affected with ADHD than children with ADHD and good inhibitory performance (Crosbie & Schachar, 2001). Finally, unaffected siblings of children with ADHD showed poorer interference contol (indicated by Stroop interference) and exhibited more inhibition errors in a Go/No-Go task than unaffected control children (Slaats-Willemse, Swaab-Barneveld, de Sonneville, van der Meulen, & Buitelaar, 2003). Thus, behavioral genetic studies revealed heritybility and familial clustering of measures of behavioral inhibition in children with ADHD, thereby establishing the condition for molecular genetic studies and candidate gene studies in particular.

As the dopamine system is thought to be involved in the function of the PFC and especially in response inhibition (Arnsten, 2011; Colzato, van den Wildenberg, van Wouwe, Pannebakker, & Hommel, 2009; Hershey et al., 2004), molecular genetic studies using a candidate genes approach mainly focused on dopaminergic genes (for reviews see Barnes, Dean, Nandam, O'Connel, & Bellgrove, 2011 and Greene, Braet, Johnson, & Bellgrove, 2008). The dopamine D4 receptor has been of particular interest as it is most abundant in the PFC[1] (Oak, Oldenhof, & van Tol, 2000). Thus, the dopamine D4 receptor gene (DRD4) has been investigated in relation to behavioral inhibition. While the 4-repeat allele is the most frequent polymorphism of the DRD4 gene, the 7-repeat allele has been related to behavioral

[1] apart from the retina (Oak et al., 2000)

inhibition. However, results are mixed with studies reporting either a higher response inhibition (Krämer et al., 2009) or a lower response inhibition (Congdon, Lesch, & Canli, 2008) in carriers of the 7-repeat allele of the DRD4 gene as compared to carriers of the 4-repeat allele.

Due to the involvement of dopamine in response inhibition the dopamine transporter gene (DAT1) has been considered an additional candidate gene. As with polymorphisms in the DRD4 gene, there are mixed results with one study reporting a poorer response inhibition in carriers of the 10/10-genotype (carriers of two 10-repeat alleles) of the DAT1 gene compared to carriers of at least one 9-repeat allele (9/9 or 9/10 genotyoe of the DAT1; Cornish et al., 2005). However, another study reported a higher activation in the pre-SMA and the subthalamic nucleus of the basal ganglia during a Stop-Signal task for carriers of at least one 9-repeat allele compared to carriers of the 10/10-genotype but failed to detect differences in behavioral performance (Congdon, Constable, Lesch, & Canli, 2009). Interestingly, there might be a shift in the effect of the 10/10-genotype of the DAT1 gene on response inhibition from successful inhibition in childhood to less efficient inhibition in adolescence and adulthood (Brocki, Clerkin, Guise, Fan, & Fossella, 2009). However, the reported studies relied on relative small sample sizes (N = 119, Cornish et al., 2005; N = 43, Congdon et al., 2009) and a recent study, using a larger sample size (N = 405), did neither find the 9-repeat alleles nor the 10/10-genotype to be associated with response inhibition in a Stop-Signal task (Cummins et al., 2012). Instead two different polymorphisms on the DAT1 gene have been connected to a diminished inhibitory performance (Cummins et al., 2012) revealing evidence for an association of polymorphisms in the DAT1 gene and response inhibition, although the concrete polymorphisms further need to be investigated.

Besides polymorphisms in the dopaminergic genes DRD4 and DAT1, a tryptophan hydroxylase-2 (TPH2) polymorphism coding for an enzyme for serotonin biosynthesis has been linked to response inhibition as measured by the SSRT (Stoltenberg et al., 2006). In addition, mixed results have been reported for polymorphisms in the monoamine oxidase-A gene (MAO-A) coding for an enzyme primarily involved in catecholamine and serotonin catabolism. While one study reported an association between polymorphisms in the MAO-A gene and neural activity in prefrontal areas (Passamonti et al., 2006), there was no association between polymorphisms in the MAO-A gene and behavioral inhibition as assessed by the SSRT (Passamonti et al., 2006; Stoltenberg et al., 2006).

Meta-anlyses (Gizer et al., 2009; Li, Sham, Owen, & He, 2006), and reviews (Albayrak, Friedel, Schimmelmann, Hinney, & Hebebrand, 2008; Banaschewski et al., 2010;

Faraone et al., 2005) of candidate gene studies consistently reported a higher risk for ADHD in carriers of the DRD4 7-repeat allele, for carriers of the DAT1 10/10-genotype, and different polymorphisms on the TPH2 gene. However, the results for the DAT1 10/10-genotype are less clear (see Li et al., 2006) and might only hold for participants with ADHD but without a comorbid conduct disorder (CD; Zhou et al., 2008). Furthermore, a recent study reported a dissociation between effects of the DRD4 7-repeat allele on ADHD and effects of the DAT1 10/10 genotype on ADHD: Using a Go/No-Go task, the DRD4 7-repeat allele was associated with indices of inattention, while the DAT1 10/10 genotype was associated with indices of response inhibition (Gizer & Waldman, 2012). In addition, a recent EEG study revealed a reduced P300 during a Go/No-Go task both in adults with and without ADHD carrying a risk variant of the TPH2 gene. However, behavioral effects of the TPH2 risk variant were only found on omission errors and RTV and might thus reflect deficits in sustained attention and not behavioral inhibition (Baehne et al., 2009). In contrast to polymorphisms in the DRD4, DAT1, and TPH2 genes, reports on the involvement of polymorphisms in MAO-A in ADHD are inconsistent (cf. Banaschewski et al., 2010) and a recent study investigating the relationship of MAO-A polymorphisms and various neuropsychological functions revealed no associations between MAO-A polymorphisms and response inhibition tasks (Rommelse, Altink, Arias-Vásquez et al., 2008).

Taken together, there is evidence for heritability of behavioral inhibition from behavioral genetics studies as well as molecular genetics studies. Partcularly, polymorphisms in dopaminergic genes as the DRD4 and the DAT1 gene might be involved in the behavioral inhibition deficit in children with ADHD.

2.1.4. Environmental Effects

Regarding behavioral inhibition, neither situation non-specific factors nor situation specific factors have been systematically investigated. Situation non-specific factors mainly focused on prenatal factors, parenting, and physical activity whereas situation specific factors included stimulus properties and motivation.

Situation non-specific factors

This section reports on prenatal alcohol and tobacco exposure, SES, parenting, and physical activity. For each environmental factor first associations are reported with measures of behavioral inhibition. Second, associations with the disorder of ADHD are described and finally evidence is reviewed for a mediation of these effects through behavioral inhibition.

Prenatal alcohol and tobacco exposure. Prenatal alcohol and tobacco exposure has been related to decreased cognitive functioning including learning, memory behavioral inhibition, and intelligence (see Huizink & Mulder, 2006 for a review). For instance, children with heavy prenatal exposure to alcohol or fetal alcohol spectrum disorder exhibited higher interference scores in a Stroop task (Mattson, Goodman, Caine, Delis, & Riley, 1999) and committed more errors in an Anti-Saccade task (Paolozza et al., 2014). However, several studies did not report any behavioral differences between children prenatally exposed to alcohol and control children in a Stroop task (Richardson, Ryan, Willford, Day, & Goldschmidt, 2002) or in a Go/No-Go task (Fryer et al., 2007; O'Brien et al., 2013).

In similar, mixed results have been reported for an association between prenatal tobacco exposure and behavioral inhibition. While one study reported an association of prenatal tobacco exposure during the first trimester of pregnancy with higher interference scores on a Stroop task in 10-year-old children (Cornelius et al., 2011), other studies did not find any differences in Stroop interference scores or in the amount of inhibition errors or post-error-slowing in an inhibition of prepotent responses task between adolescents prenatally exposed to tobacco and a control group (Fried, Watkinson, & Gray, 2003; Huijbregts, Warren, de Sonneville, & Swaab-Barneveld, 2008).

With regard to ADHAD, prenatal alcohol exposure and prenatal tobacco exposure have been considered as environmental risk factors (Braun, Kahn, Froehlich, Auinger, & Lanphear, 2006; Linnet et al., 2003; Pineda et al., 2007; see Nigg et al., 2010 for an overview) that increase the likelihood of a later ADHD diagnosis by odds ratios of OR = 14.8 for prenatal alcohol exposure and OR = 10.5 for prenatal tobacco exposure (Pineda et al., 2007). However, only one study examined the effects of prenatal alcohol exposure on behavioral inhibition in young adults with ADHD (Burden et al., 2010). While young adults with ADHD made more inhibition errors in a Go/No-Go task than young adults without ADHD, no interaction with prenatal alcohol exposure was found. Yet, prental alcohol exposure influenced the No-Go P300 amplitude. Participants with ADHD but without prenatal alcohol exposure showed a smaller No-Go P300 amplitude compared to participants with ADHD and prenatal alcohol exposure or participants without ADHD (Burden et al., 2010) indicating

differences in the etiology of a behavioral inhibition deficit in adults with ADHD and prenatal alcohol exposure on the one hand and adults with an idiopathic ADHD on the other hand.

Socioeconomic status. In addition to prenatal risk factors a low socioeconomic status (SES) has been identified as one of the most severe post-natal risk factors for the development of psychopathology (Flouri, Mavroveli, & Tzavidis, 2010). SES has been conceptualized as either representing social class in terms of economic position, as social status in terms of social prestige, or as general capital in terms of material resources, educational resources, and social connections (Bradley & Corwyn, 2002). Therefore, most measures of SES therefore consist of quantifications of family income, parental education, and occupational status (Bradley & Corwyn, 2002). SES has consistently been found to correlate with behavioral inhibition and other executive functions (Ardila, Rosselli, Matute, & Guajardo, 2005; Mezzacappa, 2004; Sarsour et al., 2011). For instance, relative to children with a high SES, children with a low SES were impaired in behavioral inhibition tasks like the Stroop task and the Go/No-Go task (Mezzacappa, 2004; Sarsour et al., 2011; Wiebe et al., 2011). Furthermore, a recent study revealed an association between children's SES and the thickness of prefrontal cortical areas, especially in areas related to behavioral inhibition and conflict-monitoring as the ACC (Lawson et al., 2013).

However, only few studies investigated the relation between SES and ADHD. Children from a lower SES were at higher risk for an ADHD diagnosis (Biederman, Faraone, Monuteaux, 2002; Pineda et al., 1999). However, the association disappeared after controlling for parental ADHD and marital conflict (Counts, Nigg, Stawicki, Rappley, & von Eye, 2005) and might be driven by a relationship between ADHD and school problems or learning disabilities that are both predicted by low SES (Biederman et al., 2002). In addition, only one study investigated associations of ADHD, SES, and behavioral inhibition in a population-based sample of preschoolers (Martel, 2013). Preschoolers from low-income families showed an increase in ADHD symptoms and a deficit in response inhibition compared to preschoolers from high-income families. However, no association was found between SES and behavioral inhibition (Martel, 2013).

Parenting. Most research investigating the effects of parenting on behavioral inhibition has been conducted in toddlers (Blair et al., 2011, Hughes & Ensor, 2009) or preschool children (Blair, Raver, & Berry, 2014; Hammond, Müller, Carpendale, Bibok, & Liebermann-Finestone, 2012; Olson et al. 2011), whereas only few studies have been conducted with school children (Hewage, Bohlin, Wijewardena, & Lindmark, 2011; Schroeder & Kelley, 2010). While various dimensions of parenting have been related to

behavioral inhibition, different studies disagree whether positive parenting (e.g., maternal sensitivity, maternal attachment, expression of positive feelings towards the child) fosters behavioral inhibition or whether negative parenting (e.g., intrusiveness, harsh discipline, expression of negative feelings towards the child, inconsistent parenting) harms behavioral inhibition (Blair et al., 2011, 2014; Hammond et al., 2012; Hewage et al., 2011; Hughes & Ensor, 2009; Olson et al., 2011).

Studies investigating associations of parenting and ADHD yielded mixed results. For instance, harsh or inconsistent discipline, conflicted parent-child interactions, parental overreactivity, and disorganized attachment have been related to ADHD (DuPaul, McGoey, Eckert, & van Brankle, 2001; Ellis & Nigg, 2009; Harold et al., 2013; Harvey, Danforth, Ulaszek, & Eberhardt, 2001; Lifford, Harold, & Thapar, 2009; Thorell, Rydell, & Bohlin, 2012; but see Burke, Pardine, & Loeber, 2008). However, these relations might only hold for children with ADHD and a comorbid CD or a comorbid oppositional defiant disorder (ODD; Harvey et al., 2001; Seipp & Johnston, 2005; for reviews see Deault, 2010 and Johnston & Mash, 2001). In addition, it is unclear whether harsh discipline or conflicted parent-child interactions contribute to ADHD symptoms or whether they are a reaction to the child's symptoms (Lifford, Harold, & Thapar, 2008, 2009). Moreover, parents of children with ADHD often suffer from an ADHD themselves (cf. Minde et al., 2003). It could be shown, that mothers of children with ADHD who had ADHD themselves were poorer in monitoring their child's behavior and more inconsistent disciplinarians compared to mothers of children without ADHD (Murray & Johnston, 2006; see Johnston, Mash, Miller, & Ninowski, 2012 for a review).

However, most studies investigated parenting only in relation to ADHD and not in relation to deficits in behavioral inhibition and ADHD. One study that combined ADHD, CD, ODD to a measure of externalizing problems reported a partial mediation of the effects of negative parenting on externalizing problems by behavioral inhibition (Hewage et al., 2011), but another study failed to replicate such a mediation (Olson et al., 2011). Another study examined attachment in relation to behavioral inhibition in ADHD, but found no correlation between the two factors (Thorell et al., 2012).

Physical activity. Within the last decade, research intensively investigated the effects of physical activity on behavioral inhibition and other cognitive functions (see Hillman, Erickson, & Kramer, 2008 and Hillman, Kamijo, & Scudder, 2011 for reviews). For instance, children with a higher aerobic fitness showed better interference control in a paper-pencil Stroop task (Buck, Hillman, & Castelli, 2008) and in a modified Flanker task (Pontifex et al.,

2011). In addition, children with a higher aerobic fitness exhibited a larger P300 but a lower ERN than children with a lower aerobic fitness (Hillman, Buck, Themanson, Pontifex, & Castelli, 2009; Pontifex et al., 2011) and a recent fMRI study revealed a stronger activation in a cluster of areas including the ACC during congruent trials in high-fit children compared to low-fit children (Voss et al., 2011). Thus, the neuronal results confirmed en enhanced behavioral inhibition in high-fit children that might be accompanied by a more effective conflict-monitoring.

Interest in the effects of physical activity interventions on ADHD has grown over the last few years (Berwid & Halperin, 2012) and two recent studies reported an improvement of ADHD symptoms after physical activity training programs lasting for several weeks reported (Smith et al., 2013; Verret, Guay, Berthiaume, Gardiner, & Béliveau, 2012). However, as one study reported additional effects on a behavioral inhibition task (Smith et al., 2013), another study failed to replicate these results (Verret et al., 2012), leaving it unclear whether the improvement of ADHD symptoms after the training program was due to an improvement in behavioral inhibition.

Summary. Taken together, prenatal alcohol exposure, prenatal tobacco exposure, SES, parenting and physical activity have both been related to behavioral inhibition and ADHD. However, these associatons have not been investigated systematically and so far, studies yielded mixed results. In particular, it remains unclear whether associations between these environmental factors and ADHD are mediated by deficits in behavioral inhibition.

Situation specific factors

While the last section reported on associations between situation non-specific factors that might have a general and longlasting effect on ADHD symptoms, this section reports on environmental factors that influence behavioral inhibition in a specific situation, in particular during cognitive tasks. Therefore, the first part of this section reviews effects of various stimulus properties on the performance in behavioral inhibition tasks (e.g., Hübner, Lehle, & Steinhausen, 2010). Effects of different stimulus properties will be reviewed in general and afterwards be related to ADHD. Subsequently, the similar procedure will be applied to effects of arousal and motivation.

Stimulus properties. Cognitive psychologists investigated various stimulus properties that influence the performance in behavioral inhibition tasks, especially in the Stroop and Flanker task (e.g., Hübner et al., 2010). Most consistently, experiments have revealed higher interference scores for experimental blocks with a low proportion (e.g., 25%) of incongruent

trials as compared to experimental blocks with a high proportion (e.g., 75%) of incongruent trials (Hübner et al., 2010; Wendt & Luna-Rodriguez, 2009; Wendt et al., 2012). This reduced interference effect within experimental blocks with a low proportion of incongruent trials is generally assumed to reflect attentional focusing (Hübner et al., 2010; Wendt et al., 2012, White, Ratcliff, & Starns, 2011), expectancy effects (Gratton et al., 1992), or contingency learning (Schmidt, 2013; Schmidt & Besner, 2008).

Furthermore, several studies have suggested that cues affect the interference effect in Flanker tasks (Facoetti 2001; Fernandez-Duque & Knight, 2008), Stroop tasks (Fernandez-Duque & Knight, 2008), and Simon tasks (Wühr & Kunde, 2008). Cues that indicated the appearance of incongruent trials led to smaller interference effects, however, the cueing effect might only hold in blocks with a low proportion of incongruent trials (Goldfarb & Henik, 2013).

According to the load theory of attention (Lavie, 2010; Lavie, Hurst, de Fockert, & Viding, 2004), both, perceptual load and cognitive load, influence interference effects of task-irrelevant distractors. Under high perceptual load, the perceptual capacity to process task-irrelevant distractors is reduced and hence, the interference effect of task irrelevant distractors is diminished relative to conditions of low perceptual load (Forster & Lavie, 2007, 2008). In contrast, under a high cognitive load, the capacity to suppress already processed distractors is diminished. Thus, the interference effect of task irrelevant distractors is enhanced relative to conditions of low cognitive load (Lavie et al., 2004).

With regard to ADHD, only one study directly manipulated the proportion of incongruent trials within a Flanker task in female colleage students (Merkt et al., 2013). While female colleage students without ADHD showed higher error rates in experimental blocks with a low proportion of incongruent trials compared to experimental blocks with a high proportion of incongruent trials, female colleage students with ADHD did not show any adaptation to the proportion of incongruent trials. However, as female students with ADHD are a very specific group of participants with ADHD these results need to be replicated in other participants with ADHD as for instance in children.

Studies investigating cueing effects in ADHD mainly focused on cues signaling when a stimulus would occur. However, studies investigating effects of cues signaling the necessity for inhibitory control are rare. One study (King, Colla, Brass, Heuser, & von Cramon, 2007) reported that adults with ADHD were less efficient in using cue information about upcoming stimulus modalities in a task-switching paradigm, but no studies were conducted to examine similar effects in pure inhibition tasks.

Research based on the load theory of attention (Lavie, 2010; Lavie et al., 2004) explored individual differences in interference effects of task-irrelevant distractors under low and high perceptual load. Participants who reported a high distractibility in daily life situations showed higher interference effects but only under a low perceptual load. No individual differences were found for high perceptual load (Forster, 2007). In addition, three studies examined the effects of low versus high load on interference control in children with ADHD (Chan et al., 2009; Huang-Pollack, Nigg, & Carr, 2005) and adults with ADHD (Forster, Robertson, Jennings, Asherson, & Lavie, 2014). Within each study, high load was found to reduce interference effects and this reduction in interference was comparable in patients with and without ADHD.

Arousal. While in ADHD only few studies examined stimuli properties that have classically been investigated in cognitive psychology, several studies investigated the role of stimulus presentation rates determined by the interstimulus interval (ISI) on cognitive and inhibitory performance (e.g., Wiersema, van der Meere, Roeyers, van Coster, & Baeyens, 2006; see van der Meere 2002 for an overview). In comparison to children without ADHD, children with ADHD showed deficits in behavioral inhibition in Go/No-Go tasks only for slow ISIs of one second or large ISIs of about 8 seconds, but not for medium ISIs of 4 seconds (Hervey et al., 2006; Wiersema et al., 2006; but see Epstein et al., 2011). This effect of ISI is generally interpreted in terms of a too low or too high arousal and that children with ADHD have problems in regulating their arousal according to task demands (Sergeant, 2005; see Section 1.1.4.). A recent meta-analysis of ISI manipulations in Go/No-Go tasks (Metin et al., 2012) confirmed the impairing effects of slow and fast ISIs in ADHD. Large ISIs were especially associated with inhibition errors, while slow ISIs were especially associated with a slowing down in response times. However, manipulations of ISIs did not fully explain task performance leaving space for a general deficit in behavioral inhibition as well as other environmental factors (Metin et al., 2012).

Reward and punishment. In studies investigating motivational effects on behavioral inhibition, either correct/fast responses were rewarded or false/slow response were punished. Evidence for effects of reward or punishment on behavioral inhibition is mixed. While one study reported that rewarding of fast and correct responses led to a faster response style, more errors and a larger interference effect in a Flanker task (Seifert, Naumann, Hewig, Hagemann, & Bartussek, 2006) other studies failed to find a modulation of the interference effect by rewards (Hübner & Schlösser, 2010), but reported a modulation of interference effects only for punishments of slow responses (Dambacher, Hübner, & Schlösser 2011). This

heterogeneity in results is in line with the general conclusion that effects of reward and punishment on task performance might be mediated by various variables including the incentive scheme and personal characteristics (Bonner & Sprinkle, 2002).

Studies in children with ADHD paralleled results from general population samples. While some studies reported shorter SSRTs in children with ADHD when correct responses were rewarded and incorrect responses were punished (Epstein et al., 2011; Konrad, Gauggel, Manz, & Schöll, 2000; Slusarek, Velling, Bunk, & Eggers, 2001), other studies observed no effects of reward or punishment on SSRTs (Ooserlaan & Sergeant, 1998) or even an increase in inhibition errors in a Go/No-Go task (Wodka et al., 2007). However, as rewards and punishments differed between studies from verbal feedback (e.g., "Good!") to points or tokens that could later be exchanged for small presents, incentive types and reward schemes might be possible mediators (Bonner & Sprinkle, 2002).

Self-regulatory strategies. In addition to rewarding or punishing responses or errors, several studies examined whether self-regulatory strategies could foster behavioral inhibition. For instance, in the self-regulatory strategy of implementation intentions (Gollwitzer, 1999) participants are taught to form an implementation intention to reach their desired goal (e.g., not to press a button during a No-Go trial in a Go/No-Go task). The implementation intention has the structure of "If situarion x arises, I will perform the goal-directed behavior y". This procedure forms a direct link between the situation x and the goal-directed behavior y that is assumed to trigger two underlying mechanisms. First, detection of the situational cue defined in the if-component of the implantation intention is improved and second, the response to the situational cue is automatized through the situation-response linkage defined in the then-component of the implementation intention (Gollwitzer, 1999; Parks-Stamm, Gollwitzer, Oettingen, 2007). Implementation intentions have been shown to foster behavioral inhibition in Go/No-Go tasks and Simon tasks as compared to normal plans (e.g., goal intentions) that did not use the specific structure of implementation intentions (Cohen, Bayer, Jaudas, & Gollwitzer, 2008; Gawrilow & Gollwitzer, 2008).

Although a review (Reid, Trout, & Schartz, 2005) concluded that self-regulatory strategies (e.g., sef-monitoring, self-monitoring, self-reinforcement) are generally effective to improve ADHD symptoms, no research program has investigated the effects of single specific interventions. Only recently, several studies were conducted to examine the effects of implementation intentions on children with ADHD. Implementation intentions but not goal intentions were found to improve ADHD symptoms (Guderjahn, Gold, Stadler, & Gawrilow, 2013). Furthermore, concerning school management children with ADHD benefited more

from implementation intentions than from a learning style intervention (Gawrilow, Morgenroth, Schultz, Oettingen, & Gollwitzer, 2013). In addition, several studies tested for implementation intentions related improvements in behavioral inhibition in children with ADHD. Children with ADHD made fewer inhibition errors in a Go/No-Go when they formed implementation intentions compared to children with ADHD who formed goal intentions or no intentions at all. Furthermore, differences between children with and without ADHD in behavioral inhibition disappeared after the instruction of implementation intentions (Gawriolow & Gollwitzer, 2008; Paul et al., 2007; Paul-Jordanov et al., 2010). This improvement in behavioral inhibition was accompanied by changes in electrophysiological signals. Implementation intentions increased the No-Go related P300 in children with ADHD (Paul et al, 2007; Paul-Jordanov et al., 2010) and were as effective as medication with methylphenidate (Paul-Jordanov et al., 2010).

Summary. Taken together, although several stimulus properties have been shown to alter the performance during behavioral inhibition tasks, research in ADHD mainly focused on the effects of different ISIs on behavioral inhibition, but ignored other stimulus properties. In addition, motivational effects of reward and punishment are mixed but self-regulatory strategies and especially implementation intentions might improve behavioral inhibition in general and particularly in children with ADHD.

2.1.5. Summary

Examining the criteria for endophenotpes (Crosbie et al., 2008; Doyle et al., 2005; Hasler et al., 2006; Kendler & Neale, 2010; see Section 1.2.), it can be concluded, that behavioral inhibition represented by withholding of responses, inhibition of already initialized responses, and interference control is mainly measured by tasks of a moderate reliability. Deficits in performance on these tasks have consistently been related to ADHD, although several studies reported no differences between affected and unaffected children. Deficits in behavioral inhibition in ADHD are biological plausible, as fronto-basal ganglia circuits that are thought to underly behavioral inhibition, are impaired in children with ADHD. However, it might be possible that some of the neuronal alterations and behavioral deficits reflect impairments in conflict-monitoring rather then impairments in behavioral inhibition per se. Yet, with regard to genetics, higher heritability rates for behavioral inhiition have been reported for unaffected family members as compared to unaffected non-family members. In addition, molecular genetic studies revealed polymorphisms in dopaminergic genes like the

DRD4 and the DAT1 gene to be associated with behavioral inhibition and ADHD. Therefore, behavioral inhibition can be considered as an endophenotype for ADHD. Several non-specific environmental factors have been discussed in relation to behavioral inhibition and ADHD but have not been investigated systematically so far. For instance, while some environmental markers like prenatal alcohol or tobacco exposure are clearly associated with ADHD, it is rather unclear whether this association is mediated by deficits in behavioral inhibition. In contrast, physical activity has been linked to behavioral inhibition and has only recently been shown to have a positive effect on ADHD symptoms. A similar pattern emerges with situation specific environmental factors. While cognitive and social psychologists identified several factors that influence behavioral inhibition in cognitive tasks, these factors have only sparsely been investigated in ADHD. Yet, arousal and self-regulatory deficts are the most promising situation specific factors to impact on behavioral inhibition in ADHD.

2.2. Sustained Attention

In addition to behavioral inhibition, sustained attention has been suggested as another possible cognitive endophenotype for ADHD (de Zeeuw et al., 2012). Sustained attention or vigilance can be defined as a person's readiness to detect signals over a prolonged period of time (Sarter, Givens, & Bruno, 2001). Maintaining a vigilant or alert state (alerting attention) is thought to be one of three major functions of the attentional system, besides orienting attention and executive attention (Petersen & Posner, 2012; Posner & Petersen, 1990). In addition, recent developments in cognitive neuroscience identified two anti-correlated networks within the resting brain: a task-positive network and a task-negative or default mode network (DMN) that both show patterns of very low frequency (< .01 Hz) coherence (Fox et al., 2005; Fransson, 2005; Raichle et al., 2001). While the task positive network has been implicated in response selection and planning of intentional acts, the DMN is associated with introspective attentional orientation and task-unrelated thoughts (Sonuga-Barke & Castellanos, 2007). Due to their anti-correlation, engagement in one of the two networks is incompatible with engagement in the other network and thus an attenuation of the DMN during goal-direct tasks is necessary (Fox et al., 2005). According to the default mode interference hypothesis (Sonuga-Barke & Castellanos, 2007), under circumstances such as a low task motivation the DMN activity persists into or reemerges during the goal directed task, giving rise to attentional lapses or cyclical deficits in performance.

2.2.1. Behavioral Performance

This section reports on the behavioral performance of children with and without ADHD on several measures of sustained attention that can be derived from cognitige tasks. An overview of the tasks and measures reviewed in this section, and whether high or low values of the measures are associated with a deficit in sustained attention is displayed in Table 1.

In order to investigate sustained attention, various forms of Continuous Performance Tasks (CPT; Rosvold, Mirsky, Sarason, Bransome, & Beck, 1956) have been developed, turning CPTs into one of the most common clinical measures of sustained attention (Riccio, Reynolds, Lowe, & Moore, 2002). In most CPTs participants have to detect rare targets among rapidly presented non-targets over a time course of 10-30 min (Huang-Pollack, Karalunas, Tam, & Moore, 2012). Different indices measuring sustained attention have been derived from CPTs, including a higher amount of omission errors and a larger RTV. While no estimates of the reliability of omission errors have been reported, a good split-half reliability (*r*s = .83 - .86) and moderate test-retest reliability (*r*s = .60 - .62) were revealed for RTV (Shalev, Ben-Simon, Mevorach, Cohen, & Tsal, 2011). In addition, more complex indices measuring sustained attention have been derived from signal-detection theory (Sarter et al., 2001; Swets, 1961) and diffusion modeling (Huang-Pollock et al., 2012; Ratcliff & McKoon, 2008). Following signal-detection theory, the number of detected signals is a combination of signal sensitivity and the participant's criterion to report detection of a signal. Given the mean hit and false alarm rates, an index of signal sensitivity (*d'*) can be computed with lower values indicating less signal sensitivity and thus a deficient sustained attention (Sarter, 2001). In comparison, diffusion models for two-choice tasks assume that information about the stimulus accumulate over time until a boundary is reached, determining the initiation of the response. Combining response times and accuracy, three primary parameters can be assessed. The drift rate *v* represents the rate at which an individual gathers information about the encoded stimuli, with a smaller drift rate leading to slower responses. The boundary separation parameter *a* is a measure for response caution with a larger boundary separation leading to slower responses. Finally, the non-decision parameter *Ter* contributes to all preparatory and encoding processes preceding the information accumulation with a larger non-decision parameter leading to slower responses (Huang-Pollck et al., 2012; Ratcliff & McKoon, 2008).

As proposed, several studies revealed a deficit in sustained attention in children and adults with ADHD compared to unaffected children or adults that was evident by a higher amount of omission errors (e.g., de Zeeuw et al., 2012; Epstein et al., 2003; Helps, Broyd,

Bitsakou, & Sonuga-Barke, 2011). In addition, a higher RTV is one of the most reliable findings in individuals with ADHD (Helps et al., 2011; Johnson et al., 2007; for a review see Tamm et al., 2012; for meta-analyses see Karalunas, Geurts, Konrad, Bender, & Nigg, 2014; Kofler et al., 2013). Moreover, lower values for *d'* have been reported in ADHD (Epstein et al., 2003; Nikolas & Nigg, 2013; see Huang-Pollock et al., 2012 for a meta-analysis). With regard to parameters from diffusion models, a smaller drift rate for children with ADHD has consistently been reported in experimental studies and meta-analyses (Huang-Pollack et al., 2012; Karalunas et al., 2014; Karalunas, Huang-Pollock, & Nigg, 2012). However, another study did not report a smaller drift rate but altered boundary separations depending on task instrcutions (Mulder et al., 2010). If instructions emphasized accuracy, children with ADHD had a smaller boundary separation than unaffected children, whereas the opposite hold true, if instructions emphasized on speed suggestimg a smaller speed-accuracy trade-off in children with ADHD (Mulder et al., 2010).

Taken together, measures for sustained attention (omission errors and RTVs) display a moderate to good reliability. In addition, the high consistency of a deficient sustained attention in children and adults with ADHD suggests that deficits in sustained attention might be regarded as a core deficit in ADHD

2.2.2. Neuronal Correlates

Electrophysiology. Electrophysiological studies on sustained attention traditionally focused on spectral changes in the background electroencephalogram (EEG). Deficits in sustained attention are associated with a higher spectral power in the alpha band (8-12 Hz), the theta band (4-8 Hz), and the delta band (2-4 Hz) especially during attentional lapses (Hoedlmoser, Freunberger, Klimsch, Gruber, & Schabus, 2010; Peiris, Jones, Davidson, Carroll, & Bones, 2006). In addition, a higher spectral power in the beta band (12-30 Hz) is regarded as a state of high arousal and has been interpreted as a compensatory effort to maintain sustained attention (Ramautur, Romain, Gómeu-Herrero, Piantoni, & van Someren, 2013). Several studies reported an increased amplitude in the theta band (Clarke, Barry, McCarthy, Selikowitz, & Brown, 2002; White, Hutchens, & Lubar, 2005), as well as a reduced amplitude in the beta band in participants with ADHD compared to unaffected participants (Clarke et al., 2002; Loo et al., 2009; Heinrich et al., 2014; for a review see Barry, Clarke, & Johnstone, 2003; for a meta-analysis see Arns, Connors, & Kraemer, 2013) revealing a general deficit in sustained attention and a problem in coping with this deficit.

However, according to their EEG profiles different EEG-defined subtypes of ADHD might exist but it remains unclear whether these EEG-defined subtypes actually reflect behavioral differences within the disorder (Barry et al., 2003; Clarke et al., 2002).

In addition, based on the default mode interference hypothesis, recent studies tried to find features within the EEG that have the DMN as a source, but it remains unclear whether such features actually exist (Chen, Feng, Zhao, Yin, & Wang, 2008; Knyazev, Slobodskoj-Plusnin, Bocharov, & Pylkova, 2011; but see Helps, James, Debener, Karl, & Sonuga-Barke, 2008). However, less attenuation of very low frequency (VLF) oscillations in the DMN EEG in the transmission from a resting state to active attention was related to symptoms of inattention in undergraduate psychology students (Helps, Broyd, James, Karl, & Sonuga-Barke, 2009; Helps et al., 2008). The results were replicated with adolescents with ADHD showing less attenuation of VLFs than unaffected adolescents in the transmission from resting state to active attention (Helps et al., 2010).

Taken together, deficits in sustained attention in children and adults with ADHD are confirmed by an increased amplitude of the theta EEG band and a decreased amplitude of the beta EEG band, indicating a general deficit in sustained attention and a problem in coping with this deficit. In addition, less attenuation of resting state EEG activity in the DMN in adolescnets and adults with ADHD revealed preliminary evidence for the DMN interference hypothesis to explain deficits in sustained attention in ADHD.

Imaging. Neural models of sustained attention propose a right lateralized fronto-parietal sustained attention network (SAN) including the dorsolateral prefrontal cortex, the mid- and ventrolateral prefrontal cortex as well as the intraparietal sulcus and the temporoparietal junction (Posner & Petersen, 1990; Sarter et al., 2001; see Langner & Eickhoff, 2013 for a meta-analytic review). Activation in the SAN is considered to reflect top-down processes in order to bias sensory processing, especially when attentional effort is needed to maintain performance (Sarter, Gehring, & Kozak, 2006; Sarter et al., 2001). The activity of the SAN is thereby initialized by cholinergic innervations from the basal forebrain that in turn is innervated by norepinephrine projections from the locus coeruleus (LC; Sarter et al., 2001). On an additional route, norepinephrine projections starting in the LC impact on sustained attention via thalamic projections in sensory areas and the SAN (Langner & Eickhoff, 2013; Sarter et al., 2001). However, it is unclear whether the norepinephrine projections to the basal forebrain and the thalamus reflect attentional processes or a more general form of cortical arousal (Aston-Jones & Cohen, 2005; Sarter et al., 2006).

In addition and consistent with the default mode interference hypothesis, trial-by-trial

fMRI studies reported an increased DMN activity accompanied by a decreased activity in the task positive network preceding errors (Eichele et al., 2008) and slow responses (Weissman, Roberts, Visscher, & Woldorff, 2006). However, an increased DMN activity might be especially predictive for a diminished performance within phases where the general performance is high while a decreased activity in the task positive network might be especially predictive for a diminished performance within phases where the general performance is low and error prone (Esterman, Noonan, Rosenberg, & DeGutis, 2013).

Using fMRI, studies investigating sustained attention in ADHD reported a reduced activity in the SAN (Cao et al., 2008; Tamm, Menon, & Reiss, 2006) or the thalamus, especially the pulvinar nuclei (Li et al., 2012) compared to unaffected participants. Moreover, consistent with the default mode interference hypothesis, studies revealed an increased DMN activity in ADHD both during task (Liddle et al., 2011) and resting state (Hoekzema et al., 2014).

These results were supported by sMRI revealing a correlation of cortical thinning in SAN areas and symptoms of inattention in healthy children and adolescents (Ducharme et al., 2012). Furthermore, a structural atrophy of the thalamus and especially the pulvinar nuclei was observed in adolescents with ADHD (Ivanov et al., 2010; Xia et al., 2012). An additional DTI analysis revealed less FA between the thalamus and the PFC, and striatum in ADHD (Xia et al., 2012) and this decreased structural connectivity was accompanied by a decreased functional connectivity between the thalamus and the PFC (Li et al., 2012; Qiu et al., 2011).

Taken together, there is evidence for a sustained attention related impairment of fronto-parietal circuits and the DMN in children with ADHD from a functional, structural, and connectivity perspective.

2.2.3. Genetic Associations

Heritability of RTV has been revealed by twin studies (Groot, de Sonneville, Stins, & Boomsma, 2004; Friedman et al., 2008) and family-genetic studies (Crosbie et al., 2013; Frazier-Wood et al., 2012; Kuntsi et al., 2010). Unaffected siblings of children with ADHD displayed a higher RTV (Rommelse, Altink, Oosterlaan et al., 2008; Slaats-Willemse, Swaab-Barneveld, de Sonneville, & Buitelaar, 2007; Uebel et al., 2010) and committed more omission errors than unaffected control children (Uebel et al., 2010). In addition, a recent study revealed familial clustering of the EEG alpha, beta and theta band (Loo et al., 2010). Thus, behavioral genetic studies revealed heritybility and familial clustering of measures of

sustained attention and EEG correlates of sustained attention in children with ADHD, thereby establishing the condition for molecular genetic studies and candidate gene studies in particular.

Molecular genetic studies using a candidate genes approach mainly focused on polymorphisms in norepinephrine and dopaminergic genes as dopamine is associated with prefrontal cortex functionining in general and norepinephrine with the involvement of norepinephrine projections from the LC in attentional processes (for reviews see Barnes et al. 2011, Bellgrove & Mattingley 2008). Within the norepinephrine system special interest has been paid to polymorphisms of the α2A and α2C receptor genes (ANDRA2A, ANDRA2C; Arnsten & Pliszka, 2011) as well as to polymorphisms of the norepinephrine transporter gene (NET1) and the dopamine beta hydroxylase gene (DBH) that encodes for an enzyme that catalyzes for the conversion of dopamine to norepinephrine within norepinephrine neurons (Banaschewski et al., 2010; Bellgrove & Mattingley, 2008). Within the dopamine system various genetic polymorphisms have been analyzed in relation to sustained attention including polymorphisms of the dopa decarboxylase gene (DDC), DAT1, DRD2, DRD4, and MAO-A (Bellgrove & Mattingly, 2008; Fossella et al., 2002; Zhu et al., 2013). However, so far only few studies have analyzed effects of these genetic polymorphisms on behavioral outcomes during sustained attention tasks in non-clinical samples. One study each reported an effect of a functional MAO-A variant (Fossella et al., 2002) and DDC variant (Zhu et al., 2013) on response times while another study found T homocygotes of the DBH C-1021T genotype tending to make more omission errors than carriers of at least one C allele (Greene, Bellgrove, Gill, & Robertson, 2009). Despite the relevance of the cholinergic system in sustained attention (Sarter et al., 2006) only one study examined the associations between a polymorphism (the Ile89Val variant) of the choline transporter gene (CHT) and measures of attention (Berry et al., 2014). The Ile89Val variant of the CHT reduces the rate of choline transport by approximately 40%-60% (Okuda, Okamura, Kaitsuka, Haga, & Gurwitz, 2002) and was positively related to the impact of external distractors on adolescents' performance in search task as well as their reports of distraction during daily life (Berry et al., 2014)

Meta-analyses on candidate gene studies in ADHD (Faraone et al., 2005; Gizer et al., 2009) tested effects of various polymorphisms of the DBH gene with only the Taq1 A polymorphism showing a significant association. However, there was a huge heterogeneity within the effect sizes and further studies are needed to confirm or falsify the reported effects (Gizer et al., 2009). In addition, different polymorphisms on the NET1 gene have been related to ADHD (e.g., Bobb et al., 2005; Kim et al., 2006) making meta-analyses difficult, as the

absence of an effect might be due to heterogeneity in the reported polymorphisms (Gizer et al., 2009). Furthermore, an involvement of polymorphisms of the ANDRA2A gene is inconsistent with a meta-analysis inferring no association of polymorphisms in this gene with ADHD (Gizer et al., 2009) and a review coming to a completely opposite conclusion (Banaschewski et al., 2010). Furthermore, a recent study reported polymorphisms of the CHT being related to ADHD (English et al., 2009). However, the association seemed to be stronger with the combined presentation of ADHD than with the preliminary inattentive presentation, thus questioning the connection between the CHT and sustained attention in ADHD.

Using behavioral measures of a CPT, an association between increased RTV and the A2 allele of the DBH Taq1 A polymorphism (Bellgrove, Hawi, Gill, & Robertson, 2006) has been reported as well as an association to a polymorphism of the NET1 gene (Kollins et al., 2008). However, one of the two studies (Bellgrove et al., 2006) did not genotype the control group and the other study only used participants from families with at least one child having a diagnosed ADHD (Kollins et al., 2008). However, a recent study reported that an association between polymorphisms on the ANDRA2A gene and self-reported ADHD symptoms in adults was mediated by a larger RTV.

Concerning the dopamine system the DAT110/10-genotype and a polymorphism of the dopamine receptor D5 gene have been associated with increased RTV in children with ADHD (Bellgrove, Hawi, Kirley, Gill, & Robertson, 2005; Loo et al., 2003; Manor et al., 2004). Furthermore, both the DRD4 7-repeat allele and the DRD4 4-repeat allele have been related to a higher number of omission errors in a CPT (Bellgrove, Hawi, Lowe, et al., 2005; Gizer & Waldman, 2012). However, again these studies either did not genotype the control group (Bellgrove, Hawi, Kirley et al., 2005; Bellgrove, Hawi, Lowe et al., 2005) or only used participants with diagnosed ADHD (Loo et al., 2003) or used participants from families with at least one child having diagnosed ADHD (Gizer & Waldman, 2012). In addition, two different polymorphisms on the DRD4 gene have been related to symptoms of inattention acquired by a clinical interview (Lasky-Su et al., 2008). Only one study analyzed associations of polymorphisms in the ANDRA2A gene and various executive functions measures (Waldman et al., 2006) reporting the most consistent associations to dependent variables derived from planning tasks, and shifting tasks as well as response speed. However, besides one index of RTV in a stop signal tasks, no measures of inattention had been included leaving it unclear whether there is an association between polymorphisms in the ANDRA2A gene and measures of inattention in ADHD.

Taken together, there is evidence for heritability of sustained attention from behavioral genetics studies as well as molecular genetics studies. Partcularly, polymorphisms in norepinephrine genes as the NET1 gene and the ANDRA2A gene might be involved in the sustained attention deficit in children with ADHD, but polymorphisms in dopaminergic genes might be involved as well.

2.2.4. Environmental Effects

Regarding sustained attention, neither situation non-specific factors nor situation specific factors have been systematically investigated. Situation non-specific factors mainly focused on prenatal factors, parenting, and physical activity whereas situation specific factors included mind wandering and motivation.

Situation non-specific factors

This section reports on prenatal tobacco exposure, parenting, and physical activity. For each environmental factor first associations are reported with measures of sustained attention. Second, associations with the disorder of ADHD are described and finally evidence is reviewed for a mediation of these effects through sustained attention.

Prenatal tobacco exposure. While most studies on prenatal tobacco exposure have focused on general cognitive outcomes or executive functions (Huizink & Mulder, 2006; see Section 2.1.4.) only few studies related prenatal tobacco exposure to measures of sustained attention (Leech, Richardson, Goldschmidt, & Day, 1999). Prenatal tobacco exposure during the second and third trimester of pregnancy predicted higher amounts of omission errors in a CPT in six-year-old children (Leech et al., 1999) and despite being a a risk factor for ADHD (Nigg et al., 2010; Pineda et al., 2007; see Section 2.1.4.), only one study examined prenatal tobacco exposure in relation to sustained attention (Motlagh et al., 2011). In children and adolescents with ADHD prenatal tobacco exposure was related to a larger RTV derived from a CPT. However, as the study did not include children without ADHD, it remains unclear whether prenatal tobacco exposure can explain differences between children with and without ADHD on measures of sustained attention.

Parenting. Several studies investigated the effects of parenting and family environment on sustained attention in early infants and children (Graziano, Calkins, & Keane, 2011; Razza, Martin, & Brooks-Gunn, 2010). However, studies disagree whether aspects of positive parenting (e.g., maternal warmth, maternal sensitivity, maternal responsivity) foster sustained attention or whether aspects of negative parenting (e.g., maternal intrusiveness, maternal hostility, overcontrolling) impair sustained attention (Dilworth-Bart, Khurshid, & Vandell, 2007; Graziano et al., 2011; Miller, Ables, King, & West, 2009; Razza et al., 2010)

Evidence on the influence of parenting on sustained attention in ADHD is scarce. While a better quality of the home environment, for instance a home environment high in structural and temporal routines, might be associated with fewer ADHD symptoms (Mokrova, O'Brien, Calkins, & Keane, 2010; Mulligan et al., 2013), the effect might not be related to inattention but to impulsivity and hyperactivity (Mulligan et al., 2013). In addition, parental intrusiveness positively predicted ADHD symptoms, while parental affirmation, warmth and affection towards the child had a protective effect (Keown, 2012). However, no study investigated whether parenting effects on ADHD might be mediated by sustained attention.

Physical activity. Recent studies investigated the effects of physical activity on sustained attention. A 30 minutes physical activity program fostered sustained attention in preschoolers relative to a sedentary condition. Preschoolers in the physical activity program committed less omission errors in an age-appropriate behavioral CPT (Palmer, Miller, & Robinson, 2013). Similarly, as compared to children with a higher cardiorespiratory fitness, less fit children made more omission errors and more sequential omission errors (two or more omission errors in a row) in a modified Flanker task (Pontifex, Scudder, Drolette, & Hillman, 2012) and displayed a higher RTV than physically fit children (Wu et al., 2011).

As most studies on physical activity (Berwid & Halperin, 2012; see Section 2.1.4.) examine associations to behavioral inhibition or cognitive control, evidence for effects of physical activity on sustained attention is rare. Yet, investigating physical activity in relation to sustained attention might be particularly interesting in ADHD, as a catecholamine hypothesis of physical activity (McMorris, Collard, Corbett, Dicks, & Swain, 2008) states that physical activity increases the production of neurotransmitters norepinephrine and dopamine, which are both involved in sustained attention (Sarter et al., 2001). However, evidence for the catecholamine hypothesis of physical activity is mixed (McMorris et al., 2008) and although one study, which investigated the effects of physical activity on sustained attention in children with ADHD, reported an improvement in CPT performance, these effects were independent from physical activity related catecholamine production (Medina et al., 2010).

Summary. Taken together, prenatal tobacco exposure, parenting and physical activity have both been related to sustained attention and ADHD. However, these associatons have not been investigated systematically and only few studies investigated whether associations between these environmental factors and ADHD are mediated by deficits in sustained attention.

Situation specific factors

While the last section reported on situation non-specific factors that might have potentially longlasting effects on the ability to sustain attention, this section reports on environmental factors that influence sustained attention performance in specific situations, in particular during cognitive tasks. The first part of this section reviews effects of task unrelated thoughts of sustained attention, whereas the second part of this section reports on effects of arousal.

Task unrelated thoughts. Many problems in sustained attention have been attributed to task-unrelated thoughts (Smallwood et al., 2004). For instance, during a sustained attention task a participant might shift from processing external information belonging to the task towards processing internal information that is task unrelated and might instead reflect current life goals or concerns (Klinger, 1999; Smallwood et al., 2004). These task-unrelated thoughts have been termed mind wandering (Smallwood et al., 2004; Smallwood & Schooler, 2006) and have been related to higher DMN activity (Mason et al., 2007). However, although the DMN has been implicated in ADHD, sustained attention, and mind wandering (Liddle et al., 2011; Sonuga-Barke & Castellanos, 2007), no study directly related mind wandering to deficits in sustained attention in children with ADHD.

Arousal. Instead research on ADHD focused on deficits in arousal or energetic states to explain task-unrelated thoughts (Sergeant, 2000; Zentall & Zentall, 1983). Arousal has been thought to improve sustained attention in general (Matthews & Davies, 2001) and to be a means for overcoming task-unrelated thoughts due to DMN activity in particular (Sonuga-Barke & Castellanos, 2007). For instance, according to the default mode interference hypothesis, activity of the DMN interferes with task performance only under the condition of low motivation or arousal. However, only few studies investigated effects of arousal manipulation on sustained attention in children with ADHD (van der Meere, 2002). When arousal was manipulated by event rates, only high event rates were found to produce deficits in sustained attention, but not medium event rates (van der Meere, 2002). In addition to a

simple comparision of performance for different durations of ISIs, a recent study compared the effects of fixed ISIs to randomized ISIs of different durations (Lee et al., 2014). Results revealed that children with ADHD displayed a higher RTV only under the condition of fixed ISIs, but not under the condition of randomized ISIs of different duration (Lee et al., 2014). These results were consistent with the assumption that randomized ISIs of different duration might increase arousal to a more optimal energetic state.

Summary. Taken together, mind wandering and a reduced arousal might be related to deficits in sustained attention. However, mind wandering has not been investigated in children with ADHD yet and only few studies examined effects of arousal on sustained attention in ADHD by manipulating ISIs. Hence, the role of situation specific environmental factors on sustained attention is understudied and no final conclusions can be drawn whether situation specific factors moderate effects of sustained attention on ADHD.

2.2.5. Summary

Examining the criteria for endophenotypes (Crosbie et al., 2008; Doyle et al., 2005; Hasler et al., 2006; Kendler & Neale, 2010; see Section 1.2.) a modest reliability was found for measures of sustained attention. However, as different measures of sustained attention have been suggested, including omission errors, RTV as well as measured derived from signal-detection theory and diffusion modeling future studies need to examine whether these measures are correlated and actually measure the same construct. Nevertheless, deficits in sustained attention and in particular a higher RTV (Kofler et al., 2013; Tamm et al., 2012) have been related to ADHD. On a neuronal level, fronto-parietal circuits and the DMN are thought to underline sustained attention. In addition, there is preliminary evidence for familial clustering of sustained attention deficits. However, the molecular genetic contributions to sustained attention need further study, although polymorphisms in norepinephrine candidate genes, as for instance the ANDRA2A and the NET1 gene, might be promising. Despite the notion of motivational aspects and especially the notion of arousal in the default mode interference hypothesis as well as the state regulation theory of ADHD (Sergeant, 2000; see Section 1.1.4.), studies investigating situation specific factors like arousal, motivation, or load in relation to sustained attention in children with ADHD are rare. Similarly, there is no systematic investigation of situation non-specific factors with regard to a sustained attention deficit in ADHD, although parenting and physical activity might be candidates for future studies.

2.3. Beyond Endophenotypes: Impulsivity, Risky Decision Making, and ADHD

Relying on the previously outlined research framework, ADHD symptom dimensions are assumed to consist of several facets of higher-order psychological functions, which are more homogeneous than the total symptoms dimensions. In addition, basal cognitive functions, as for instance behavioral inhibition or sustained attention, might not be related to the total ADHD symptom dimension but rather to single higher-order facets. Exemplarily, in this section risky decision-making will be introduced as a facet of impulsivity and assoications with behavioral inhibition and sustained attention will be examined.

Impulsivity. Impulsivity is one of the three core symptoms of ADHD. According to the DSM-V (American Psychiatric Association, APA 2013) impulsivity refers to hasty actions that occur without forethought and are potentially harmful. In addition, impulsivity reflects the inability to delay gratification, social intrusiveness, and decision-making without consideration of long-term consequences. However, while these are mainly a list of behavioral examples, no explicit definition of impulsivity can be found in the DSM-V and although several definitions for impulsivity exist (e.g., see Moeller, Barratt, Dougherty, Schmitz, & Swann, 2001 for a clinical context) different researchers are unable to agree upon a single definition (Enticott & Ogloff, 2006). Furthermore, a huge variety of different behaviors has been related to impulsivity (Evenden, 1999), but only little overlap has been found for measures of impulsivity derived from questionnaires and behavioral tasks (Cyders & Coskunpinar, 2011) leading to the assumption that impulsivity is not a unitary construct (Cyders & Coskunpinar, 2011; Enticott & Ogloff, 2006). Accordingly, impulsivity is thought to consist of several facets that each might have several subcomponents (Evenden, 1999; Whiteside & Lynam, 2001; see Stahl et al., 2014 for a behavioral components model). For instance, a lack of premeditation, urgency, sensation seeking, and a lack of perseverance have been suggested possible facets for impulsivity (Whiteside & Lynam, 2001). Lack of premeditation refers to the inability think before acting, urgency reflects the tendency to commit rash or regrettable actions, sensation seeking measures the tendency to seek excitement and adventure, and lack of perseverance refers to a person's inability to complete a task (Whiteside & Lynam, 2001).

Risky decision-making is a complex behavior that has been associated with impulsivity and with urgency and sensation seeking in particular (Bayard, Raffard, & Gely-Nargeot, 2011; Billieux, Gay, Rochat, & van der Linden, 2010). In decision-making under risk, participants have to choose between two alternatives, which are associated with particular probabilities (Kahneman & Tversky, 1979) and a risky decision can be defined as a

decision for an alternative with a lower probability. In most tasks, alternatives with lower probabilities are often assigned higher gains (e.g., payoff) but also higher losses. Therefore, risky decisions refer to decisions that are associated with a lower probability for a higher gain and a higher probability for a loss.

Items on risky decision-making have been part of most questionnaires measuring impulsivity as for instance the Urgency, Premediation, Perseverance, and Sensation Seeking (UPPS) Impulsive Behavior Scale (Magid & Colder, 2007). In addition, risky decision-making is highly common in populations that are supposed to be impulsive as for instance pathological gamblers, and people suffering from substance abuse or schizophrenia (Bechara et al., 2001; Reddy et al., 2014; see Buelow & Suhr, 2009 for further discussion). Thus, risky decision-making might be a valid facet of impulsivity with a less diverse behavioral expression, albeit still being a complex behavior itself.

In the following sections, I will describe common tasks to assess risky decision-making (Section 2.5.1.) as well as cognitive and affective mechanisms underlying risky decision-making (Section 2.5.2) and associations between risky decision-making and ADHD (Section 2.5.3).

2.3.1. Measures of Risky Decision-Making

Several behavioral tasks have been developed to assess risky decision-making in clinical and non-clinical samples, but the tasks most common used are the Iowa Gambling Task (IGT; Bechara, Damasio, Damasio, & Anderson, 1994), the Balloon Analogue Risk Task (BART; Lejuez et al., 2002), and the Game of Dice Task (GDT; Brand, Fujiwara, et al., 2005).

In the IGT (Bechara et al., 1994; Bowman, Evans, & Turnbull, 2005) participants receive $2000 play money to start and are instructed to maximize their profit over the course of the game (usually 100 trials). In each trial participants can choose one card from four different decks. Each card from Deck A and Deck B yield a profit of $100, while each card from Deck C and Deck D yield a profit of $50. However, different loss amounts are associated with the specific decks, in a sense that after ten selections from Deck A or Deck B participants would have incurred a net loss of $250, whereas after ten selections from Deck C or Deck B participants would have incurred a net win of $250 (Bechara et al., 1994). Thus, while providing larger gains in the short run but an overall loss in the long run, the decks A and B are disadvantageous decks. In contrast, although providing smaller gains in the short

run but an overall gain in the long run, the decks C and D are advantageous decks (Buelow & Suhr, 2009). The IGT has been administered both using decks of paper cards and in a computerized task with no differences regarding risky decision-making between the two versions (Bowman et al., 2005). Several outcome measures can be derived from the IGT, including the final outcome, the number of choices for each separate deck, the number of advantageous choices, and the number of risky choices (Buelow & Suhr, 2009). However, the most frequently used outcome measure is the difference between total advantageous choices and total disadvantageous choices. This difference score can be computed for the task in total as well as for subsets of blocks, in order to investigate learning effects or strategies (Bueleow & Suhr, 2009). Normative data exists for the final outcome, outcomes for block subsets, and the total number of cards selected from each deck (Bechara, 2007). However, while reliability and construct validity might depend on personality and emotional states (Lin, Song, Chen, Lee, & Chiu, 2013; Buelow & Suhr, 2009) the IGT generally detects decision-making deficits in clinical populations, as for instance in pathological gamblers and patients suffering from substance abuse or schizophrenia (Buelow & Suhr, 2009).

The Balloon Analogue Risk Task (BART) has been developed to simulate risky decision-making in natural settings (Lejuez et al., 2002). In a computerized task, participants are instructed to inflate a series of 90 balloons by pressing an indicated key. With every pump the size of the balloon increases and money is added to the participant's temporary bank account. However, each balloon has an individual explosion point and when the number of pumps exceed this point, a pop sound emerges and the money on the temporal bank account is lost. The next trial either starts after an explosion or when the participant decides to stop pumping. Participants can stop any time by clicking a cash button. Clicking this button then transfers the money from the temporal bank to a permanent bank. Several outcome measures can be derived from the BART including the total number of pumps, the number of pumps on the non-exploded trials, and the number of exploded balloons (Lejuez et al., 2002; Hunt, Hopko, Bare, Lejuez, & Robinson, 2005). Separate BART tasks have been developed for adults and adolescents (Lejuez et al., 2007; Lejuez et al., 2002) and associations with impulsivity confirmed construct validity (Hunt et al., 2005; Lauriola, Panno, Levin, & Lejuez, 2013).

In contrast to the IGT and the BART, in the Game of Dice Task (GDT; Brand, Fujiwara et al., 2005) the magnitude and probability of outcomes is explicitly stated and does not require learning or guessing. In the computerized task participants start with a virtual starting capital (e.g., 1000 €). Then a virtual dice is thrown 18 times and participants have to

maximize their money by betting on the dice outcome. Participants can bet on a single dice (e.g., that the thrown dice will be a one; chance 1:6), or on a combination of two dice (e.g., that the thrown dice will be either a one or a two; chance 2:6), three dice (e.g., that the thrown dice will be either a one, a two or a three; chance 3:6), or four dice (that the thrown dice will be either a one, a two, a three or a four; chance 4:6). Bets are related to different rewards and punishments (e.g., 1000, 500, 200, or 100 €) with riskier choices being related to higher gains or losses of the same magnitude.

Construct validity of the GDT has been shown by a worse performance in pathological gamblers and highly impulsive clinical groups as compared to healthy controls (Brand, Fujiwara et al., 2005; Brand, Kalbe, et al., 2005; Labudda et al., 2010).

2.3.2. Mechanisms in Risky Decision-Making

Recent neuroscientific theories assume that two neural systems contribute to risky decision-making: a phylogenetically older affective system and a phylogenetically younger deliberative, cognitive-control system (Casey, Getz, & Galvan, 2008; Steinberg, 2008). The affective system has been located to brain regions associated with reward processing and motivation, specifically to the amygdala and ventral striatum as well as the orbitofrontal cortex and medial prefrontal cortex (Steinberg, 2008). On a behavioral level both reward sensitivity and loss aversion have been related to risky decision-making (Cavanagh et al., 2012; Crone, Vendel, & van der Molen, 2003; Ert & Erev, 2013). For instance, larger rewards were associated with higher risk taking in older adults above the age of 60 in the BART (Cavanagh et al., 2012) and in children in a coin game task (Paulsen, Platt, Huettel, & Brannon, 2011). Adults who scored higher on reward sensitivity played more risky in the IGT (Davis, Patte, Tweed, & Curtis, 2007; Franken & Muris, 2005). Similar results have been reported for adolescents. Adolescents who scored higher on reward sensitivity measures or need for arousal scales played more risky in a card game, where they had to turn over card after card to gain or lose points (Figner, Mackinlay, Wilkening, & Weber, 2009; Penolazzi, Gremigni, & Russo, 2012), a procedure that was shown to trigger neuronal activity in the affective system (Aron et al., 2004). In contrast, loss aversion describes the phenomenon that losses are more significant than gains (Kahneman & Tversky, 1979). That is, the absolute subjective value of a specific loss is larger than the absolute subjective value of an equivalent gain because people expect losses to have greater hedonic impact than gains (Ert & Erev, 2013; Kahneman & Tversky, 1979; Kermer, Driver-Linn, Wilson, & Gilbert, 2006; McGraw,

Larsen, Kahneman, & Schkade, 2010). Loss aversion has been observed in the IGT and the BART (Bishara et al., 2009; Fukunaga, Brown, & Bogg, 2012; Stocco, Fum, & Napoli, 2009) and other gambling tasks (Tom, Fox, Trepel, & Poldrack, 2007) but might be especially present when losses and gains can be directly compared (McGraw et al., 2010), when the safe alternative is framed as the status quo, or when loss aversion maximizes the probability of positive rather then zero or negative outcomes (see Ert & Erev, 2013 for a review).

The deliberative, cognitive-control system has been located to brain regions associated with behavioral inhibition and attentional processing, specifically to the dorsolateral prefrontal cortex and the posterior parietal cortex (Casey et al., 2008; Steinberg, 2008). The deliberative processing system is assumed to accumulate information about the consequences of decision and to evaluate these consequences before making a decision (Weber, Shafir, & Blais, 2004). As the deliberative, cognitive control system evaluates long-term consequences of a decision, it is assumed to inhibit affective impulses that are driven by short-term gains or losses (Knoch & Fehr, 2007). Indeed, recent studies revealed evidence for an involvement of a deliberative, cognitive-control system in the IGT (Stocco et al., 2009) and a card game, where gains, potential outcomes, and their probabilities were explicitly stated (Figner et al., 2009). However, evidence for an association between measures of behavioral inhibition and risky decision-making is mixed. While deficits in behavioral inhibition were generally observed in pathological gamblers (Goudriaan, Oosterlaan, de Beurs, & van den Brink, 2006; Kertzman et al., 2008; Odlaug, Chamberlain, Kim, Schreiber, & Grant, 2011; but see Lawrence, Luty, Bogdan, Sahakian, & Clark, 2009) and a short training of behavioral inhibition through a Stop-Signal Task reduced risky decision-making in a monetary gambling task up to two hours after the training session (Verbruggen, Adams, & Chambers, 2012), several studies reported no or only small associations between behavioral inhibition and risky decision-making in healthy adolescents or adults (Crone, Vendel, et al., 2003; Hooper, Luciana, Conklin, & Yarger, 2004; see Toplak, Sorge, Benoit, West, & Stanovich, 2010 for a review).

Beyond processes of behavioral inhibition, a recent study reported associations between sustained attention and risky decision-making (Gansler, Jerram, Vannorsdall, & Schretlen, 2011). Confirming these results and beyond loss aversion, losses were found to increase the allocation of attention to task relevant parameters thereby decreasing the likelihood of random responses (Yechiam & Hochman, 2013a, 2013b).

2.3.3. Risky Decision-Making in ADHD

Symptoms of ADHD have been related to problem or pathological gambling among children, adolescents, and adults in general samples (Breyer, Botzet, Winters, Stinchfield, August, & Realmuto, 2009; Derevensky, Pratt, Hardoon, & Gupta, 2007; Faregh & Derevensky, 2011) and because pathological gambling is characterized by risky decision-making, several studies investigated risky decision-making in relation to ADHD (Drechsler, Rizzo, & Steinhausen, 2008; Ibanez et al., 2012; Mäntylä, Still, Gullberg, & del Missier, 2012; Matthies, Philipsen, & Svaldi, 2012; see Groen, Gaastra, Lewis-Evans, & Tucha, 2013 for a review). Mapping the affective system and the deliberative, cognitive control system in riksy decision-making (Casey et al., 2008; Steinberg, 2008; see Section 2.5.2) on multiple pathway models of ADHD (Sonuga-Barke, 2002; Sonuga-Barke et al., 2010), it is assumed that delay aversion particularly affects the affective risky decision-making system (e.g., in tasks with implicit probabilities, gains, and losses), whereas behavioral inhibition particularly affects the deliberative, cognitive control system (e.g., in tasks with explicit probabilities, gains, and losses; see Groen et al., 2013).

However, evidence for a deficit in implicit risky decision-making tasks like the IGT and the BART is mixed. While some studies reported higher risk taking in the IGT in both children with ADHD (Garon, Moore, & Waschbusch, 2006; Luman, Oosterlaan, Knol, & Sergeant, 2008) and in adults with ADHD (Malloy-Diniz, Fuentes, Leite, Correa, & Bechara, 2007; Mäntylä et al., 2012), other studies failed to replicate these results (Agay, Yechiam, Carmel, & Levkovitz, 2010; Geurts, van der Oord, & Crone, 2006). In addition, compared to unaffected children or adults, children with ADHD (Humphreys & Lee, 2011), but not adults with ADHD, played more risky in the BART (Mäntylä et al., 2012; Weafer, Milich, & Fillmore, 2011). However, the inconsistencies between the studies might be due to different reward magnitudes and frequencies used in the different studies, as children with ADHD might differ from unaffected children only in their response to reward frequency but not reward magnitude (Groen et al., 2013; Luman et al., 2008; Masunami, Okazaki, & Maekawa, 2009).

The few studies that tested ADHD in relation to explicit decision-making tasks like the GDT yielded mixed results. For instance, higher risky decision-making was reported for children and adults with ADHD in the GDT (Drechsler et al., 2008; Matthies et al., 2012 study 1). However, children with ADHD only made more risky decisions in the GDT than unaffected children if the task was played a second time, indicating problems in learning and adapting to the task demands (Drechsler et al., 2008). Furthermore, no differences were found

in adults with and without ADHD in the GDT after an boredom induction (Mathies et al., 2012 study 2) and another study reported no differences at all between adults with and without ADHD (Wilbertz et al., 2012).

2.3.4. Summary

Impulsivity is not a unitary construct but consists of several facets (Enticott & Ogloff, 2006; Stahl et al., 2014; Whiteside & Lynam, 2001; see Section 2.5.). One of these facets is risky decision-making. Risky decision-making in turn is underlined by two systems: an affective system and a deliberative, cognitive control systems (Steinberg, 2008). As these systems can be linked to multiple pathway models of ADHD and to delay aversion and behavioral inhibition in particular (Sonuga-Barke, 2002), patients with ADHD are assumed to play more risky in risky decision-making tasks (Groen et al., 2013). However, evidence for a relation between risky decision-making and ADHD is inconclusive and further studies are needed. These studies should not only associate risky decision-making with ADHD per se but should link risky decision-making to psychological processes implicated in multiple pathway models of ADHD (see Section 1.1.6).

2.4. A Research Framework for ADHD: Summary

Based on multiple pathway models of ADHD (de Zeeuw et al., 2012; Sonuga-Barke et al., 2010; see Section 1.1.6.) and the endophenotype approach (Gottesman & Gould, 2003; see Section 1.2.) this section presents a research framwork for ADHD that incorporates psychological, neuronal, genetic and environmental factors to deal with different forms of heterogeneity in ADHD (see Figure 2). Examining the criteria for endophenotypes, behavioral inhibition and sustained attention have been related to ADHD, although the reliability of most measures was only modest. Behavioral inhibition and sustained attention can be assigned to different neuronal circuits with fronto-basal ganglia circuits mainly related to behavioral inhibition and fronto-parietal circuits mainly related to sustained attention (Durston et al., 2011; Petersen & Posner, 2012; Posner & Petersen, 1990), thereby ensuring biological plausibility of behavioral inhibition and sustained attention as endophenotypes. Heritability has been established for both behavioral inhibition and sustained attention through behavioral genetics studies and confirmed by molecular genetics studies. Polymorphisms in dopaminergic genes and in particular in the DRD4 and DAT1 gene have been related to

behavioral inhibition, while polymorphisms in norepinephrine genes like the NET1 have been associated with sustained attention. Several situation non-specific factors have been related to ADHD and the psychological endophenotypes. Prenatal exposure to alcohol or tobacco has been related to behavioral inbibition, sustained attention and their underlying neuronal circuits and SES, parenting and physical activity might operate on both psychological functions as well. With regard to situation specific factors both motivation/arousal and cognitive/perceptual load influence behavioral inhibition and sustained attention. Thus, although situation specific and situation non-specific environmental factors have not been investiagated systematically in relation to psychological functions in ADHD, the reported evidence suggests that environmental factors might constitute a general rather than specific risk factor for deficient neuropschological functions and psychopathology (see Caspi et al., 2014).

Moving beyond endophenotypes (Sabb et al., 2009), the framework proposes that psychological functions are not related to ADHD as a syndrome but rather to facets of the core symptoms inhibition, hyperactivity, and inattention. Risky decision-making has been identified as a candidate facet of impulsivity that has been related to ADHD as well, although the results were mixed. However, given that an affective, motivational and a deliberative, inhibitory process are assumed to underly risky decision-making (Casey et al., 2008; Steinberg, 2008) and that behavioral inhibition and shortened delay-of-reinforcement gradient are related to ADHD, it might be that deficits in risky decision-making in ADHD are driven by deficits in the more basal functions of behavioral inhibition and delay aversion.

To summarize, this framework moves beyond existing theories of ADHD (Crosbie et al., 2008; Durston et al., 2011; Sonuga-Barke et al., 2010) by relating genes and neuronal structures not only to behavioral inhibition but also to sustained attention and the framework can easily be extended to include further potential endophenotypes as for instance delay aversion or temporal processing. In addition, environmental factors have been incorporated and divided into situation specific and situation non-specific factors as the latter might particullarly influence neuronal circuits while the former might determine whether these neuro-cognitive deficits matter in a specific situation (Bäckman & Dixon, 1992). The framework further acknowledges that ADHD as a syndrome is compounded of different symptoms that in turn have various facets. Analyzing these facets (e.g., risky decision-making) in relation to endophenotypes can benefit further research on ADHD as these facets represent smaller well-defined, quantitative behavioral outcomes instead of categorical

diagnoses that ignore heterogeneity within the disorders (Cuthbert, 2005; Hyman, 2010; Morris & Cuthbert, 2012).

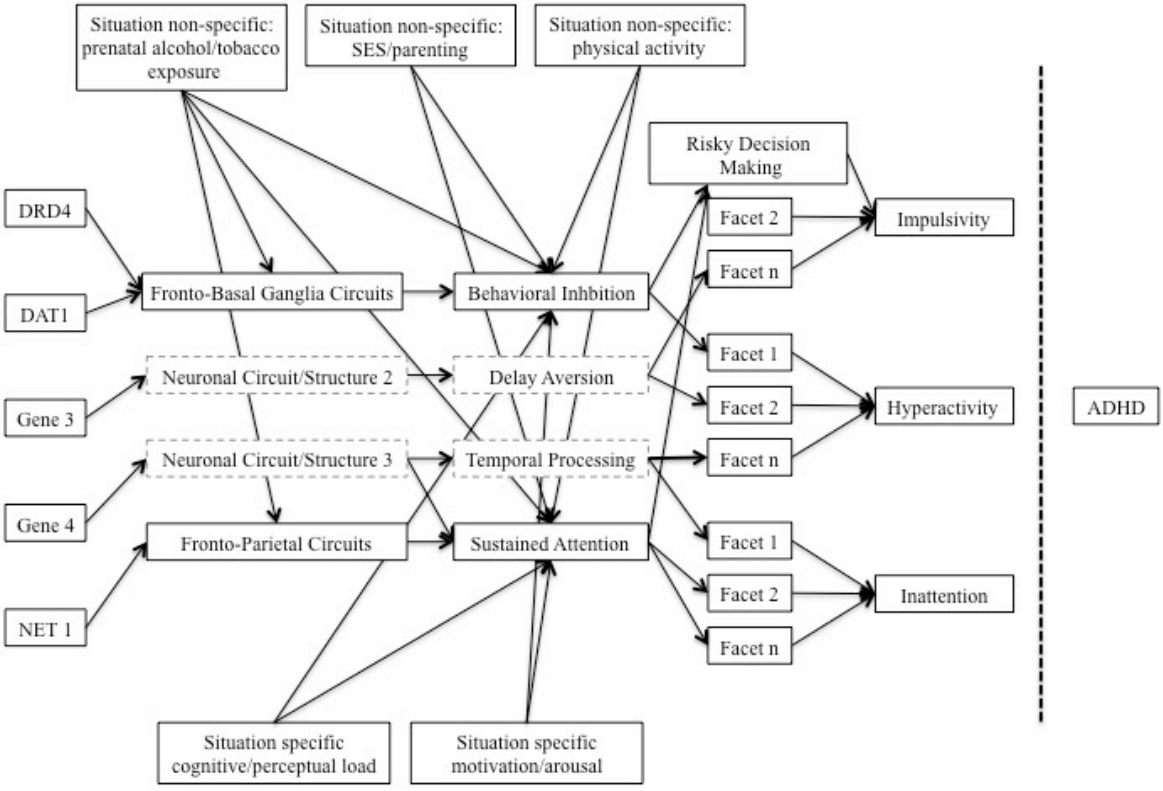

Figure 2. Research framework to study ADHD. The first layer displays several genes that impact on psychological functions (dashed frames indicate psychological functions not reviewed in this thesis) through their influence of neuronal structures and functions. Genetic and neuronal effects on psychological functions are accompanied by situation specific and situation non-specific factors. Finally, psychological functions are related to risky decision-making and other behavioral manifestations of ADHD symptoms. If the severity of the symptoms exceeds a threshold (dashed line), ADHD can be diagnosed. For a better readability the figure does not include connections within the layers (e.g., connections between genes, neuronal circuits or psychological functions) although such connections are possible and likely.

3. Present Study

The presented research framework for the study of ADHD provokes several research questions, of which the following questions have been addressed in the present study: (1) With regard to psychological endophenotypes, it was examined whether sustained attention and behavioral inhibition constitute independent endophenotypes of ADHD and whether sustained attention or behavioral inhibition was more strongly impaired in children with ADHD relative to unaffected children. Concerning behavioral inhibition it was additionally assumed that deficits in children with ADHD were partly due to deficits in conflict-monitoring. (2) With regard to higher-order psychological functions an increase in risky decision-making (a facet of impulsivity, see Section 2.3.) was predicted for children with ADHD compared to unaffected children. Since previous studies relying on single measures of risky decision-making yielded mixed results (see Section 2.3.3.), several measures of risky decision-making were used to obtain information about the reliability and consistency of an increase in risky decision-making in children with ADHD compared to unaffected children. The third research question (3) addressed the associations of sustained attention, behavioral inhibition, conflict-monitoring, risky decision-making and ADHD symptoms. In particular, it was investigated whether the associations were stronger for ADHD symtoms than for the categorical ADHD diagnosis and whether associations between sustained attention, behavioral inhibition, conflict-monitoring and risky decision-making were equally related to every symptom of ADHD. Stronger associations were assumed for symptoms of inattention and measures of sustained attention on the one hand and for associations between symptoms of impulsivity and measures of behavioral inhibition and risky decision-making on the other hand. Finally, the last research question (4) concerns the prediction that associations of psychological endophenotypes and ADHD are mediated by higher-order psychological functions. Specifically, it was examined whether associations of deficits in sustained attention, behavioral inhibition, and conflict monitoring with ADHD symptoms were mediated through enhanced risky decision-making.

This chapter provides the reader with necessary information on methodological aspects of the study including participant characteristics (Section 3.1.), the study's procedure (Section 3.2.), and the measures of sustained attention, behavioral inhibition, and risky decision-making that were applied (Section 3.3.). The section ceases with a description of the data preparation procedure (Section 3.4.).

3.1. Participants

The study was part of a larger project on "Gene-by-Environment Interactions on Decision Making in Children with ADHD" (GIDeCA), conducted at the Center for Individual Development and Adaptive Education of Children at Risk (IDeA), Frankfurt/Main. The study was approved by the psychological and medical local ethical committees (Appendix A) and children and parents gave written informed consent prior to the start of study.

Children were eligible for participation either if they currently had a clinical ADHD diagnosis received from clinicians external to the study (ADHD group) or if they never had a clinical ADHD diagnosis (control group) before. Children were recruited from schools, local child psychiatric outpatient centers, and a clinic for child and adolescent psychiatry and the following inclusion criteria had to be met: (1) an IQ ≥ 70 on the Culture Fair Test (CFT 20-R; Weiß, 2006), (2) no pervasive developmental disorder, autism, or psychotic disorders, and (3) a birth weight ≥ 2500g.

Altogether, a total of 115 children (29 female; 47 with a clinical ADHD diagnosis) aged between 7 years and 4 months and 13 years and 8 months (M_{age} = 10.1 years, SD_{age} = 1.60 years) participated in this study together with one parent (11 fathers). However, clinical ADHD diagnoses might not always be valid (Bruchmüller, Margraf, & Schneider, 2012) and children recruited for the control group, yet undiagnosed, could also have ADHD. Therefore, the following procedure was used to the validity of group membership (see Rauch et al., 2012, for a similar procedure). Children were excluded from the ADHD group if they scored above of the cut-off point on less than two standardized ADHD screening or diagnosis instruments. The ADHD screening and diagnosis instruments included the Kinder-DIPS, a standardized clinical interview (Kinder-DIPS; Schneider, Unnewehr, & Margraf, 2009), the attention problem scale of the Child Behavior Checklist (CBCL; Arbeitsgruppe Deutsche Child Behavior Checklist, 1998; cut-off point: *T* score > 67), the hyperactivity scale of the Strengths and Difficulties Questionnaire (SDQ; Woerner, Becker, & Rothenberger, 2004; cut-off point: scale value > 5), and the overall scale of the German ADHD symptom list (FBB-ADHS; Döpfner, Lehmkuhl, & Steinhausen, 2006; cut-off point: stanine score > 6). In addition, parents of children who generally took medication to reduce ADHD symptoms were asked that their children should not take their medication within 24 hours before the study. However, being on medication during the study did not lead to an exclusion from participation. Regarding the control group, children were excluded if they scored above of the cut-off point on more than one of the standardized screening or diagnosis instruments for ADHD. Based on this procedure, 20 children (seven children with a previous ADHD

diagnosis) had to be excluded so that the final sample was reduced to $N = 95$ children (40 children with ADHD). Table 2 displays sample characteristics for both groups with regard to raw scores of ADHD symptoms, medication status, IQ, and SES. The latter was measured by a multi-dimensional aggregated index of parental education, parental occupation, and family income (DEGS1-Index; Lampert, Kroll, Müters, & Stolzenberg, 2013).

3.2. Procedure

Children and their parents attended two sessions in our laboratory rooms. Including breaks between the tasks Session I lasted about 2.5 hours and Session II about 2 hours (including breaks; see Table B2 in Appendix B for an overview of the applied tasks and questionnaires and their approximate duration in each session). When children and parents arrived at the laboratory rooms, they were greeted by two experimenters with one experimenter being assigned to the child and the other to the parent.

The procedure will be reported separately for children and parents. Children were videotaped and wore the triaxial GT3X ActiGraph (Pensacola, FL), a widely used accelerometer for assessing physical activity (Hänggi, Phillips, & Rowlands, 2013), during the tasks of both sessions. Specifically for Session I, children first worked on the CFT 20-R, to measure their intelligence. Afterwards children worked on various tasks assessing behavioral inhibition, delay aversion, sustained attention, and risky decision-making. Computerized tasks and behavioral tasks alternated to prevent children from boredom (see Table B2 in Appendix B). Subsequently, children provided a saliva sample for a molecular genetic analysis, answered the BIS/BAS questionnaire (Behavioral Inhibition Behavioral Activation Scales; Carver & White, 1994; Strobel, Beauducel, Debener, & Brocke, 2001), and worked on two parent-child interaction tasks. At the end of the session children received a certificate and a small present. During Session II children worked on various tasks assessing behavioral inhibition, delay aversion, temporal processing, and risky decision-making. As in Session I computerized tasks and behavioral tasks alternated to prevent children from being bored by the tasks (see Table B2 in Appendix B). Subsequently, children answered the Zurich Brief Questionnaire for the Assessment of Parental Behaviors (ZKE; Reitzle, Winkler Metzke, & Steinhausen, 2001) and participated in a delay of gratification task (Mischel & Baker, 1975). At the end of the session children received a certificate and a voucher equivalent to 20 €.

Table 2

Means and Standard Deviations in Parenthesis of the Main Characteristics of Children With ADHD and Control Children

Variables	ADHD (n = 40)	Control (n = 55)
Age		
Session I, years	10.36 (1.63)	9.93 (1.55)
Session II, years	10.43 (1.66)	9.95 (1.52)
Gender		
Male, %	82.50	69.09
IQ[a]	103.77 (14.73)	112.56 (14.16)
SES[b]	14.45 (3.74)	16.26 (3.31)
Clinical diagnosis[c]	77.50	0.00
CBCL		
Attention problems	8.28 (2.70)	1.90 (2.14)
SDQ		
Hyperactivity scale	7.23 (2.06)	1.84 (1.86)
FBB-ADHS		
Total score	1.67 (0.44)	0.39 (0.32)
Impulsivity	1.80 (0.75)	0.47 (0.49)
Hyperactivity	1.30 (0.68)	0.17 (0.25)
Inattention	1.91 (0.52)	0.53 (0.49)
ADHD symptoms[d]		
Composite score[d]	5.73 (1.37)	1.38 (1.27)
Medication		
Medication naïve, %	32.50	100.00
Medication Session I, %	50.00	0.00
Medication Session II, %	57.50	0.00

Note. [a] IQ scores derived from the CFT 20-R. [b] according to the DEGS1-Index. The index has a range of 3.0 – 21.0 and a high SES is indicated by values above 13.9. [c] according to the Kinder-DIPS. [d] composite measure of CBCL inattention problems, SDQ hyperactivity scale, and FBB-ADHS total score (see Section 3.5.1.).

Regarding the parents, an experimenter interviewed one parent at the beginning of Session I. The interview started with a short assessment of expressed emotion (Daley, Sonuga-Barke, & Thopmson, 2003) followed by the Kinder-DIPS, a diagnostic interview for mental disorders in childhood and adolescence (Schneider et al., 2009). Afterwards parents worked on various questionnaires (see Table B2 in Appendix B) screening first for children's ADHD symptoms and subsequently for parental psychopathology. The session closed with two parent-child interaction tasks, one problem solving task and one free play task. Session II began with another interview assessing the immigrant background and SES of the family followed by various questionnaires on parenting and child temperament (see Table B2 in Appendix B). The session closed with a short interview on parental problem solving (Hansen, Palotta, Christopher, Conaway, & Lundquist, 1995).

3.3. Measures

This section contains a description of all measures from the GIDeCA project that were used in the current investigation. These measures include questionnaires to assess continuous ADHD symptoms (Section 3.3.1.), computerized cognitive tasks to assess sustained attention (Section 3.3.2.) and behavioral inhibition (Section 3.3.3.) as well as behavioral tasks to assess risky decision-making (Section 3.3.4.). Computerized tasks were programmed in DirectRT® (Jarvis, 2008) and administered on a laptop computer. To respond, children pressed a left, blue button or a right, red button on an external DirectRT® button box that was placed between the child and the monitor. Throughout the tasks, the distance between the child and the monitor averaged approximately 50 cm. The experimenter observed the child during the practice trials but withdrew behind a partition wall during the experimental trials.

3.3.1. Continuous ADHD Symptom Measures

Continuous ADHD symptoms were assessed by parental ratings on the FBB-ADHS (Döpfner et al., 2006) as well as on the inattention subscale of the CBCL (Arbeitsgruppe Deutsche Child Behavior Checklist) and the hyperactivity subscale of the SDQ (Woerner et al., 2004).

FBB-ADHS. The FBB-ADHS is a German ADHD rating scale that has been developed in accordance with the ADHD symptoms described in the ICD-10. Thetotal FBB-ADHS scale consists of 20 items, which can be assigned to either an inattentive subscale (nine

items; e.g.,"has difficulties maintaining focus on one task"), a hyperactive subscale (seven items; e.g., "fidgets and squirms in her/his seat "), or an impulsive subscale (four items; e.g., "often interrupt conversations or others' activities"). All items are answered on a 4-point rating scale ranging from 0 (*not at all*) to 3 (*in particular*). In the current study, high internal consistencies were found for the FBB-ADHS total scale (α = .96), the inattentive subscale (α = .94), the impulsive subscale (α = .92), and the hyperactive subscale (α = .89).

CBCL inattention scale. The CBCL inattention scale primarily assess attentional problems (e.g., "Can't concentrate, can't pay attention for long"), however, the scale also includes items on hyperactivity (e.g., "Can't sit still, restless, or hyperactive") and more general problems (e.g., "Poor school work"). Altogether, the subscale consists of 11 items, which are answered on a 3-point scale: *not true*, *somewhat or sometimes true*, and *very true or often true*. Within the present study, the scale showed a good reliability, α = .81.

SDQ hyperactivity scale. The hyperactivity subscaleof the SDQ mainly focuses on children's hyperactivity (e.g., "restless, overactive, cannot stay still for long"), yet the scale also contains items related to deficits in sustained attention (e.g., "easily distracted, concentration wanders"). The subscale consists of five items, which are answered on a 3-point scale: *not true*, *somewhat true*, and *certainly true*. Regarding the present study, a high reliability of the subscale was revealed, α = .89.

3.3.2. Sustained Attention Task

A Continuous Performance Task (CPT) was used to specifically assess sustained attention. Yet, RTV and omission errors were not only computed from the CPT but also from other cognitive tasks (Go/No-Go, Flanker task) as measures for sustained attention (Shalev et al., 2011; see Section 2.4.1.).

Continuous Performance Task (CPT). The CPT was a modified version of a long-lasting computerized two alternatives forced choice task that had previously been applied in children with ADHD of a similar age range as in the current study (Gawrilow & Gollwitzer, 2008). Children repeatedly had to indicate whether a stimulus was a picture of an animal (i.e., cat, chicken, cow, mouse, pig) or a vehicle (i.e., car, plane, ship, train, truck). Stimuli were of approximately the same size, had been painted by a professional artist, and the distance between the child and the monitor averaged approximately 50 cm. At the beginning of the task, the experimenter told the children to respond as quickly and accurately as possible, while the same instructions were simultaneously presented on the computer screen.

Afterwards the children completed a practice block of ten trials. Each trial started with the presentation of a white fixation cross in the middle of a black screen. The fixation cross was randomly presented for 600 ms, 900 ms, or 1200 ms, and was immediately followed by the stimulus that appeared for a maximum duration of 550 ms. Children had a fixed 900 ms interval beginning from stimulus onset to indicate their response by pressing either the left, blue button (for animals) or the right, red button (for vehicles). During the practice block an 800 ms feedback was given whether the response was correct (green screen), false (red screen), or too slow (yellow screen) and subsequently the next trial started. The experimenter observed the entire practice block and if he or she had any doubts about whether the child understood the task, the practice block was repeated. The following experimental block consisted of 325 trials that were presented in a pseudo-randomized order securing an equal number of response repetitions and response switches. In contrast to the practice block, the maximum response duration was extended to 1550 ms and no feedback was given.

3.3.3. Behavioral Inhibition Tasks

Two tasks measured different forms of behavioral inhibition: (a) Withholding of responses was assessed by a Go/No-Go Task (Rauch et al., 2012), and (b) interference control was assessed by a Flanker task (Eriksen & Eriksen, 1974).

Go/No-Go Task. We used a modified version of a task that had previously been used in studies including children with ADHD of a similar age range as in the current study (Rauch et al., 2012). Whenever a Go stimulus (a picture of a chicken, a dog, a lion, or a sheep) appeared on the screen children had to press a button with their right index finger. However, when the No-Go stimulus (a picture of a goose) appeared on the screen, children were asked to refrain from responding. At the beginning of the task, the experimenter told the children to respond as quickly and accurately as possible, while the same instructions were simultaneously presented on the computer screen. Afterwards the children completed two practice blocks: In the “slow” practice block stimuli were presented for 1500 ms (ten trials), while in the “fast” practice block stimuli were presented for 450 ms (15 trials). Each trial started with the presentation of a white fixation cross in the middle of a black screen. The fixation cross was randomly presented for 600 ms, 900 ms, or 1200 ms, and was immediately followed by the stimulus. After stimulus onset children had a fixed interval of 2000 ms (in the slow condition) or 1550 ms (in the fast condition) to response. During practice blocks, green, red, or yellow screens were presented for 2000 ms after the response interval indicating

whether the response was correct, false, or too slow. Afterwards the next trial started. The experimenter observed the entire practice block and if he or she had any doubts about whether the child understood the task, the practice block was repeated. The final experimental block consisted of 300 trials, which were randomly presented under the condition that each stimulus occurred equally often resulting in a 20% No-Go rate. In contrast to the practice blocks, only fast trials were administered and no feedback on the performance was given.

Flanker Task. We used a modified version of the Eriksen flanker task (Eriksen & Eriksen, 1974). A central target arrow and two distractor arrows each at the left and at the right of the central arrow were always presented in the middle of the screen (e.g., > > > > >). Children were asked to indicate the direction of the central arrow, regardless of the direction of the distractor arrows. When the central arrow pointed to the left, children had to press the left, blue button, whereas they had to press a right, red button when the central arrow pointed to the right. At the beginning of the task, the experimenter told the children to respond as quickly and accurately as possible, while the same instructions were simultaneously presented on the computer screen. Afterwards the children completed a practice block (eight trials). Each trial started with the presentation of a white fixation cross in the middle of a black screen. The fixation cross lasted for 200 ms and was followed by a black screen for an additional 300 ms. Subsequently the stimulus appeared for a maximum duration of 500 ms and the children had 900 ms to respond beginning from stimulus onset. An 800 ms feedback was given whether the response was correct (green screen), false (red screen), or too slow (yellow screen) and then the next trial started. If a child did not understand the task or committed to many errors, the practice block was repeated. The final task consisted of 390 trials that were presented in a pseudo-randomized order securing an equal number of congruent (target and distractors pointing in the same direction) and incongruent (target and distractors pointing in different directions) trials and congruent (and incongruent) trials equally often following congruent and incongruent trials respectively. In contrast to the practice block, the maximum response duration was extended to 2000 ms and no feedback was given.

3.3.4. Risky Decision-Making Tasks

Recently, it has been argued that psychological research overly relied on measures of response times and questionnaires instead of measures of actual behavior (Baumeister, Vohs, & Funder, 2007). However, actual behavior might be more representative for behavior in real-world situations (Rozin, 2001) and a better predictor of future performance (Nisbett & Wilson, 1977) as compared to response times or questionnaires. Therefore, three risky decision-making tasks were administered to the children requiring actual behavioral responses similar to responses in real-world card games or games of dice. Children worked on the tasks in the presence of a trained experimenter who guided the children through the tasks and recorded the data.

Game of Dice Task (GDT). A behavioral version of the computerized GDT (Brand et al., 2005; see Section 2.3.1.) was developed. Each trial started with children betting on the outcome of a game of dice. Children could bet on a single number (e.g., that the number would be a one; chance 1:6), or on a combination of two numbers (e.g., that the rolled dice would show either a one or a two; chance 2:6), three numbers (e.g., that the rolled dice would show either a one, a two or a three; chance 3:6), or four numbers (e.g., that the rolled dice would be either a one, a two, a three or a four; chance 4:6). Different kinds of bets were associated with different amounts of gains and losses (e.g., ten, five, two, or one points) with less probable outcomes (riskier choices) being related to higher gains or losses of the same magnitude (see Figure 3). To ensure that children always had a positive amount of points regardless of any losses, children started with a point account of 180 points that was updated and mentioned to children after every trial. After the children had made their bet they rolled one dice. If the number on the dice corresponded to one of the numbers of the preceding bet children earned points, however, if the number on the dice did not correspond to one of the numbers of the preceding the bet, children lost points. Two practice trials familiarized the children with the task and various control questions were asked to ensure that the children understood the procedure (see Appendix B.3.). The following experiment block consisted of 18 trials.

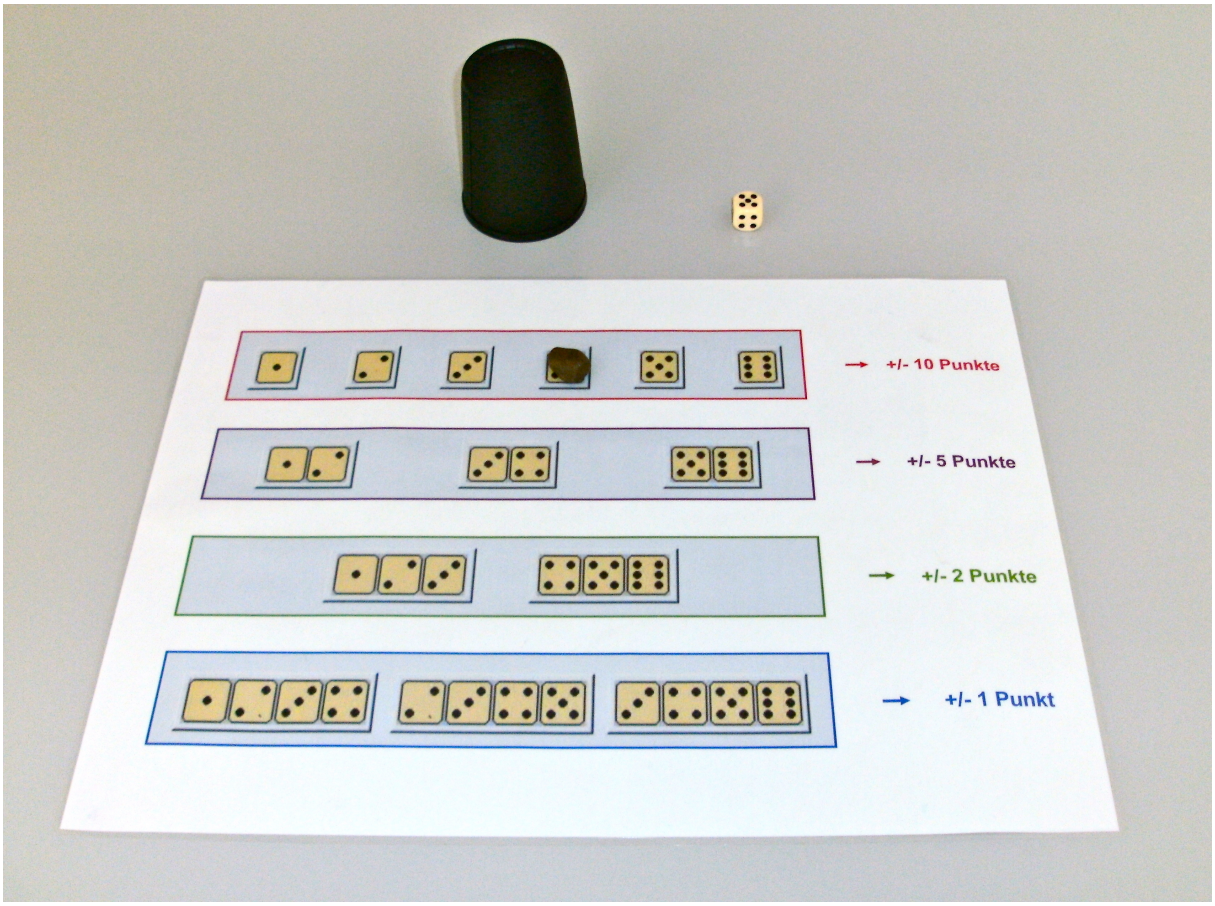

Figure 3. Example trial of the GDT. Children made their bets by placing a stone on one of these fields. The numbers at the right side indicated the corresponding gain or loss amounts. In this example, the child bet on the single number four, but lost as he/she rolled a five.

Columbia Card Task (CCT). A behavioral version of the computerized CCT (Figner et al., 2009) was developed. During each trial a child faced 12 cards turned upside down on a table (three rows of four cards each; see Figure 4). In each trial, there was a certain number of gain cards (10 or 11) and of loss cards (one or two). Children had to turn over at least one card per trial. For each gain card they turned over, they received as many points as were pictured on the card; however, when a loss card was encountered, the trial terminated and a specific loss amount was subtracted from the children's point account. Children could stop turning over cards anytime they liked or until a loss card was encountered. In addition to the number of gain and loss cards, the gain amount (one or five points) and the loss amount (-10 or -40 points) varied over trials. Gain amounts, loss amounts, and the number of loss cards for the current trial were displayed on a game board and the experimenter pointed out every change to that configuration. Two practice trials familiarized the children with the task and various standardized control questions were asked to ensure that the children understood the task (see Appendix B.3.). The final experiment consisted of 16 trials that were presented in a fixed order (see Table B1 in Appendix B). To ensure that children always had a positive amount of points regardless of any losses, children started with a point account of 400 points that was updated and mentioned to children after every trial.

No Six Task (NST). The NST was a self-developed behavioral risky decision-making task adopted from a popular game of dice (Ravensburger Schlag den Raab – Das Spiel®). Children were allowed to roll a dice as often as they liked and all the pips were counted out loud by the experimenter. A trial terminated if the children voluntarily stopped and claimed their payoff or if they rolled a six. In the latter case all points were lost. One practice trial familiarized the children with the task and a series of control questions were asked to ensure that the children understood the procedure (see Appendix B.3.). The final experiment consisted of ten trials.

Figure 4. Example trial of the CCT. The game board at the top displays the trial configuration. The gain amount is one point, the loss amount is -10, and there is one loss card in the deck. The child had successfully turned over three cards until the loss card was turned over.

3.5. Data Preparation

This section entails a description of the data preparation for the measures of ADHD symptoms, the computerized cognitive tasks (Section 4.1.1.) and the risky decision-making tasks (Section 4.1.2.), and the continuous measures of ADHD symptoms (Section 4.1.3). As the process of data preparation both includes the exclusion of participants and the calculation of composite scores, Table 3 offers a summary of the final sample sizes for each variable.

3.5.1. Data Preparation: Continuous ADHD Measures

The following procedure was applied to the three continuous measures of ADHD symptoms (CBCL attention problem scale, FBB-ADHS, SDQ hyperactivity scale). All raw data was entered into the statistical software IBM SPSS Statistics 22. Afterwards two independent coders checked whether the data was entered correctly. Sum scores were calculated for the CBCL attention problem scale and the SDQ hyperactivity scale. For each subscale of the FBB-ADHS as well as for the total FBB-ADHS scale mean scores were computed. Descriptive statistics are displayed in Table 2.

As correlations between the different measures were high (see Table 4), a composite score for continuous ADHD symptoms was computed by averaging the z-standardized scores of the CBCL inattention problem scale, the SDQ hyperactivity scale, and the FBB-ADHS total scale.

3.5.2. Data Preparation: Computerized Cognitive Tasks

The following procedure was applied to the three computerized cognitive tasks (CPT, Flanker, Go/No-Go). First, only experimental trials but not practice trials were analyzed. Second, first three trials of each task were considered as “warm-up” trials and were not analyzed (see Wendt & Luna-Rodriguez, 2009; Wendt et al., 2012). In addition, all trials with response times shorter than 150 ms were discarded from further analyses, as they were regarded as invalid responses (Wentura & Degner, 2010). Moreover, all cognitive tasks had a maximum response duration less or equal to 2000 ms, hence there was no need to eliminate outliers on the upper end of the response time distribution. Beyond eliminating outliers on a trial level, participants were excluded from further analyses on a task, if they (a) did not finish the task, (b) had an omission error rate larger than 50%, or (c) their combined rate of

inhibition and commission errors exceeded 50%. The final sample sizes for each task are listed in Table 3.

The following measures were calculated from each task to assess deficits in sustained attention: the rate of omission errors, the number of at least two omission errors in a row, and the variation coefficient of response times to represent RTV. However, while all hit trials from the Flanker and CPT were used to assess RTV, only correct Go trials were used to calculate RTV in the Go/No-Go task. In order to get a more reliable measure of sustained attention, a principle components analysis was conducted for the three measures of omission errors and the three measures of RTV (see Table 5). Both, the Minimum Average Partial test (MAP; O'Connor, 2000) and the scree plot revealed a one-factor solution; hence, the factor score was taken as a combined measure of sustained attention deficits.

Behavioral inhibition was assessed through the following measures: the rate of inhibition errors in the Go/No-Go task and the Flanker interference scores for errors, calculated as the difference in the error rate for incongruent Flanker trials and the error rate for congruent Flanker trials. However, as both measures did not correlate, $r(72) = .17$, $p = .884$, no composite score for behavioral inhibition was computed. Instead, both measures were analyzed separately. The rate of inhibition errors in the Go/No-Go task reflected deficits in withholding of responses, whereas the Flanker interference scores for errors represented deficits in interference control (for a discussion of the construct validity of both measures see Section 5.2.2.).

Table 3

Final Sample Sizes for Each Task (Number of Excluded Children in Parentheses)

Task	Control	ADHD	Total
Computerized Cognitive Tasks			
CPT	50 (5)	36 (4)	95
Flanker	48 (5)	33 (6)	92[a]
Go/No-Go	47 (5)	35 (4)	91[a,b]
Risky Decision-Making			
GDT	52 (0)	40 (0)	92[b]
CCT	53 (2)	39 (1)	95
NST	55 (0)	40 (0)	95

Note. . [a] missing data due to technical problems. [b] three children did not show up to Session II.

Table 4

Correlations Between the Continuous Measures of ADHD Symptoms

Measure	1	2	3	4	5
1. SDQ hyperactivity	-				
2. CBCL attention problems	.81***	-			
3. FBB-ADHS total	.86***	.82***	-		
4. FBB-ADHS inattention	.79***	.78***	.92***	-	
5. FBB-ADHS impulsivity	.71***	.67***	.86***	.67***	-
6. FBB-ADHS hyperactivity	.80***	.72***	.90***	.69***	.78***

Note. *** $p < .001$.

Table 5

Factor Component Loadings for the Three Measures of Omission Errors and the Three Measures of RTV

	Component 1
CPT RTV	.83
Flanker OE	.76
CPT OE	.75
Flanker RTV	.72
Go/No-Go RTV	.66
Go/No-Go OE	.58
Eigenvalue	3.13
Percentage of total variance	52.08
Number of test measures	6

3.5.3. Data Preparation: Risky Decision-Making Tasks

The following procedure was applied to all three risky decision-making tasks (GDT, CCT, NST). All raw data were entered into the statistical software IBM SPSS Statistics 22. Afterwards two independent coders checked whether the data was entered correctly. In case of implausible data, video recordings from the tasks were used for clarification. Three children had to be excluded from the CCT because they did not play all variations of gain amounts, loss amounts, and loss cards. The final sample sizes for each task are listed in Table 3.

Risky decision-making was assessed through the following measures similar to previous studies: the average number of cards turned over in the CCT (see Figner et al., 2009), the number of dice throws in the NST (equivalent to the number of cards turned over in the CCT), and the number of risky decisions in the GDT (Brand et al., 2005). The latter were defined by bets on single numbers or bets on combinations of two numbers, as these bets were associated with a negative expectancy value. Correlations between the three measures were weak, $r_{CCT\text{-}NST}(95) = .29$, $p = .004$, $r_{CCT\text{-}GDT}(92) = .22$, $p = .037$, $r_{GDT\text{-}NST}(92) = .22$, $p = .032$. Therefore, no composite score for risky decision-making was computed. Instead, risky decision-making was separately analyzed for each task.

4. Results

This chapter presents the results of the current thesis in the order of the four research aims. Pursuing Research Aim 1, Section 4.1. examined associations between sustained attention, behavioral inhibition and an ADHD diagnosis. Section 4.2. addresses on Research Aim 2 and describes associations between risky decision-making and an ADHD diagnosis, while Section 4.3. compares effect sizes for these associations with ADHD as a composite of continuous symptoms and ADHD as a categorical syndrome. Finally, Section 4.4. investigates whether associations between sustained attention, behavioral inhibition and ADHD are mediated by risky decision-making.

4.1. Research Aim 1 – Associations between Sustained Attention, Behavioral Inhibition and an ADHD Diagnosis

This section reports on associations between measures of sustained attention (Section 4.1.1.) and an ADHD diagnosis as well as on associations between measures of behavioral inhibition and an ADHD diagnosis (Section 4.1.2.). All analyses control for age but not for ADHD medication, as medication did not improve task performance (see Table C1 in Appendix C). However, the prescription of medication is related to the diagnosis of ADHD. Hence, as medication did not improve task performance, controlling for medication would control for ADHD or ADHD symptoms rather than for treatment effects on task performance.

4.1.1. Associations between Sustained Attention and an ADHD Diagnosis

Descriptive statistics for all measures of sustained attention are displayed in Table 6. However, to increase reliability and to prevent alpha error accumulation only the sustained attention deficit factor score of omission errors and RTVs (see Section 3.5.2.) was used to test for a deficit in sustained attention in children with ADHD. An analysis of covariance (ANCOVA) with the sustained attention deficit factor score as a dependent variable, ADHD diagnosis as a between factor, and age as a centered covariate yielded a significant effect for ADHD diagnosis, $F(1, 67) = 8.15$, $p = .006$, $\eta_p^2 = .108$, but no effect for age, demonstrating

that children with an ADHD diagnosis had higher scores on the sustained attention deficit factor (M_{ADHD} = 0.39, $M_{Control}$ = -0.26)[2].

4.1.2. Associations between Behavioral Inhibition and an ADHD Diagnosis

Descriptive statistics for the measures of behavioral inhibition are displayed in Table 6. Table 6 also depicts descriptive statistics for measures of conflict monitoring, as deficits in behavioral inhibition might be partly due to deficits in conflict-monitoring (see Section 2.1.1.). Analyses are presented separately for the Go/No-Go task and the Flanker task.

Go/No-Go. To analyze whether children with an ADHD diagnosis committed more inhibition errors than unaffected control children (M_{ADHD} = 6.18%, $M_{Control}$ = 5.68%, see Table 6), an ANCOVA was calculated with the inhibition error rate as dependent variable, ADHD diagnosis as a between factor and age as a centered covariate. No significant effects for age or ADHD diagnosis were found.

Flanker. To simultaneously investigate deficits in behavioral inhibition and conflict-monitoring in children with ADHD, response times and error rates were separately entered into a 2 x 2 x 2 mixed within- and between-subjects ANCOVA with the within factor Flanker congruency (congruent vs. incongruent), the within factor Flanker congruency in the preceding trial (congruent vs. incongruent), the between factor diagnosis (ADHD vs. control), and age as a centered covariate.

For response times, significant main effects were observed for congruency, $F(1, 78) = 36.29$, $p < .001$, $\eta_p^2 = .318$, and age, $F(1, 78) = 16.11$, $p < .001$, $\eta_p^2 = .171$, indicating slower response times for incongruent trials than for congruent trials (502 ms vs. 476 ms, congruency effect = 26 ms) and for younger children. In addition, a significant three-way interaction of congruency, congruency in the preceding trial, and diagnosis, $F(1, 78) = 4.01$, $p = .049$, $\eta_p^2 = .049$, indicated a reduced sequential congruency effect for children diagnosed with ADHD (difference between congruency effects if the preceding trial was congruent or incongruent was -12 ms) as compared to unaffected children (difference between congruency effects if the preceding trial was congruent or incongruent was 8 ms; see Figure 5) . No other main effects or interactions reached significance.

[2] A multivariate analysis of variance (MANCOVA) with all sustained attention variables from Table 6 as dependent variables and ADHD and age as predictors yielded a significant overall effect for ADHD diagnosis, $F(9, 76) = 3.73$, $p = .001$, $\eta_p^2 = .307$. For additional results regarding each single sustained attention measure, see Table C2 in Appendix C.

Table 6

Mean Performances (Standard Deviations in Parentheses) in the Cognitive and Behavioral Tasks for Children With ADHD and Control Children

Measures	Control	ADHD
Sustained Attention		
CPT OE, %	2.85 (4.72)	4.31 (5.00)
Go/No-Go OE, %	3.54 (5.41)	6.39 (7.19)
Flanker OE, %	5.45 (9.24)	7.87 (9.61)
CPT OE ≥ 2, n	0.54 (1.25)	1.67 (2.88)
Go/No-Go OE ≥ 2, n	1.23 (2.61)	3.06 (4.09)
Flanker OE ≥ 2, n	2.06 (4.55)	3.76 (5.81)
CPT RTV	0.34 (0.11)	0.41 (0.16)
Go/No-Go RTV	0.26 (0.07)	0.33 (0.10)
Flanker RTV	0.36 (0.17)	0.46 (0.19)
Factor score	-0.26 (0.78)	0.39 (1.25)
Behavioral Inhibition		
Go/No-Go IE, %	5.68 (3.30)	6.18 (3.64)
Flanker IFE, %	16.62 (12.01)	12.82 (10.95)
Conflict Monitoring,		
Flanker PCS, ms	5.70 (18.33)	0.74 (29.29)
Flanker PCE, %	-2.21 (5.60)	-0.48 (4.94)
Flanker SCRT, ms	11.84 (42.51)	-12.10 (59.28)
Flanker SCE, %	6.98 (7.86)	6.41 (10.81)
Risky Decision-Making		
GDT risky decision, n	6.96 (4.84)	10.43 (4.58)
CCT, n cards	4.40 (1.13)	4.65 (1.10)
NST, n dices	36.02 (12.62)	37.83 (13.73)

Note. IE = inhibition errors; IFE = interference for errors; OE = omission errors; OE ≥ 2 = at least two omission errors in a row; PCE = post-conflict errors; PCS = post-conflict slowing; RTV = response time variability; SCE = sequential congruency effect for errors; SCRT = sequential congruency effect for response times.

For error rates significant main effects were observed for congruency, $F(1, 78) = 125.10$, $p < .001$, $\eta_p^2 = .616$, and congruency in the preceding trial, $F(1, 78) = 5.45$, $p = .022$, $\eta_p^2 = .065$, indicating higher error rates for incongruent trials as compared to congruent trials in general (37.65% vs. 22.58%, congruency effect = 15.07 percentage points) as well as lower error rates after incongruent trials as after congruent trials (30.89% vs. 29.34%, post-conflict error rate effect = 1.51 percentage points). A significant interaction between congruency and congruency in the preceding trial, $F(1, 78) = 40.64$, $p < .001$, $\eta_p^2 = .343$, revealed a sequential congruency effect (difference in congruency effects if the preceding trial was congruent and if the preceding trial was incongruent was 6.81 percentage points). The marginally significant three-way interaction of congruency, congruency in the preceding trial, and age $F(1, 78) = 2.82$, $p = .097$, $\eta_p^2 = .035$, indicated that this sequential congruency effect was reduced for younger children. No other main effects or interactions reached significance.

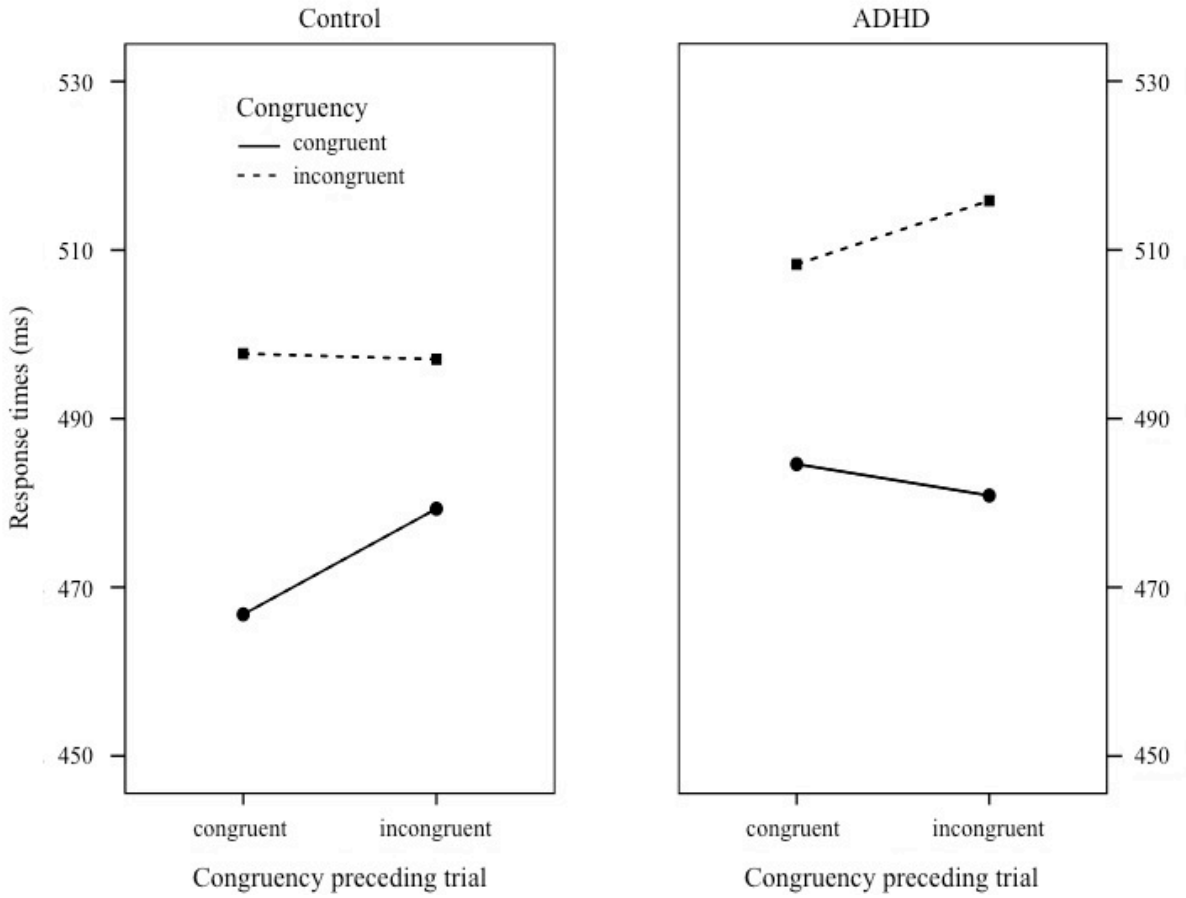

Figure 5. Flanker Effects for Response Times. Mean response times for congruent and incongruent trials as a function of congruency in the preceding trial for control children and children with ADHD. While control children showed the typical sequential congruency effect as indicated by a reduced Flanker congruency effect after incongruent trials (left panel), no such an effect was found for children with ADHD. Effects are shown for a centered age value of age_{cent} = 0.09 years.

4.2. Research Aim 2 – Associations between Risky Decision-Making and an ADHD diagnosis

This section reports on associations between measures of risky decision-making and an ADHD diagnosis. As no composite score of risky decision-making could be calculated (see Section 3.5.3.), separate analysis were conducted for each risky decision-making task (GDT, CCT, NST). All analyses control for age but not for medication, as medication did not improve task performance. Descriptive statistics for the task performance in each risky decision-making task are displayed in Table 6.

GDT. To analyze whether children with an ADHD diagnosis played more risky in the GDT than unaffected control children, an ANCOVA was calculated with the number of risky decisions as dependent variable, ADHD diagnosis as a between factor and age as a centered covariate. There were no effects of age, however, as evident by a significant main effect of diagnosis, $F(1, 89) = 11.51$, $p = .001$, $\eta_p^2 = .115$, children with ADHD made on average a higher number of risky decisions ($n = 10.43$) as compared to unaffected children ($n = 6.96$).

CCT. To analyze whether children with an ADHD diagnosis played more risky in the CCT than unaffected control children, an ANCOVA was calculated with the average number of cards turned over as the dependent variable, ADHD diagnosis as a between factor and age as a centered covariate. No significant effects for age or ADHD diagnosis were found.

NST. To analyze whether children with an ADHD diagnosis played more risky in the NST than unaffected control children, an ANCOVA was calculated with the number of dice throws in the NST as the dependent variable, ADHD diagnosis as a between factor and age as a centered covariate. No significant effects for age or ADHD diagnosis were found.

4.3. Research Aim 3 – ADHD as a Composite of Continuous Symptoms vs. ADHD as a Categorical Syndrome

This section addresses whether associations between sustained attention, behavioral inhibition, and risky decision-making are stronger for continuous ADHD symptoms than for a categorical ADHD diagnosis. Therefore, all analyses conducted to analyze Research Aim 1 and Research Aim 2 were repeated with the composite score of continuous ADHD symptoms instead of the categorical ADHD diagnosis. The composite score of continuous ADHD symptoms was chosen over the single ADHD symptoms scales (SDQ, CBCL, and FBB-

ADHS total score), as a composite score is more reliable than single scales[3]. To further investigate whether associations were particularly strong for symptoms of inattention and measures of sustained attention on the one hand and for associations between symptoms of impulsivity and measures of behavioral inhibition on the other hand, the analyses were again repeated with the inattentive, impulsive, and hyperactive subscale of the FBB-ADHS instead of the composite score of continuous ADHD symptoms. Results for the composite score of continuous ADHD symptoms (see Section 3.5.1) are reported in Section 4.3.1., whereas results for the subscales of the FBB-ADHS are reported in Section 4.3.2..

4.3.1. Associations Between Cognitive and Behavioral Performance Measures and a Composite Score of Continuous ADHD Symptoms

This section examines the association between measures of sustained attention, behavioral inhibition, conflict monitoring, risky decision-making and a composite score of continuous ADHD symptoms. All analyses controlled for age but not for medication, as medication did not improve task performance.

Sustained Attention. A general linear model (GLM) was calculated with the sustained attention deficit factor score as a dependent variable and the composite score of continuous ADHD symptoms and age as centered predictors. A significant effects was revealed for ADHD symptoms, $F(1, 67) = 20.10$, $p < .001$, $\eta_p^2 = .231$, but not for age indicating worse sustained attention children with more or stronger ADHD symptoms.

Go/No-Go. A GLM was calculated with the inhibition error rate as a dependent variable and the composite score of continuous ADHD symptoms and age as centered predictors. No significant effects for age or ADHD symptoms were found.

Flanker. To simultaneously investigate whether children with an ADHD diagnosis exhibited a larger Flanker interference effect or a reduction in conflict-monitoring as compared to unaffected children response times and error rates were separately entered into a 2 x 2 repeated measures ANCOVA with the within factors Flanker congruency (congruent vs. incongruent) and Flanker congruency in the preceding trial (congruent vs. incongruent), and age and the composite score of continuous ADHD symptoms as centered covariates.

For response times significant main effects were observed for congruency, $F(1, 78) = 37.07$, $p < .001$, $\eta_p^2 = .322$, and age, $F(1, 78) = 14.92$, $p < .001$, $\eta_p^2 = .161$, indicating

[3] Results for the single ADHD symptom scales (SDQ, CBCL, FBB-ADHS total scale) are reported in Appendix C.3..

slower response times for incongruent trials as compared to congruent trials (502 ms vs. 476 ms, congruency effect = 26 ms) and for younger children. No other main effects or interactions reached significance.

For error rates significant main effects were observed for congruency, $F(1, 78) = 134.81$, $p < .001$, $\eta_p^2 = .633$, congruency in the preceding trial, $F(1, 78) = 7.18$, $p = .009$, $\eta_p^2 = .084$, and ADHD symptoms, $F(1, 78) = 10.47$, $p = .002$, $\eta_p^2 = .118$, indicating higher error rates for incongruent trials than for congruent trials (37.65% vs. 22.58%, congruency effect = 15.07 percentage points) and children with more or stronger ADHD symptoms as well as a reduced error rate after incongruent trials (30.89% vs. 29.34%, post-conflict error rate effect = 1.51 percentage points). In addition, a significant interaction effect was found for congruency and congruency in the preceding trial, $F(1, 78) = 45.75$, $p < .001$, $\eta_p^2 = .370$, indicating a sequential congruency effect (difference in congruency effects if the preceding trial was congruent and if the preceding trial was incongruent was 6.81 percentage points). Marginally significant three-way interactions demonstrated that this sequential congruency effect tended to be further modulated by age, $F(1, 78) = 3.80$, $p = .055$, $\eta_p^2 = .046$, and ADHD symptoms, $F(1, 78) = 4.52$, $p = .076$, $\eta_p^2 = .040$. Younger children and children with more or stronger ADHD symptoms tended to exhibit a smaller sequential congruency effect. Furthermore, a marginally significant interaction was observed for congruency in the preceding trial and ADHD symptoms, $F(1, 78) = 2.97$, $p = .089$, $\eta_p^2 = .037$, indicating that error rates for children with more or stronger ADHD symptoms were less effected by the congruency in the preceding trial, whereas children with less or weaker ADHS symptoms displayed a reduced error if the preceding trial was incongruent as if the preceding trial was congruent. No other main effects or interactions were found.

GDT. To analyze whether children with more or stronger ADHD symptoms played more risky in the GDT, a GLM was calculated with the number of risky decisions as dependent variable and the composite score of continuous ADHD symptoms and age as centered predictors. Results yielded a significant effect of ADHD symptoms, $F(1, 78) = 10.06$, $p = .002$, $\eta_p^2 = .102$, with stronger affected children making a higher number of risky decisions.

CCT. To analyze whether children with more or stronger ADHD symptoms played more risky in the CCT, a GLM was calculated with the average number of cards turned over as the dependent variable and the composite score of continuous ADHD symptoms and age as centered predictors. No significant effects for age or ADHD symptoms were found.

NST. To analyze whether children with more or stronger ADHD symptoms played more risky in the NST, a GLM was calculated with the number of dice throws in the NST as the dependent variable and the composite score of continuous ADHD symptoms and age as centered predictors. No significant effects for age or ADHD symptoms were found.

4.3.2. Associations Between Cognitive and Behavioral Performance Measures and the ADHD Symptom Scales of the FBB-ADHS

This section examines the association between measures of sustained attention, behavioral inhibition, conflict monitoring, risky decision-making and the three subscales of inattentive symptoms, impulse symptoms, and hyperactive symptoms of the FBB-ADHS. All analyses controlled for age but not for medication, as medication did not improve task performance.

Sustained Attention. Separate GLMs were calculated for the three subscales with the sustained attention deficit factor score as a dependent variable and the FBB-ADHS subscale scores and age as centered predictors.

Regarding the inattentive subscale, only ADHD inattentive symptoms, $F(1, 67) = 9.51$, $p = .003$, $\eta_p^2 = .124$, predicted sustained attention indicating that children with higher scores on the FBB-ADHS inattention subscale exhibited stronger deficits in sustained attention.

In addition, for the FBB-ADHS impulsivity subscale an effect of ADHD impulsive symptoms, $F(1, 67) = 6.13$, $p = .016$, $\eta_p^2 = .084$, but not of age was found indicating stronger deficits in sustained attention in more impulsive children.

Finally, regarding the FBB-ADHS hyperactive subscale a significant effect of ADHD hyperactive symptoms, $F(1, 67) = 11.94$, $p = .001$, $\eta_p^2 = .151$, was observed revealing stronger deficits in sustained attention in children with higher hyperactivity scores.

Go/No-Go. Separate GLMs were calculated for the three ADHD subscales with the inhibition error rate as a dependent variable and the FBB-ADHS subscale scores and age as centered predictors. No significant effects for any of the FBB-ADHS subscales or age were found.

Flanker. To simultaneously investigate whether children with higher scores on the FBB-ADHS subscales exhibited a larger Flanker interference effect or a reduction in conflict-monitoring as compared to unaffected children, for each subscale response times and error rates were separately entered into a 2 x 2 repeated measures ANCOVA with the within factors

Flanker congruency (congruent vs. incongruent) and Flanker congruency in the preceding trial (congruent vs. incongruent), and age and the FBB-ADHS subscale scores as centered covariates.

Regarding the FBB-ADHS inattentive subscale and response times significant main effects were observed for congruency, $F(1, 78) = 36.77$, $p < .001$, $\eta_p^2 = .320$, and age, $F(1, 78) = 15.10$, $p < .001$, $\eta_p^2 = .162$, indicating slower response times for incongruent trials as compared to congruent trials (502 ms vs. 476 ms, congruency effect = 26 ms) and for younger children. No other main effects or interactions reached significance.

For the FBB-ADHS inattentive subscale, significant main effects on error rates were observed for congruency, $F(1, 78) = 133.63$, $p < .001$, $\eta_p^2 = .631$, congruency in the preceding trial, $F(1, 78) = 6.80$, $p = .011$, $\eta_p^2 = .080$, and the FBB-ADHS inattentive subscale, $F(1, 78) = 7.67$, $p = .007$, $\eta_p^2 = .089$, indicating higher error rates for incongruent trials as compared to congruent trials (37.65% vs. 22.58%, congruency effect = 15.07 percentage points), a reduced error rate after incongruent trials (30.89% vs. 29.34%, post-conflict error rate effect = 1.51 percentage points), and a generally increased error rate in children with higher scores on the FBB-ADHS inattentive subscale. In addition, a significant interactions effect was found for congruency and congruency in the preceding trial, $F(1, 78) = 44.07$, $p < .001$, $\eta_p^2 = .361$, revealing a sequential congruency effect (difference in congruency effects if the preceding trial was congruent and if the preceding trial was incongruent was 6.81 percentage points). No other main effects or interaction effects were found.

Regarding the FBB-ADHS impulsive subscale and response times significant main effects were observed for congruency, $F(1, 78) = 37.12$, $p < .001$, $\eta_p^2 = .322$, and age, $F(1, 78) = 16.34$, $p < .001$, $\eta_p^2 = .173$, indicating slower response times for incongruent trials than for congruent trials (502 ms vs. 476 ms, congruency effect = 26 ms) and for younger children. No other main effects or interactions reached significance.

For the FBB-ADHS impulsive subscale, significant main effects on error rates were observed for congruency, $F(1, 78) = 134.44$, $p < .001$, $\eta_p^2 = .633$, and congruency in the preceding trial, $F(1, 78) = 6.98$, $p = .010$, $\eta_p^2 = .082$, indicating higher error rates for incongruent trials than for congruent trials (37.65% vs. 22.58%, congruency effect = 15.07 percentage points) and a reduced error rate after incongruent trials (30.89% vs.29.34%, post-conflict error rate effect = 1.51 percentage points). In addition, a significant interactions effect was found for congruency and congruency in the preceding trial, $F(1, 78) = 45.10$, $p < .001$, $\eta_p^2 = .366$, revealing a sequential congruency effect (difference in congruency effects if the

preceding trial was congruent and if the preceding trial was incongruent was 6.81 percentage points). No other main effects or interaction effects were observed.

Regarding the FBB-ADHS hyperactivity subscale and response times significant main effects were observed for congruency, $F(1, 78) = 36.90$, $p < .001$, $\eta_p^2 = .321$, and age, $F(1, 78) = 16.41$, $p < .001$, $\eta_p^2 = .172$, indicating slower response times for incongruent trials as compared to congruent trials (502 ms vs. 476 ms, congruency effect = 26 ms) and for younger children. In addition, a significant interaction between congruency in the preceding trial and the FBB-ADHS hyperactivity score, $F(1, 78) = 5.75$, $p = .019$, $\eta_p^2 = .069$, revealed less post-conflict slowing in children with more or stronger ADHD hyperactivity symptoms. No other main effects or interactions reached significance.

For the FBB-ADHS hyperactivity subscale, significant main effects on error rates were observed for congruency, $F(1, 78) = 133.84$, $p < .001$, $\eta_p^2 = .632$, congruency in the preceding trial, $F(1, 78) = 6.79$, $p = .011$, $\eta_p^2 = .080$, and the FBB-ADHS hyperactivity subscale, $F(1, 78) = 4.12$, $p = .046$, $\eta_p^2 = .050$, indicating higher error rates for incongruent trials than for congruent trials (37.65% vs. 22.58%, congruency effect = 15.07 percentage points), a reduced error rate after incongruent trials (30.89% vs. 29.34%, post-conflict error rate effect = 1.51 percentage points), and a generally pronounced error rate in children with higher scores on the FBB-ADHS total scale. In addition, a significant interactions effect was found for congruency and congruency in the preceding trial, $F(1, 78) = 44.68$, $p < .001$, $\eta_p^2 = .364$, revealing a sequential congruency effect (difference in congruency effects if the preceding trial was congruent and if the preceding trial was incongruent was 6.81 percentage points). As indicated by a marginally significant three-way interaction between congruency, congruency in the preceding trial and the FBB-ADHS hyperactivity subscale, $F(1, 78) = 3.28$, $p = .074$, $\eta_p^2 = .040$, this sequential congruency effect was less pronounced in children with more or stronger ADHD hyperactivity symptoms. In addition, a marginally significant interaction between congruency in the preceding trial, $F(1, 78) = 2.98$, $p = .088$, $\eta_p^2 = .037$, indicated a smaller error reduction after incongruent trials for children with higher hyperactivity scores. No other main effects or interaction effects were found.

GDT. Separate GLMs were calculated for the three ADHD subscales with the number of risky decisions as dependent variable and the FBB-ADHS subscale scores and age as centered predictors. Significant effects were revealed for all three subscales, in particular the inattentive subscale, $F(1, 89) = 5.144$, $p = .022$, $\eta_p^2 = .058$, the impulsive subscale, $F(1, 89) = 4.90$, $p = .029$, $\eta_p^2 = .052$, and the hyperactive subscale, $F(1, 89) = 9.41$, $p = .003$,

η_p^2 = .097. Children with more or stronger ADHD symptoms made a higher number of risky decisions. No effects of age were observed.

CCT. Separate GLMs were calculated for the three subscales with the number of cards turned over as dependent variable and the FBB-ADHS subscale scores and age as centered predictors. No significant effects for any of the FBB-ADHS subscales or age were found.

NST. Separate GLMs were calculated for the three subscales with the number of dice throws as dependent variable and the FBB-ADHS subscale scores and age as centered predictors. No significant effects for any of the FBB-ADHS subscales or age were found.

4.4. Research Aim 4 – Risky Decision Making as a Mediator: Indirect Effects of Cognitive Endophenotypes on ADHD

This section examines possible mediation effects of the cognitive endophenotypes on ADHD symptoms though risky decision-making. In order to claim a mediation effect this indirect effect and the total effect of cognitive endophenotypes on ADHD symptoms need to be significant (Mathieu & Taylor, 2006). However, an indirect effect can be present even in the absence of a total effect (Preacher & Hayes, 2004; Rucker, Preacher, Tormala, & Petty, 2011). Therefore, three separate mediation models were calculated for sustained attention, behavioral inhibition, and conflict-monitoring. Sustained attention was operationalized through the sustained attention deficit factor score, behavioral inhibition through the inhibition error rate in the Go/No-Go task, and conflict monitoring as the sequential congruency effect for errors in the Flanker task. Risky decision-making as the mediator was operationalized through the number of risky decisions in the GDT, and ADHD symptoms as the dependent variable were operationalized through the continuous ADHD composite score. All analyses controlled for age but not for medication, as medication did not improve the performance in these tasks. The resulting mediation models were analyzed in IBM SPSS 22 using Model 4 of the SPSS macro PROCESS (Hayes, 2013). A graphical presentation of all three mediation models is displayed in Figure 6.

Sustained Attention. Sustained attention, as assessed by the sustained attention deficit factor score, predicted both ADHD symptoms, b = 1.22, $t(67)$ = 4.48, $p < .001$, and risky decision-making, b = 1.84, $t(67)$ = 3.46, $p < .001$. Children with more severe inattention problems had more or stronger ADHD symptoms and made a higher number of risky decisions in the GDT. When ADHD symptoms were predicted simultaneously by sustained attention and risky decision-making, sustained attention remained significant, b = 1.03,

$t(67) = 3.53$, $p = .001$, while risky decision-making tended to remain significant as well, $b = 0.10$, $t(67) = 1.67$, $p = .100$. A bias-corrected bootstrap 90% confidence interval (CI) based on 20.000 bootstrap samples indicated a marginally significant indirect effect of sustained attention on ADHD symptoms through risky decision-making, $b = 0.19$, 90% CI: [0.01, 0.41].

Behavioral Inhibition. Behavioral inhibition assessed by the inhibition error rate in the Go/No-Go task did not predict ADHD symptoms, $b = 7.44$, $t(79) = 0.90$, $p = .373$, but risky decision-making, $b = 49.67$, $t(79) = 3.27$, $p = .002$. Children, who committed more inhibition errors in the Go/No-Go task made a higher number of risky decisions in the GDT. When ADHD symptoms were predicted simultaneously by behavioral inhibition and risky decision-making, risky decision-making remained significant, $b = 0.17$, $t(79) = 2.94$, $p = .004$, whereas behavioral inhibition did not, $b = -1.13$, $t(79) = -0.13$, $p = .894$. A bias-corrected bootstrap 99% CI based on 20.000 bootstrap samples indicated that the indirect effect of behavioral inhibition on ADHD symptoms through risky decision-making was significant, $b = 8.57$, 99% CI: [1.38, 21.20].

Conflict-Monitoring. Conflict-monitoring assessed by the sequential congruency effect for errors in the Flanker task tended to predict both ADHD symptoms, $b = -6.12$, $t(75) = -1.98$, $p = .052$, and risky decision-making, $b = -10.27$, $t(75) = -1.70$, $p = .094$. Children with a smaller sequential congruency effect for errors had more or stronger ADHD symptoms and made a higher number of risky decisions. When ADHD symptoms were predicted simultaneously by conflict-monitoring and risky decision-making, risky decision-making remained significant, $b = 0.17$, $t(75) = 3.00$, $p = .004$, whereas conflict-monitoring did not, $b = -4.39$, $t(75) = -1.46$, $p = .148$. A bias-corrected bootstrap 95% CI based on 20.000 bootstrap samples indicated no indirect effect of conflict-monitoring through risky decision-making, $b = -1.73$, 95% CI: [-4.86, 0.33].

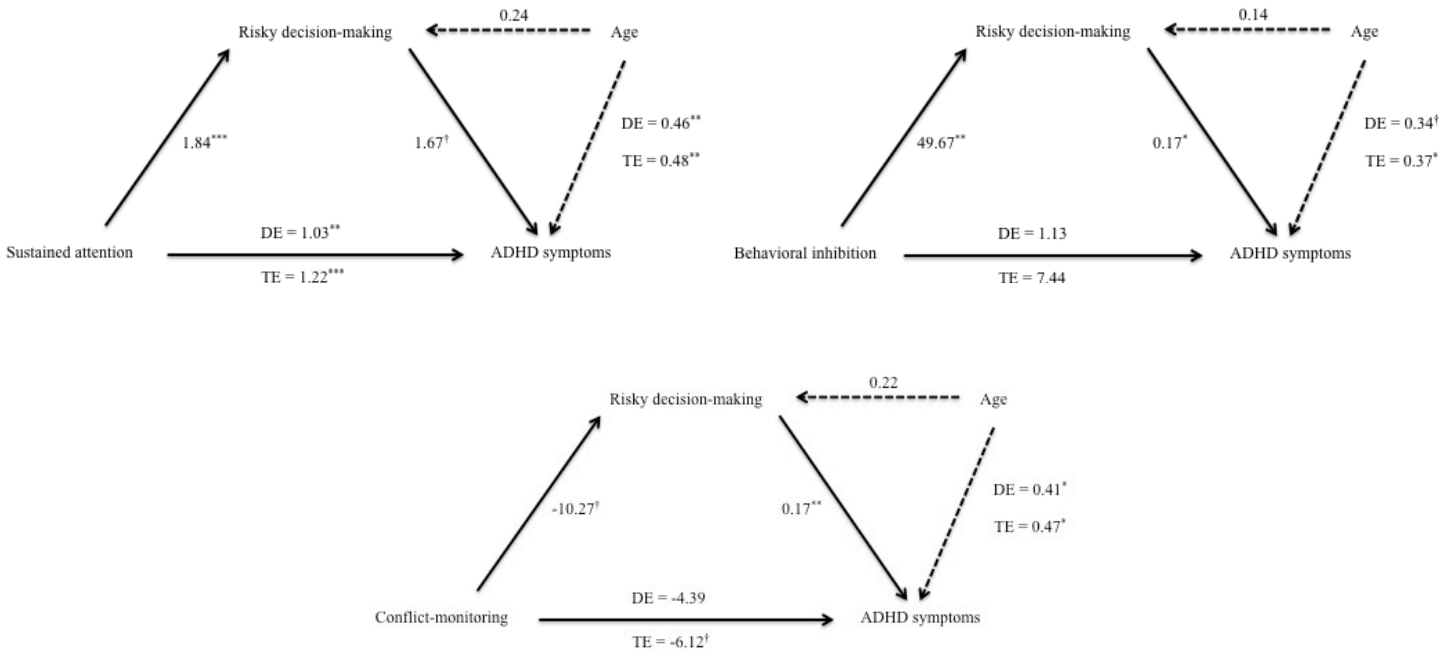

Figure 6. Mediation Models. Risky decision-making as a mediator of the associations between ADHD symptoms and sustained attention (left), behavioral inhibition (right), and conflict-monitoring (bottom); TE = total effect; DE = direct effect; dashed lines indicate covariates; † $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$.

5. Discussion

Research on ADHD is an interdisciplinary field and researchers from psychology, neuroscience, genetics, medicine, and social sciences have contributed to our understanding of the disorder. However, although ADHD is a heterogeneous disorder and no single research area can explain the etiology and pathogenesis of ADHD on its own, results from the different disciplines have only rarely been related to each other. Hence, a research framework, which incorporates psychological, neuronal, genetic, and social-environmental factors, should be especially useful for the development of comprehensive theories of ADHD. Accordingly, the main purpose of the theoretical part of this thesis was the development of a research framework that could guide researchers in planning studies, choosing measures, and selecting research questions to investigate ADHD (Imenda, 2014; Wacker, 1998).

The presented framework is based on the endophenotype concept (Cannon & Keller, 2006; Gottesman & Gould, 2003; Miller & Rockstroh, 2013) and assumes that genetic polymorphisms are not directly related to ADHD, instead gene polymorphisms alter protein synthesis and thereby impact on neuronal circuits. Neuronal circuits in turn are related to basal cognitive functions (the endophenotypes) that underline the disorder. However, the presented framework moves beyond a mere endophenotype approach by postulating that endophenotypes, similar to genetics or neuronal circuits, are not directly related to ADHD either. Instead they are related to higher-order cognitive functions that might be facets of ADHD symptoms. This distinction is important, as different endophenotypes might not equally relate to each ADHD symptom dimension (inattention, impulsivity, hyperactivity). In addition, ADHD symptom dimensions are not homogenous constructs, but constructs that consist of various facets and facets of the even the same symptom dimension might be differentially related to various endophenotypes (see Wittmann & Klumb, 2006). Hence, analyzing associations of endophenotypes with higher-order psychological functions could be beneficial for the understanding of ADHD. Finally, environmental factors are supposed to interact at each level of the framework. In particular, situation non-specific environmental factors are primarily supposed to influence neuronal circuits and long-term developments of psychological functions, whereas situation specific factors are primarily supposed to influence whether deficits in psychological functions lead to symptoms of ADHD in specific situations.

Several research questions can be inferred from the presented framework. For instance, it could be investigated which basal psychological functions could be regarded as endophenotypes, whether these functions independently contribute to ADHD, or whether these functions are related to all ADHD symptoms alike. It could be examined, how

impairments in psychological endophenotypes relate to deficits in higher-order psychological functions and whether these impairments in higher-order psychological functions mediate associations of endophenotypes and ADHD symptoms. In addition, situation non-specific environmental factors could be analyzed with regard to their influence on neuronal circuits and endophenotypes on the one hand, whereas situation specific environmental factors could be analyzed with regard to their influence on ADHD symptoms in specific situations. The latter could be especially interesting for analysis of resilience factors or compensatory strategies a child or adult with ADHD might have learnt to use (Merkt, 2014). Furthermore, situation specific environmental factors might explain day-to-day variability in ADHD symptoms (Schmid, 2014) and help to create interventions to improve ADHD symptoms in daily life.

The current thesis aimed at investigating four possible research questions inspired by the presented framework. First, associations of sustained attention, behavioral inhibition, and conflict-monitoring with the categorical diagnosis of ADHD were examined in order to compare their strength and to establish sustained attention as a core deficit of children with ADHD. Second, it was investigated whether risky decision-making as a facet of impulsivity relates to ADHD. Next, it was compared whether associations between sustained attention, behavioral inhibition, conflict-monitoring, and risky decision-making with ADHD were higher for ADHD symptoms than for the categorical diagnosis. Finally, as the framework assumes that endophenotypes are only indirectly related to ADHD symptoms through higher-order psychological functions, indirect effects of sustained attention, behavioral inhibition, and conflict monitoring on ADHD symptoms through risky decision-making were tested.

This chapter summarizes and discusses the results presented in Chapter 4. Section 5.1. addresses results concerning the specific research aims while Section 5.2. evaluates limitations of the current investigation. Finally, implications for future research on ADHD and clinical practice are discussed in Sections 5.3. and 5.4, respectively.

5.1. Discussion of Results Concerning the Four Research Aims

5.1.1. Research Aim 1: Associations Between Sustained Attention, Behavioral Inhibition, Conflict-Monitoring and a Categorical ADHD Diagnosis

The first research aim of this thesis was to investigate the associations of measures of sustained attention, behavioral inhibition, and conflict-monitoring with the categorical diagnosis of ADHD and compare the strength of these associations. The results revealed a deficit in sustained attention in children with ADHD as compared to unaffected children. An additional but weaker deficit was found for conflict monitoring, whereas children with ADHD exhibited no deficit in behavioral inhibition relative to children without ADHD.

This strong impairment in sustained attention is consistent with previous studies and meta-analyses, in which children with ADHD had higher omission error rates, committed two or more omission errors in a row more often, and displayed a higher RTV than unaffected control children (Helps et al., 2011; Karalunas, Geurts, Konrad, Bender, & Nigg, 2014; Kofler et al., 2013; Metin et al., 2012; Tamm et al., 2012). However, these findings could be explained by at least two different, but not excluding theoretical accounts. First, the results are in line with theories explaining a larger RTV and higher omission error rates by the presence of periodic attentional lapses (Leth-Steensen, Elbaz, & Douglas, 2000; Sonuga-Barke & Castellanos, 2007; Weissman et al., 2006). According to the DMN hypothesis, periodic attentional lapses occur at low frequency bands of response times due to a failure to suppress activity of the DMN and a simultaneously reduced activity of the task positive network (Castellanos, Kelly, & Milham, 2009; Sonuga-Barke & Castellanos, 2007).

However, the DMN hypothesis has recently been challenged (Karalunas et al., 2014; Karalunas, Huang-Pollock, & Nigg, 2013) as a higher RTV was associated with both very low and high frequency bands of response times (Karalunas et al., 2013). Similarly, both hypo- and hyperactivation of the DMN have been proposed for ADHD and disagreement exists whether differences in DMN activity between children with and without ADHD are present at rest (Konrad & Eickhoff, 2010). Moreover, attentional lapses merely define an event and besides a failure to suppress DMN activity, various processes might contribute to a higher RTV, as for instance, stimulus encoding, processing speed, speed-accuracy trade-offs, post-error slowing, motor preparation, response execution, effort and arousal (Karalunas et al., 2014).

Focusing on arousal, a second explanation of higher RTV in ADHD has been derived from diffusion models. Meta-analyses of diffusion models revealed a slower drift rate in

children with ADHD as compared to unaffected children (Huang-Pollock et al., 2012; Karalunas et al., 2014). Like the signal sensitivity parameter *d'* (Ratcliff & McKoon, 2008), drift rates can be interpreted as an index of arousal (Karalunas et al., 2014; Sanders, 1983; van der Meere & Sergeant, 1988) and in line with the Petersen-Posner perspective on brain networks involved in sustained attention (Petersen & Posner, 2012; Posner & Petersen, 1990), arousal is responsible for both establishing and maintaining an alert stage. From a neurobiological perspective, optimal arousal and better sustained attention are accounted for by phasic norepinephrine activation of the LC, whereas a less phasic and more tonic norepinephrine activation of the LC is associated with less optimal arousal and higher distractibility (Aston-Jones & Cohen, 2005; Langner & Eickhoff, 2013; Petersen & Posner, 2012; Posner & Petersen, 1990; Sarter, 2001). Pharmacological interventions further support the account, as methylphenidate and especially atomoxetine, two of the most common pharmacological substances for the treatment of ADHD, influence norepinephrine transmission and reuptake (Bari & Aston-Jones, 2013; Hannestad et al., 2010).

While non-optimal arousal is a promising alternative explanation to a failure to suppress DMN activity, several aspects of the account can be challenged. First, the phasic/tonic dynamics of norepinephrine and the LC activity have recently been transferred to the DMN hypothesis (Aboitiz, Ossandón, Zamorano, Palma, & Carrasco, 2014). Within this account the DMN was associated with tonic catecholamine activity in a sense that a deregulated tonic activity results in an unbalanced DMN leading to a higher distractibility (Aboitiz et al., 2014). Second, the account strongly relies on the interpretation of drift rates obtained from diffusion models. However, it is unclear whether cognitive tasks like the Flanker task, or CPT really meet the criteria for a diffusion model analysis. For instance, the diffusion model approach assumes a continuous sampling process of information (Ratcliff & McKoon, 2008; Voss, Nagler, & Lerche, 2013). While this assumption is obviously plausible for ambivalent visual stimuli like fields of pixels with different colors used in brightness or color-discrimination tasks (Ratcliff, 2002), it is less clear whether there is a continuous sampling process in typical cognitive tasks like the Flanker task, or CPT. Yet, even if a continuous sampling process could be assumed various diffusion models exist to model decision processes in the Flanker task (Hübner et al., 2010; Hübner & Töbel, 2012; White et al., 2011) so that different drift rates can be obtained from the same data leaving it unclear whether the association of slower drift rates and ADHD holds for different diffusion models. Furthermore, drift rates are generally not interpreted in terms of arousal. Instead the drift rates are though to map the speed of information uptake or the quality of information extracted

from the stimulus (Ratcliff, Love, Thompson, & Opfer, 2012; Voss et al., 2013). Even if changes in a person's level of arousal influence drift rates in cognitive tasks, drift rates are affected by other processes as well and therefore cannot be regarded as a pure indicator of arousal. Finally, diffusion models cannot deal with omission errors as omission errors are excluded from the analysis (Voss et al., 2013). However, given the factor-analytical results from this study, omission errors and RTV should be considered indicators of the same construct (sustained attention). Therefore, theoretical accounts explaining RTV need to explain omission errors as well. The DMN hypothesis addresses both RTV and omission errors, and omission errors and especially two or more omission errors in a row literally correspond to attentional lapses. Hence, although effects of arousal cannot be excluded, the results of the current study might be better explained by the DMN hypothesis than by arousal.

While the present study revealed deficits in sustained attention in children with ADHD, no deficits were found in measures of behavioral inhibition. Neither did children with ADHD commit more inhibition errors in the Go/No-Go task than unaffected children, nor did they exhibit a larger Flanker interference effect for errors. Indeed, although deficits in behavioral inhibition lie at the core of theories of ADHD, as for instance the executive dysfunction theory (Barkley, 1997) or multiple pathway models (Sonuga-Barke, 2002; Sonuga-Barke et al., 2010), several studies failed to report deficits in children with ADHD with regard to No-Go inhibition (Börger & van der Meere, 2000; Collings, 2003; Fallgatter et al., 2004; Smith et al., 2006) and Flanker interference errors (Booth et al., 2007; Crone, Jennings et al., 2003; Drechsler, Brandeis, Földényi, Imhof, & Steinhausen, 2005; Johnstone & Galetta, 2013). Furthermore, it is assumed that only about 20%–50% of children with ADHD exhibit deficits in behavioral inhibition (Coghill et al., 2014; de Zeeuw et al., 2012; Sonuga-Barke et al., 2010; Tsal et al., 2005). Thus, taking into account the sample size of this study, it is not unusual that we did not find any differences between children with and without ADHD on behavioral inhibition. Furthermore, the behavioral inhibition indicators from the Go/No-Go and Flanker task did not correlate. This absence of a correlation indicates that withholding of responses and interference control might at least be separable components of behavioral inhibition, if not even unrelated constructs (see Section 5.2.2. for a detailed discussion on measurement and validity concerns). For instance, it was previously suggested that a Go/No-Go task involves response related inhibition only, while the Flanker task involves both, response related inhibition, and stimulus (distractor) related inhibition, and consequently correlations between these measures should be small (Stahl et al., 2014).

Within the Flanker task measures of behavioral inhibition can be separated from measures of conflict-monitoring (Botvinick et al., 2001, 2004). Unaffected children showed a reduction in the Flanker interference effect for response times after incongruent trials (sequential congruency effect for response times), whereas children with ADHD did not display such a modulation of the Flanker interference effect or rather in the opposite direction. While there were no differences between children with and without ADHD in the Flanker congruency effect for response times, the differences in the sequential congruency effect might reflect a deficit in conflict-monitoring. Interestingly, neuronal activity in parietal networks implicated in sustained attention has been related to activity in the ACC (Fassbender et al., 2004). Similarly, in our study correlations of medium size have been observed between various measures of conflict-monitoring and sustained attention (see Table C2 in Appendix C), indicating that the absence of conflict-monitoring in children with ADHD might be a byproduct of deficits in sustained attention. This interpretation is further supported by results from mind wandering. By definition, mind wandering state implies a deficit in sustained attention and higher mind wandering has been associated with deficits in conflict-monitoring (Kam et al., 2012).

Taken together and in line with one previous study (Tsal et al., 2005) it can therefore be concluded that deficits in sustained attention might be more pronounced and characteristic for ADHD than deficits in behavioral inhibition. In addition, reconsidering the deficit in conflict-monitoring and its relation to sustained attention and mind wandering, sustained attention might even be a prerequisite for conflict-monitoring. Hence, incorporating a sustained attention pathway in multiple pathway models of ADHD should be beneficial for understanding the disorder in more detail.

5.1.2. Research Aim 2: Risky Decision-Making and ADHD

The second research aim of this thesis was to investigate the associations between measures of risky decision-making with a categorical diagnosis of ADHD. Therefore, three behavioral risky decision-making tasks (CCT, GDT, NST) were applied, of which only the GDT had previously been used in children with ADHD (Drechsler et al., 2008). While in the study by Drechsler and colleagues (2008) differences in risky decision-making between children with and without ADHD were present only when the task was played for a second time, in our study differences were present right from the start. Yet, Drechsler and colleagues (2008) used a computerized variant of the GDT, while our GDT was purely behavioral. Thus,

future studies should investigate whether the difference between the two studies was due to the presentation mode of the task.

However, children with ADHD played more risky in the GDT than unaffected control children, but not in the CCT or NST. These diverging results concerning the different tasks can have various reasons. For instance, deviations can be possibly be related to whether the tasks (1) are the dynamic or static, and (2) involve hot or cold properties. A risky decision-making task is called static if the probabilities of winning and loosing or the expectancy value are not changing over time (Figner et al., 2009; Lejuez et al., 2002). This condition is fulfilled by the GDT. Regardless of what has happened in the previous round, neither the probabilities, nor the outcomes associated with the single decision options change. In contrast, in the CCT both, the probability of turning over a loss card and the associated expectancy value, change with every card turned over (e.g., assuming tat only one loss card is in the deck, the probability of turning over a loss card is 1/12 for the first move and 1/11 for the second move of a trial; see Appendix D.1.). In a similar way, the expectancy value in the NST changes for every throw of the dice (e.g., throwing the dice for the first time in a trial, the expectancy value is $EV = 2.5$, while the expectancy value is $EV = 0$ if a child has already accumulated 15 points within the trial; see Appendix D.2.).

A risky decision-making task is called cool if it triggers deliberative decision making-processes that rely on cognitive control or calculating of probabilities or expectancy values. In contrast, a risky decision-making task is called hot if it triggers affective decision-making processes (Figner et al., 2009). In each trial of the GDT only one single decision could be made based on probabilities and the associated expectancy value. In addition, the affective system was triggered by immediate feedback after each trial. In contrast to the GDT, affective risky decision-making was explicitly triggered in both, the CCT and the NST. In both tasks, children were allowed to make stepwise incremental decisions about turning over an additional card or throwing the dice another time. Turning over a card or throwing a dice always led to an immediate feedback and the current point total was verbally presented to the child (Figner et al., 2009).

Thus, the GDT represented a static and more cool risky decision-making task while the CCT and the NST can be considered dynamic and hot risky decision-making tasks. As children with and without ADHD only differed on the GDT measure, deficits in risky decision-making might be primarily driven by deficits in executive functions, as for instance, sustained attention and conflict-monitoring and behavioral inhibition. However, it cannot be ruled out that a dynamic task is just easier to perform, as it requires less understanding of

probabilities and expectancy values. Indeed, classical views on risk taking in children (Hoemann & Ross, 1971; Piaget & Inhelder, 1975) argue that children do not understand probabilities before the onset of formal operations, which happens to be the case at the age of twelve. In contrast, more recent accounts argue that an intuitive understanding of probabilities and expectancy values might already be present in preschool children, but not entirely mastered before the age of 13 (Falk & Wilkening, 1998; Schlottmann, 2001; Schlottmann & Wilkening, 2011). Given the age of the children in the current study, it can therefore be assumed that understanding of probabilities and expectancy values has not been entirely reached yet. Hence, as in the GDT probabilities and expectancy values do not change over time, less understanding of probabilities and expectancy values is needed to perform the task as compared to the CCT or NST, in which probabilities or expectancy values are constantly changing over time. Consequently, the GDT should be easier to perform than the CCT or NST. In addition, the GDT consists of more trials than the CCT or the NST and performance in each trial is less dependent on chance, as a single trial consists of only one decision instead of a sequence of decisions.

Taken together, children with and without ADHD did only differ in risky decision-making in a static and more cool risky decision-making task, but not in hot and dynamic risky decision-making tasks. However, as static and cool risky decision-making tasks draw on executive functions, risky decision-making, as measured by the number of risky decisions in the GDT, might be candidate of a more complex behavior to mediate the effects of sustained attention, behavioral inhibition, and conflict-monitoring in ADHD.

5.1.3. Research Aim 3: ADHD – Categorical Diagnosis or Continuum of Symptoms

The third research aim of this thesis was to investigate whether associations between measures of sustained attention, behavioral inhibition, conflict-monitoring, risky decision-making and ADHD were stronger if ADHD was measured continuously and not as a categorical diagnosis. The rationale behind this hypothesis is based on two arguments. First, effect sizes are generally larger for continuous measures as compared to measures that have been dichotomized (MacCallum, Zhang, Preacher, & Rucker, 2002). Second, associations between two constructs are maximal when both constructs correspond in nomothetic span (Wittmann & Klumb, 2006). In other words, if measures of cognitive constructs, as for instance measures of sustained attention, are specifically related to symptoms of inattention, but not to symptoms of impulsivity or hyperactivity, then larger associations between

measures of sustained attention and symptoms of inattention should be expected as compared to the categorical diagnosis of ADHD. In particular, stronger associations were assumed for symptoms of inattention and measures of sustained attention on the one hand and for associations between symptoms of impulsivity and measures of behavioral inhibition and risky decision-making on the other hand.

Results of our study revealed stronger associations between sustained attention, conflict-monitoring, and risky decision-making in the GDT with a composite score of continuous ADHD symptoms than with the categorical diagnosis of ADHD..As with the categorical diagnosis, no associations were found for the composite score of continuous ADHD symptoms and measures of behavioral inhibition or dynamic risky decision-making. In contrast to our predictions, the associations between sustained attention, conflict-monitoring and the risky decision-making in the GDT were stronger for the composite score of ADHD symptoms than for the single ADHD symptom scales of the FBB-ADHS (Döpfner et al., 2006). Furthermore, although the association between sustained attention and the inattention subscale was larger than the association with the impulsivity subscale, the strongest associations for both sustained attention, conflict-monitoring, and risky decision-making were found for the hyperactivity subscale.

While this is evidence against the stated hypothesis that effects sizes should be larger for single symptoms of ADHD than for composite scores, there are two possible explanations. First, the expected effects could have been masked due to the sampling procedure and final sample composition. For instance, the sampling procedure was not designed to test for continuous effects and especially not for continuous effects on single ADHD symptoms. Instead, the sampling procedure aimed at differentiating children with a categorical ADHD diagnosis from unaffected control children. Accordingly, the exclusion of children who could not reliably be assigned to either group further consolidated the recruitment of two distinct groups instead of a continuum. In addition, two of our ADHD screening instruments did not address all ADHD symptoms equally, for instance the CBCL has a stronger focus on attentional problems, while the SDQ hyperactivity scale mainly addresses hyperactivity and impulsivity. However, as exclusion criteria were based on all screening instruments, children with ADHD were implicitly favored if they suffered from a combined presentation of ADHD as compared to a predominately inattentive or predominately impulsive-hyperactive presentation. Therefore, variance for single symptom scales might have been reduced as compared to the composite score and therefore effect sizes were attenuated. On the other hand, a second explanation could be that sustained attention, conflict-monitoring, and risky

decision-making are indeed related to all symptoms of ADHD, although the relation might be strongest to symptoms of hyperactivity.

This strong association of the different measures of sustained attention, conflict-monitoring, and risky decision-making with hyperactive symptoms was rather surprising. Generally, one would have assumed stronger associations for sustained attention and inattentive symptoms or for risky decision-making and impulsive symptoms (Bayard et al., 2011; Billieux et al., 2010). One explanation might be that these strong findings for hyperactive symptoms merely reflect an artifact due to the conduction of symptom ratings. Ratings were entirely obtained from parents. However, parent ratings of ADHD symptoms as compared to teacher ratings have been shown to overemphasize hyperactivity in several questionnaires including the SDQ (Papageorgiou, Kalyva, Dafoulis, & Vostanis, 2008). Thus, further research might want to also include teacher ratings of ADHD symptoms. A second, more theoretical explanation bases on deficits in sustained attention as a marker of more off-task behavior in general. Off-task behavior can be defined as disengaging from a current task and engaging in task-unrelated behavior (Baker, 2007). This definition resembles the definition of mind wandering in cognitive tasks, as mind wandering is defined as off-task thoughts (Smallwood et al., 2004; Smallwood & Schooler, 2006). Thus, by definition, engaging in mind wandering implies a reduction in sustained attention. In addition, deficits in sustained attention and stronger mind wandering have been associated with deficits in conflict-monitoring (Fassbender et al., 2004; Kam et al, 2012) and sleep deprivation. Sleep deprivation, however, not only led to deficits in sustained attention (Hoedlmoser et al., 2010), but also to enhanced risky decision-making (Killgore, Balkin, & Wesensten, 2006; McKenna, Dickinson, Orff, & Drummond, 2007), indicating that enhanced risk decision-making in sleep deprived participants might be mediated by a reduction in sustained attention. Combining these interpretations, a general task disengagement could have caused the observed deficits in sustained attention and conflict-monitoring in our study, which in turn in turn resulted in risky decision-making. This interpretation is supported by results from observational studies in classrooms. As disengaging from one task is usually accompanied by engaging in another task, children with ADHD engaged in various off-task activities, particularly off-task motor behavior like “difficulties in sitting still”, “fidgeting”, or “being off-chair” (Abikoff et al., 2002; Vile Junod, DuPaul, Jitendra, Volpe, & Cleary, 2006). Thus, the strong associations of measures of sustained attention, conflict-monitoring, and risky decision-making with hyperactive symptoms might reflect off-task behavior, with deficits in sustained attention and

conflict-monitoring indicating a general tendency to disengage from cognitive tasks, whereas hyperactivity reflects the engagement in task-unrelated behavior.

Taken together, associations between measures of sustained attention, conflict-monitoring, risky decision-making and ADHD were stronger for continuous symptoms than the categorical diagnosis. Furthermore, associations were in most cases stronger for composite continuous ADHD symptom scores than for single symptom scales, yet associations with the composite scores were mainly due to hyperactive symptoms. Off-task behavior, indicated by the disengagement from cognitive tasks and engagement in hyperactive symptoms, might be a promising explanation. However, this explanation needs to be tested in future studies by observing hyperactive symptoms during cognitive tasks (e.g., CPT, Flanker, Go/No-Go) to directly assess the link between task disengagement and engagement in hyperactive behavior.

5.1.4. Research Aim 4: Risky Decision-Making as a Mediator – Indirect Effects of Cognitive Endophenotypes on ADHD

The fourth research aim of this thesis was to investigate whether associations between measures of sustained attention, behavioral inhibition, conflict-monitoring, and ADHD were mediated by risky decision-making. The rationale behind this hypothesis relies on the assumption that psychological endophenotypes like sustained attention and behavioral inhibition are not directly related to the syndrome of ADHD. Rather, they are related to more complex behaviors, as for instance risky decision-making that in turn are related to the symptom dimensions of ADHD and thereby to ADHD as a syndrome (see Figure 2).

While mediation analyses with cross-sectional, non-experimental data cannot test the causal assumptions behind this hypothesis (Hayes, 2013), it can be tested whether the observed data is consistent with what would be expected if a causal path leading from psychological endophenotypes to risky decision-making to ADHD symptoms would be true (Kraemer, Stice, Kazdin, Offord, Kupfer, & 2001). Importantly, these indirect effects can be present even in the absence of a total effect of the cognitive constructs on ADHD symptoms (Hayes, 2009; Rucker et al., 2011). However, in order to consider the indirect effect a mediation, the total effect needs to be present initially, because only then can risky decision-making account for the relation between psychological endophenotypes and ADHD symptoms (Baron & Kenny, 1986; Mathieu & Taylor, 2006).

The analyses of indirect effects and mediations effects through risky decision making on continuous ADHD symptoms in the present study revealed a marginally significant partial mediation effect for sustained attention and an indirect effect for behavioral inhibition. through risky decision-making. No significant indirect effect on ADHD symptoms through risky decision-making was observed for conflict-monitoring. However, an indirect effect was present when children were included in the sample with exceptionally bad performances in the Flanker task, as for instance performing below chance or committing more than 50% omission errors (see Appendix D.4.1.). These children were initially excluded as it was assumed that they did not perform the task and thus measures could not be trusted.

Risky decision-making has traditionally been related to measures of behavioral inhibition and reward (Odlaug et al. 2011, Paulsen et al., 2011; Toplak et al., 2010; Verbruggen et al., 2012), as theories assume that a deliberative, cognitive control system inhibits affective impulses to overcome the urge for short-term gains (Casey et al., 2008; Knoch & Fehr, 2007; Steinberg, 2008). In the present study, riskier options in the GDT were associated with higher outcomes but lower expectancy values (Brand, Fujiwara et al., 2005). Children needed to inhibit their urge for the larger gains in the favor of long-term benefits. Therefore, the observed relation of higher inhibition error rates in the Go/No-Go task and a higher number of risky decisions in the GDT integrated well into these theories.

The observed indirect effect is in principle in line with the outlined research framework stating that deficits in behavioral inhibition might not directly relate to ADHD, but to more complex psychological functions as risky decision-making, which in turn influence ADHD symptoms. However, due to the absence of a total effect no mediation effect could be claimed. While the absence of a total effect of behavioral inhibition on ADHD symptoms was unexpected, it was not unlikely given that only 20%-50% of children with ADHD exhibit problems in behavioral inhibition (Coghill et al., 2013; de Zeeuw et al., 2012; Sonuga-Barke et al., 2010). Importantly, the observed indirect effect might be valid regardless of the significance of the total effect. Simulation studies recently revealed that for a sample size of $n = 100$ (similar to our study) and a true total effect of small or medium size (as in behavioral inhibition and ADHD), the probability the probability of finding a significant indirect effect in absence of a significant total effect is about 40% (Rucker et al., 2011).

Thus, while the indirect effect of behavioral inhibition on ADHD symptoms through risky-decision making was expected, the marginally significant partial mediation effect for sustained attention on ADHD symptoms was rather surprising, considering theories of risky decision-making. However, recently relations of risky decision-making and attention have

been proposed. Building on a clinical study that did not test but suggested that deficits in sustained attention could explain risky decision-making in mania (Clark, Iversen, & Goodwin, 2001) a recent study reported that risky decision-making in the IGT was twice as high related to measures of sustained attention than to measures of executive functions and behavioral inhibition in particular (Gansler et al., 2011). Yet, the effects of sustained attention might not relate to risky decision-making in terms of impulsivity, as it neither reflects reward processing nor the inhibition of affective impulses. Rather, it is possible that deficits in sustained attention reflect difficulties in maintaining the task set or an impaired learning of outcome frequencies. However, it is not very likely that children had difficulties maintaining the task set in our study, as all task rules were present to the children throughout the whole study. In contrast, deficits in sustained attention and attentional lapses in particular might have resulted in more risky decision-making in the GDT, as less attention was paid to the task and especially to the outcomes of the single trials. As probabilities are derived from frequencies (Cosmides & Tooby, 1996; Gigerenzer & Hoffrage, 1995; but see Johnson-Laird, Legrenzi, Girotto, Legrenzi, & Caverni, 1999), wrong estimates of the probabilities of the single outcomes in the GDT would have been inferred. Hence, the marginally significant mediation effect of sustained attention on ADHD symptoms through risky decision-making might be associated with impulsive behavior due to problems in learning expectancy values.

While indirect effects on ADHD symptoms through risky decision-making have been revealed for behavioral inhibition and conflict-monitoring, no significant indirect effect of conflict-monitoring was observed. However, an indirect effect was present when children were included in the sample with exceptionally bad performances in the Flanker task. Initially, these children were excluded, as it was assumed that they did not perform the task. However, not performing the task actually by definition is off-task behavior. As already argued for sustained attention, not paying attention to the task and off-task behavior might lead to problems in learning outcome probabilities in the GDT. In addition, conflict-monitoring might be impaired as well. For instance, losses generally lead to a reorientation of attention and less risky decision-making in subsequent trials (Yechiam & Hochman, 2013a, 2013b). However, if less attention was paid to the task, performance would not be efficiently monitored and therefore, the beneficial effect of losses on attention would be diminished.

I summary, the indirect effect of behavioral inhibition on ADHD symptoms through risky decision-making observed in the current study is in line with the outlined framework and might reflect a higher impulsivity in children with ADHD. In contrast, the indirect effects of sustained attention and conflict-monitoring were probably related to off-task thoughts and

off-task behavior. Hence, to further test this assumption, future studies should include objective measures of off-task behavior.

5.2. Limitations

This section evaluates limitations of the current investigation. First, limitations are discussed with regard to the sampling strategy and sample composition (Section 5.2.1.). Second, general issues regarding the measurement and construct validity of ADHD symptoms, sustained attention, behavioral inhibition, conflict-monitoring, and risky decision-making are evaluated (Section 5.2.2.).

5.2.1. Sampling Strategy and Sample Composition

The sampling strategy aimed at recruiting children with a categorical ADHD diagnosis and unaffected control children. Children with a categorical ADHD diagnosis were mainly recruited in a clinic for child and adolescent psychiatry. As contact data of these children could be received from an existing database, most children with ADHD could be directly contacted through the experimenters. In contrast, unaffected children were mainly recruited in schools, where they received a flyer and a study description from their teachers (see Appendix A.3.). Consequently, the initial contact with the study team had to be made by the children's parents. Although this procedure is convenient, it has some drawbacks.

First, sampling children with a categorical ADHD diagnosis and excluding all children that could not unambigously assigned to either the ADHD or control group is straightforward in order to compare affected and unaffected children on sustained attention, behavioral inhibition, conflict-monitoring and risky decision-making. However, if ADHD is treated continuusly, this procedure leaves out data from the middle of the distribution and consequently, it is unclear whether the observed associations between the different psychological functions and ADHD symptoms would hold if the comlete distribution of ADHD symptoms had been considered (Preacher, Rucker, MacCallum, & Nicewander, 2005). Hence, the only way to investigate true associations of these constructs with ADHD symptoms is by relying on representative population-based samples as in epidemiological studies (Sciberras, 2014; Sciberras et al., 2013).

However, the recruited sample of our study might not be representative for a comparison of children with and without ADHD. For instance, most of the children with

ADHD were recruited in a clinic for child and adolescent psychiatry. Yet, clinical psychiatric samples over-represent more severe cases of ADHD, males, and children with comorbid psychiatric disorders as compared to general population samples of children with ADHD (Sciberras et al., 2009, 2013). Indeed, the male to female ratio in our ADHD group amounted to 4.7:1 and thus was considerably higher than in epidemiological studies (e.g., 2.28:1; Ramtekkar, Reiersen, Todorov, & Todd, 2010) and in the control group (2.24:1, male to female ratio). Furthermore, while both, the ADHD and the control group, came from high SES households (Lampert et al., 2013), the control group had a very high mean IQ, possibly indicating a participation bias (Cuddeback, Wilson, Orme, & Combs-Orme, 2004). Finally, 17% of the children in the present study had to be excluded from the final sample, as they could not be assigned to either the ADHD or the control group. These 17% might include at least some children with a weak ADHD so that results primarily hold for a difference between children with a severe ADHD and unaffected children with a high IQ.

A further limitation concerns the sample size especially with regard to heterogeneity in ADHD. For instance, according to multiple pathway models only 20%-50% of children with ADHD exhibit deficits in behavioral inhibition (Coghill et al., 2013; de Zeeuw et al., 2012; Sonuga-Barke et al., 2010). Thus, although several studies relying on smaller samples reported differences in Go/No-Go performance between children with ADHD and unaffected children (e.g., de Zeeuw et al., 2012; Gawrilow & Gollwitzer, 2008; Rauch et al., 2012), larger samples would be needed to detect and compare behavioral inhibition deficits within children with ADHD.[4]

Finally, the main results of this study have been related to sustained attention and with sustained attention predicting a categorical ADHD diagnosis, symptoms of ADHD, and risky decision-making. However, deficits in sustained attention have been reported in various disorders besides ADHD, including autism (Chien et al., 2014), bipolar disorder (Camelo, Velasques, Ribeiro, Netto, & Cheniaux, 2013), and schizophrenia (Suwa, Matsushima, Ohta, & Mori, 2004) questioning the specificity of a sustained attention deficit to symptoms of ADHD. Thus, future research on ADHD needs to include children with other disorders than ADHD as well or should measure symptoms for various disorders in population-based samples.

[4] For instance, in order to get about 20 children with ADHD who have a deficit in behavioral inhibition, already 67 children with ADHD would be needed, when 30% of the children with ADHD are assumed to have a deficit in behavioral inhibition.

5.2.2. Measurement, Construct Validity, and Construct Interpetation

This section reflects on the measurement and construct validity of ADHD symptoms, sustained attention, behavioral inhibition, conflict-monitoring, and risky decision-making. Several concerns for the measurement, construct validity, and interpretation of these constructs will be noted, however, these concerns are not specific to the current study, but rather constitute general issues in the study of ADHD.

ADHD symptoms. Continuous ADHD symptoms were assessed by parental ratings on the SDQ (Woerner et al., 2004), the CBCL (Arbeitsgruppe Deutsche Child Behavior Checklist, 1998), and the FBB-ADHS (Döpfner et al., 2006). However, to obtain an ADHD diagnosis generally parent and teacher ratings are combined (Pelham, Fabiano, & Massetti, 2005; Shemmassian & Lee, 2012). A combination of parent and teacher ratings is assumed to increase the validity of the measure (Johnston & Murray, 2003), although parent and teacher ratings of ADHD are only moderately correlated (de Nijs et al., 2004; Gomez, 2007). While in general parents have been found to underreport symptom severity (Wolraich et al., 2004) they might also overemphasize symptoms of hyperactivity (Papageorgio et al., 2008). However, if parents underreport symptom severity relying on parent ratings on ADHD will reduce differences between children with and without ADHD symptoms. In addition, if parents simultaneously overemphasize symptoms of hyperactivity, associations of ADHD with any measures might be artificially increased for symptoms of hyperactivity as compared to symptoms of inattention or impulsivity.

Sustained Attention. Sustained attention or vigilance can be defined as a person's readiness to detect signals over a prolonged period of time (Sarter et al., 2001). In this study, several indicators of sustained attention were derived from cognitive tasks: RTV, the rate of omission errors, and the number of omission errors a person committed in a row. Due to high intercorrelations between the RTV measures and the omission error rates, convergent validity could be assumed and the latter were combined to a more reliable measure of sustained attention.

However, although this measure of sustained attention might be reliable and valid, the undelying processes and mechanisms are less clear, making an interpretation of sustained attention deficits difficult. For instance, the DMN hypothesis explains both, a larger RTV and a higher number of omission errors, by very low frequency interference of DMN activity. However, this interpretation of the cognitive-behavioral measures of sustained attention faces several problems. The main problem is that RTV and omission errors are events but not mechanisms (Karalunas et al., 2014). Thus, the cognitive-behavioral data itself cannot explain

by which mechanism of attention it has been caused or whether it has been related to an attentional process at all.

Indeed, various mechanisms are thought to contribute to a higher RTV, as for instance, stimulus encoding, processing speed, speed-accuracy trade-offs, post-error slowing, arousal, effort, motor preparation, and response execution (Karalunas et al., 2014) and not all of these mechanisms are regarded as sustained attention. For instance, post-error slowing is generally seen as a measure for conflict-monitoring (Chang, Chen, Li, & Li, 2014; Maier, Yeung, & Steinhauser, 2011; Yeung et al., 2004) or orienting attention (King, Korb, von Cramon, & Ullsperger, 2010; Notebaert et al., 2009), while arousal and effort aim at motivational processes (Locke & Latham, 2002; Sanders, 1983) and motor preparation and response execution reflect general motor behavior.

Given the involvement of the cerebellum in motor control (Manto et al., 2012), abnormalities in the cerebellum in children with ADHD (Durston et al., 2007; Goetz, Vesela, & Ptacek, 2014; Montes et al., 2011; Noreika et al., 2013) and that children with ADHD are impaired in motor tasks (Fliers et al., 2009; Klimkeit, Mattingley, Sheppard, Lee, & Bradshaw, 2005) it cannot be ruled out that the larger RTVs in children with ADHD were partly due to impairments in motor behavior.

In addition, motivational accounts of ADHD, like the state regulation theory (Sergeant, 2000, 2005), emphasize the role of arousal on cognitive performance. Accordingly, larger RTVs in children with ADHD are mainly reported for long event-rates, but not for fast event-rates (Sergeant, 2005; Tamm et al., 2012). However, while RTVs in this study were derived from three different tasks (CPT, Go/No-Go, Flanker), these tasks were similar in their event rates with inter-trial intervals of about 1550-2000 ms. Thus, effects of event-rates and arousal could not be examined.

Nevertheless, a large number of omission errors and especially omission errors in a row might reflect task motivation and not attentional processes. Critically, about 10%-15% of the children had to be excluded from the analyses of the cognitive tasks because they committed more than 50% omission or commission errors, thereby revealing a lack of task motivation. However, 50% is a rather lenient criterion (e.g., Solanto et al., 2001, excluded children with more than 20% omission errors) and thus the final sample might include children whose RTV, omission errors, and especially omission errors in a row might reflect a general lack in task motivation rather than deficits in any attentional processes. This interpretation could be further supported, if RTVs and sustained attention were regarded as a more general form of off-task related thoughts or mind-wandering (Smallwood, 2004;

Smallwood & Schooler, 2006). During mind-wandering people allocate motivational ressources to personal goals (Oettingen & Schwörer, 2013), which results in a general reduction in task-motivation for the performed cognitive tasks.

Taken together, a reliable measure of sustained attention could be derived from principle components analysis based on RTVs and omission errors of three cognitive tasks (CPT, Go/No-Go, Flanker) with similar event rates. However, these indicators themselves represent events, but not mechanisms and, while they are consistent with an account of attentional lapses due to DMN activity, possible alternative mechanisms exist with regard to motor behavior, arousal, and a more general form of task-motivation. Thus, future studies have to test the mechanisms undeling the sustained attention index of the present study.

Behavioral Inhibition. In the present study, there was no corrleation between the rate of inhibition errors in the Go/No-Go task and the Flanker interference effect. Although this result questions the validity of a behavioral inhibition construct, it is in line with seveal other studies, concluding that these different inhibitory processes (in particular withholding of responses and interference control) are only weakly correlated (Khng & Lee, 2014; Wager et al., 2005) and might represent different latent variables (Stahl et al., 2014; Friedman & Miyake, 2004[5]). In addition, withholding of responses and interference control have been associated with different EEG correlates (Brydges, Anderson, Reid, & Fox, 2013; Brydges et al., 2012) and could even be dissociated on a neuronal level relying on fMRI (Chambers et al., 2007; Luk, Anderson, Craik, Grady, & Bialystok, 2010) or a patient with left inferior frontal damage (Hamilton & Martin, 2005).

However, there is an ongoing discussion in research on construct validity whether indicators of the same constructs need to be highly correlated. Transferring this discussion to behavioral inhibition, proponents of a high correlation between indicators of behavioral inhibition would argue that weakly correlated indicators might rather represent different constructs (McGrath, 2005; Strauss & Smith, 2009), whereas their opponents would argue that too highly correlated indicators of behavioral inhibition might actually narrow the width of the construct and thereby reduce its construct validity (Loevinger, 1954). Similar positions

[5] Friedman & Miyake (2004) reported a considerably high correlation between latent factors of response inhibition (withholding of responses and inhibition of already initialized responses) and interference control. However, a reanalysis (Stahl et al., 2014) revealed that this high correlation was mainly been due to the use of the Stroop task as a marker for response inhibition and the Flanker task as marker for interference control, two task that are usually considered to share similar mechanisms (Botvinick et al., 2001, 2004).

can be derived following the distinction between reflexive and formative models (Jarvis, Mackenzie, & Podsakoff, 2003). A reflexive model of behavioral inhibition would suppose that different measures of behavioral inhibition were manifestations of a single general construct and thus, a high correlation between withholding of responses and interference control would be assumed. In contrast, a formative model of behavioral inhibition would suppose that different measures of behavioral inhibition define different parts of the general construct and thus, there would be no need for high correlations between withholding of responses and interference control. Hence, whether withholding of responses and interference control both resemble behavioral inhibition is a theoretical and not an empirical question.

However, there is an additional problem with the construct validity of behavioral inhibition, especially with interference control as a inhibitory process. In general, if behaviorally observed interference effects are attributed to behavioral inhibition and subsequently behavioral inhibition is defined on these observed interference effects, there is a danger of circularity (Klein & Taylor, 1994). Instead, it should be distinguished between interference as an empirical result and behavioral inhibition as a possible mechanism (MacLeod, 2007; MacLeod, Dodd, Sheard, Wilson, & Bibi, 2003). Indeed, some explanations for Stroop interference did not involve any mechanisms of inhibition (MacLeod et al., 2003; Roelofs, 2003), questioning whether interference control can actually be regarded as an inhibitory process.

Thus, various different mechanisms and processes are involved in tasks measuring behavioral inhibition, which becomes even more plausible considering that several tasks for the measurement of behavioral inhibition have originally been developed to study different cognitive functions (Sabb et al., 2008). For instance, the Flanker task was developed to study selective attention and the effects of noise on visual target location (Eriksen & Eriksen, 1974), while the Go/No-Go task originally measured processing speed (Donders, 1868/1969). Hence, there exists no pure measure of any cognitive function exists (Miyake et al., 2000). Consequently, theory-driven measures should be derived across multiple conditions or tasks (Silverstein, 2008) to disentangle the specific inhibitory processes from other cognitive (e.g., perception, attention, working memory, decision-making) and non-cognitive processes (e.g., arousal, motivation).

Taken together, it remains a theoretical question, whether both withholding of responses and interference control resemble the construct of behavioral. In addition, measures of behavioral inhibition, as derived from response times and errors, are often not well defined, but involve several non-inhibitory processes. However, disentangling inhibitory processes

from other cognitive and non-cognitive processes involved the measures might be especially important for research on ADHD, as precise measures of inhibition might help to clarify the heterogeneity in behavioral inhibition in children with ADHD.

Conflict-Monitoring. The conflict-monitoring theory (Botvinick et al., 2001; Botvinick et al., 2004) was proposed to explain performance in behavioral inhibition tasks, in particular in tasks of interference control. Trial-by-trial modulations of response times and error rates (e.g., PCS, sequential congruency effects) have generally been interpreted in the favor of the theory. For instance, a reduced Flanker congruency effect after incongruent trials as compared to congruent trials (sequential congruency effect) is explained by incongruent trials causing a response conflict. According to the theory, this conflict is measured in a conflict-monitoring unit and forwarded to an attentional control unit. In the subsequent trial, the attentional focus is narrowed to the center of the screen leading to a better processing of the central target stimulus and less processing of the Flanker stimuli (Botvinick et al., 2001; Botvinick et al., 2004).

However, several competing explanations have been proposed to explain sequential congruency effects (Duthoo, Abrahamse, Braem, Boehler, & Notebaert, 2014; Egner, 2007). For instance, according to the feature integration theory (Hommel, Proctor, & Vu, 2004; Mayr, Awh, & Laurey, 2003), sequential congruency effects can be explained by priming effects due to stimulus feature repetitions in consecutive trials. Therefore, many studies try to control for feature repetition either by excluding these trials from the analysis or by expanding the stimulus set while maintaining a 1:1 congruent-incongruent ratio (Ullsperger, Bylsma, & Botvinick, 2005). Another explanation focuses on participants' expectancies regarding the congruency of the upcoming trial, as it is assumed that participants expect congruency to repeat and are therefore faster and more accurate, when a congruent trial follows a congruent trial or when an incongruent trial follows an incongruent trial (Duthoo, Wühr, & Notebaert, 2013; Gratton et al., 1992).

However, the Flanker task used in the current study cannot differentiate between these accounts (Duthoo, Abrahamse, Braem, & Notebaert, 2014) and thus alternative explanations to the conflict-monitoring theory cannot be excluded. In addition, even within a conflict-monitoring framework it is impossible to decide, whether empirical findings of PCS and sequential congruency effects are related to conflict-monitoring or conflict adaptation. Thus, the deficits found in children with ADHD, namely less modulation of response times and errors following incongruent trials and smaller sequential congruency effects for errors, cannot be assigned to either of the two components.

The analysis of different indicators of conflict-monitoring (e.g., PCS, PCE, sequential congruency effects) was further impeded by the fact that these measures are not mutually exclusive (Forster & Cho, 2014; Verguts et al., 2011). For instance, sequential congruency effects for response times and errors are confounded by PCS or PCE. This confounding of different indicators of conflict-monitoring might at least partly be responsible for the results pattern presented in Sections 4.1. and 4.3. While children with ADHD were overall impaired in conflict-monitoring no specific indicator could be identified. Whereas a smaller sequential congruency effect for response times was found for children with a categorical diagnosis, continuous measures of ADHD were associated with a smaller sequential congruency effect for errors.

Hence, in order to clarify the deficits regarding the modulation of interference control in children with ADHD, future studies need to rely on tasks that are not only able to distinguish between different theories explaining these modulations, but also to differentiate between different adaptation strategies that might operate simultaneously (Duthoo et al., 2014a; Forster & Cho, 2014; Wendt et al., 2012).

Risky Decision-Making. Risky decision-making refers to decisions between two or more alternatives, in which a person opts for an alternative with a lower probability of success. This option is additionally associated with both higher gains and higher losses and therefore, a smaller expectancy value as compared to the other alternatives (Kahneman & Tversky, 1979). As Baumeister and colleagues (2007) insisted on real behavioral measures instead of questionnaires and response time, the current studies consisted of three behavioral, non-computerized risky decision-making tasks: the GDT, CCT, and NST. However, while the non-computerized character of these tasks reflected real life card games and dice games more closely, this real life character also rendered several factors influencing the task outcome uncontrollable. Most importantly, we could not control for chance. For instance, in the NST some children had a rough start, throwing a six immediately for the first two trials, whereas other children managed to score up to 42 points in the first trial. Results from previous trials influence how risky a child will play during the next trial. However, due to the relative small number of trials, especially extreme results might impede children's learning about probabilities and expectancy values, as these might be inferred from frequencies and not from probability calculation (Cosmides & Tooby, 1996; Gigerenzer & Hoffrage, 1995; but see Johnson-Laird et al.,1999). A further difference between computerized and non-computerized tasks is that the latter requires an experimenter to be present during the task. The mere presence of an experimenter might alter risky decision-making in children, as it has already

been demonstrated for adolescents (Gardner & Steinberg, 2005) or students (Zajonc, Wolosin, Wolosin, & Loh, 1970). Adolescents made more risky decisions in the presence of peers (Gardner & Steinberg, 2005), while students were more cautious during mere presence situations (Zajonc et al., 1970).

Thus, future studies need to investigate whether computerized and non-computerized tasks of risky decision-making in children measure the same construct and the same underlying processes. In addition, increasing the number of trials would reduce the influence of chance on risky decision-making by offering a possibility to correctly infer probabilities and expectancy values from outcome frequencies. However, increasing the number of trials will be practically limited as a behavioral trial takes more time than a computerized trial (e.g., due to the shuffling and dealing of cards). For instance, the 16 trials in our behavioral CCT already took approximately 20 minutes, leading to an extrapolation of more than one hour if 54 trials had been applied as in the original computerized version of the CCT (Figner et al., 2009)

However, trial numbers would need to be even larger in order to investigate cognitive and affective mechanisms underlying risky decision-making in these tasks on a trial-by-trial basis (Maia & McClelland, 2004). Possible mechanisms include performance monitoring and reorienting attention after errors (Yechiam & Hochman, 2013a, 2013b), learning (Maia & McClelland, 2004; Visagan, Xiang, & Lamar, 2012), or to what extend information about probabilities, gain amount an loss amount are taken into account (Figner et al., 2009). In addition, a larger number of trials would allow the calculation of more advanced indices for risky decision-making. For instance, in the current study the measures of risky decision-making in the CCT and NST were the average number of cards turned over and the number of dice throws. While this measure has been introduced for the original CCT (Figner et al., 2009), higher scores do not necessarily imply risky decision-making. Instead, it would be possible that lower scores reflect over-cautious decision-making. It is possible to mathematically assess an optimal strategy (see Appendix D), indicating after how many cards turned over in the CCT or at which score in the NST a child should stop turning over cards or rolling the dice. However, due to the small number of trials and effects of chance (e.g., turning over a loss card or throwing a six before reaching the point to stop) comparing the children's decision-making to an optimal strategy cannot be interpreted validly.

5.3. Implications for Research on ADHD

Based on the results of the current study and the limitations outlined in the previous section, several implications can be drawn for future research on ADHD. Accordingly, this section will first describe implications for sampling strategies to analyze ADHD and ADHD symptoms with regard to the general population and other psychiatric disorders (Section 5.3.1.). Second, implications for the measurement of basal cognitive endophenotypes, higher-order cognitive functions, and ADHD symptoms will be elucidated (Section 5.3.2.). Subsequently, implications for multiple pathway models of ADHD and the role of sustained attention and risky decision-making within such models will be emphasized (Section 5.3.3.). Finally and in line with the presented framework, it will be outlined why it is necessary to consider environmental factors within these models (Section 5.3.4.).

5.3.1. Sampling Strategies for Research on ADHD

During the last decade, psychiatric research started to emphasize the necessity of smaller well-defined, quantitative behavioral outcomes instead of categorical diagnoses (Cuthbert, 2005; Hyman, 2010; Morris & Cuthbert, 2012). While the benefits and disadvantages of quantitative outcomes (e.g., symptoms) for research on ADHD are still under debate (Coghill & Sonuga-Barke, 2012) most studies investigating the relation of cognitive functions with ADHD symptoms suffer from non-adequate sampling strategies. Participants are recruited to resemble either children with ADHD (or children at risk for ADHD) or control children but not a general population sample. However, sampling with regard to group affiliation but treating the outcome as continuous mixes these different sampling strategies and complicates combining variables through latent variables approaches, as the distribution of all variables might be bimodal rather than Gaussian. In addition, if children with ADHD are directly recruited from hospitals while unaffected children are recruited from schools, this will lead to artificial and non-representative sub-samples of affected and unaffected children as children who participate in the study might differ from children who do not participate in the study with regard to gender, SES, intelligence, and symptom severity (Cuddeback et al. 2004; Sciberras et al., 2009, 2013). Therefore, future studies should either draw on general population samples or carefully match children with ADHD and unaffected children with regard to factors that influence whether or not a child would take part in the study.

Finally, future studies should include children diagnosed with other disorders (e.g., autism, depression, anxiety) or measure symptoms associated with other disorders to investigate whether reported associations of sustained attention, behavioral inhibition, other basal or higher-order cognitive functions, genetic polymorphisms and environmental factors are specific for ADHD symptoms or a categorical diagnosis of ADHD or whether these constructs resemble more general risk factors for the development of a disorder (Caspi et al., 2014).

5.3.2. Measurement and Validation of Basal Cognitive Endophenotypes, Higher Order Cognitive Functions, and Symptoms of ADHD

One important criterion for endophenotypes is that they need to be reliably assessed by tools with sound psychometric properties (Crosbie et al., 2008; Doyle et al., 2005; Hasler et al., 2006; Kendler & Neale, 2010). Single measures of behavioral inhibition or sustained attention generally have test-retest reliabilities between r = .60 - .80 (Langenecker et al., 2007; Logan et al., 1997; Shalev et al., 2011; Williams et al., 1999; see Section 2.1.1. and 2.2.1.). However, while reliabilities of .80 can be considered acceptable to good, reliabilities > .90 are generally preferred, in particular for performance tests (Webb, Shavelson, & Haertel, 2006). Hence, strategies are needed to enhance the reliability of the behavioral measures of the cognitive endophenotypes of ADHD. One way to increase reliability is to combine several measures through factor scores or latent variables (Miyake et al., 2000). However, inter-correlations between cognitive indices that are supposed to measure the same construct are often low to modest (see Section 5.2.2.). These small inter-correlations can partly be explained by the task impurity problem (Miyake et al., 2000) meaning that several cognitive processes are involved in cognitive performance. Thus, cognitive tasks need to be altered in a way that more process specific indices can be derived across multiple tasks and conditions (Silverstein, 2008).

In addition, physiological measures could be used to further confirm the construct validity of these indices. For instance, the state regulation theory assumes that deficits in task performance are due to a non-optimal arousal level (Sergeant, 2000). Physiological measures for arousal include cortisol level (Peifer, Schulz, Schächinger, Baumann, & Antoni, 2014), heart rate variability (van der Meere, 2005), and fluctuations in skin conductance (Bach, Friston, & Dolan, 2010). However, studies investigating cortisol levels have yielded mixed results and both lower and higher cortisol levels have been observed in ADHD (Corominas et

al., 2012; Isaksson, Nilsson, Nyberg, Hogmark, & Lindblad, 2012). Only heart rate variability has been analyzed with regard to the state regulation theory. Increased heart rate variability was reported for children with ADHD for slow event-rates but not for fast event-rates (Börger & van der Meere, 2000; van der Meere, 2005). Furthermore, while it is claimed that the drift rate derived from diffusion models might be a behavioral correlate of a non-optimal arousal level (Karalunas et al., 2014), no physiological correlates of drift rates have been reported so far. In similar, eye-tracking could be used to validate deficits in sustained attention could (McIntire, McKinley, Goodyear, & McIntire, 2014) and evidence for the DMN hypothesis could be gathered by on-task measuring of DMN activity. In addition, using videography or accelerometers could validate on-task behavior. Finally, higher-order cognitive functions like risky decision-making need to include a larger number of trials than in the current study, in order to investigate and differentiate between mechanisms that are involved in inhibition, sustained attention, learning, or arousal (Maia et al., 2004; Visagan et al., 2012; Yechiam & Hochman, 2013a,b; Zajonc, 1970). Again, the interpretation of behavioral measures could benefit from physiological correlates.

Taken together, future studies should rely on multi-trait multi-method designs (Campbell & Fiske, 1959) to evaluate the associations of different functions and ADHD, thereby ensuring convergent validity throughout behavioral and physiological measures as well as divergent validity throughout the need for highly specific performance indices (Silverstein, 2008).

5.3.3. Sustained Attention, Risky Decision-Making and Multiple Pathway Models of ADHD

Multiple pathway models of ADHD (see Section 1.1.5.) explain heterogeneity on an ADHD symptom level through heterogeneity in basal psychological endophenoytpes. For instance, the dual pathway model (Sonuga-Barke, 2002) proposed that deficits in either behavioral inhibition or delay aversion could independently cause ADHD. Yet, the dual pathway model was not able to explain more than about 50% of the variance in ADHD (Coghill et al., 2014; Sonuga-Barke et al., 2010). Thus, the model incorporated a third pathway: temporal processing (de Zeeuw, Weusten et al., 2012; Durston et al., 2011; Sonuga-Barke et al., 2010). But even the thriple pathways model could not fully explain heterogeneity in ADHD and sustained attention was proposed to constitute an additional pathway (de

Zeeuw et al., 2012). However, so far no formal theoretical model of ADHD includes a pathway of sustained attention or a sustained attention deficit as a core deficit in ADHD.

The results of the current study clearly emphasize the importance of a sustained attention deficit in ADHD. Associations for sustained attention with symptoms of ADHD and the categorical ADHD diagnosis were stronger than associations with behavioral inhibition or conflict-monitoring. The strength of this association was further underpinned by the presence of a total effect of sustained attention on continuous ADHD symptoms even when indirect effects through risky decision-making were considered. In addition, the current study revealed that sustained attention could be reliable measured by a factor score derived from measures of different neuropsychological tasks. Furthermore, measures of sustained attention can be related to specific neuronal areas (Langner & Eickhoff, 2013; Petersen & Posner, 2012; Posner & Petersen, 1990; Sarter et al., 2001; Weissman et al., 2006) and EEG components (Arns, Connors, & Kraemer, 2013; Barry et al., 2003; Hoedlmoser et al., 2010). Finally, heritability has been established through behavioral genetic studies (Kuntsi et al., 2010; Uebel et al., 2010) as well as molecular genetic studies (Barnes et al., 2011; Bellgrove & Mattingley 2008; Kollins et al., 2008) indicating that sustained attention can be considered an endophenotype for ADHD.

With sustained attention as an endophenotype, current multiple pathway models of ADHD would extend to four factor models, including sustained attention, behavioral inhibition, delay aversion, and temporal processing. However, this brings up the question how many pathways actually exist and whether all of the proposed pathways can be considered as endophenotypes. There is preliminary evidence that both delay aversion (see Section 1.1.2.) and temporal processing (see Section 1.1.3.) might be endophenotypes for ADHD.

On delay aversion tasks, behavioral differences between children and adults with and without ADHD have been demonstrated both with regard to an altered delay-to-reinforcement gradient (Demurie, Roeyers, Baeyens, & Sonuga-Barke, 2012; Hurst, Kepley, McCalla, & Livermore, 2011; Paloyelis et al., 2010; Scheres, Tontsch, & Thoeny, 2013; Scheres, Tontsch, Thoeny, & Kaczkurkin, 2010; Wilson, Mitchell, Musser, Schmitt, & Nigg, 2011; but see Scheres et al., 2006) and the motivation to avoid or escape delay situations (Antrop et al., 2006; Bitsakou, Antrop, Wiersema, & Sonuga-Barke, 2006; Bitsakou et al., 2009; Marco et al., 2009; Sonuga-Barke et al., 2010). The smaller delay-to-reinforcement gradient in children and adults with ADHD was related to functional (Costa Dias et al., 2013; Hoogman et al., 2011, 2013; Rubia et al., 2009; Scheres, Milham, Knutson, & Castellanos, 2007; Ströhle et al., 2008) and structural changes in fronto-basal ganglia circuits (Carmona et al., 2009;

Durston et al., 2011), whereas the motivation to avoid or escape delay situations can be related to functional and structural changes in the amygdala (Lemiere et al., 2012; Sasayama et al., 2010; Wilbertz et al., 2013). Finally, heritability of an altered delay-to-reinforcement gradient has been established through behavioral genetic studies (Anokhin et al., 2011; but see Kuntsi et al., 2006) and molecular genetic studies (Kawamura et al., 2013; Paloyelis et al., 2010; Sonuga-Barke et al., 2011) indicating that delay aversion might be an endophenotype for ADHD.

Likewise, an examination of the criteria for endophenotypes with regard to temporal processing demonstrated behavioral differences between children with and without ADHD on a range of temporal processing tasks, including time estimation and time reproduction tasks (Barklay, Murphy, & Bush, 2001; Bauermeister et al., 2005; Pollack, Kroyzer, Yakir, & Friedler, 2009; Prevatt, Proctor, Baker, Garrett, & Yelland, 2011; Rommelse et al., 2007; Valko et al., 2010; see Noreika et al., 2013 for a review), tapping tasks (Rubia, Noorloos, Smith, Gunning, & Sergeant, 2003; Toplak & Tannock, 2005; Zelaznik et al., 2012), and time-based prospective memory tasks (Altgassen, Kretschmer, & Kliegel, 2012; Kerns & Price, 2001; Zinke et al., 2010). Deficits in temporal processing in children with ADHD have been associated with a reduced amplitude of the contingent negative variation ERP component (Banaschewski et al., 2003; Doehnert, Brandeis, Schneider, Drechsler, & Steinhausen, 2013; McLoughlin et al., 2010; Valko et al., 2009) and are thought reflect functional (Durston et al., 2007, 2011; Mulder et al., 2008; Vloet et al., 2010) and structural changes (Castellanos et al., 2002; Mackie et al., 2007; Montes et al., 2011) in fronto-cerebellar circuits. Finally, there is first evidence for heritability of temporal processing from behavioral genetic studies (Rommelse, Altink, Oosterlaan et al., 2008; Rommelse et al., 2007) and molecular genetic studies (de Zeeuw et al., 2013; Sysoeva et al., 2010; Wiener et al., 2011) indicating that temporal processing might be a potential endophenotype for ADHD.

Besides delay aversion and temporal processing, other cognitive constructs might be considered as pathways or even endophenotypes of ADHD, as for instance response speed or working memory. Reduced response speed (de Zeeuw et al., 2012; Epstein et al., 2011; Nikolas & Nigg, 2013; but see Epstein et al., 2003) and decreased working memory capacity (Alderson, Kasper, Hudec, & Patros, 2013; Martinussen et al., 2005; Marzocchi et al., 2008; Rapport et al., 2008) have been reported for children with ADHD, related to neuronal structures (Rypma et al., 2006; Turken et al., 2008; Wen et al., 2011), and have been shown to be heritable (Kumsta et al., 2010; McClearn et al., 1997; Neubauer, Spinath, Riemann, Angleitner, & Borkenau, 2000; Rommelse, Altink, Oosterlaan et al., 2008; Vernon, 1989).

However, no formal model of ADHD (see Section 1.1.) focuses on or even includes deficits in response speed or working memory.

Thus, future studies will have to investigate whether response speed and working memory might constitute additional pathways for ADHD. Furthermore, future studies need to examine whether the proposed pathways are truly independent or whether it is more likely that the single pathways interact. For instance, both working memory and sustained attention have been implicated in temporal processing, considering that a time interval can only be compared to a reference interval if attention is paid to the starting point, the endpoint, and the duration of the interval (Casini & Macar, 1997; Macar, Grondin, & Casini, 1994; Minvielle-Moncla, Audiffren, Macar, & Vallet, 2008; Pollack et al., 2009) and if a representation of the reference interval can be retrieved from working memory (Khan, Sharma, & Dixit, 2006; Matell & Meck, 2000; see Block, Hancock, & Zakay, 2010 for a meta-analysis). Similarly, neuropsychological models have been proposed relating attention, working memory, and behavioral inhibition (e.g., Knudsen, 2007). Attentional processes are supposed to control perceptional inputs that compete for entry into working memory. In turn, working memory compares the processed input with inherent task goals and organizes top-down control to modulate attentional processes in accordance with these task goals, for instance through the inhibition of irrelevant distractors (Knudsen, 2007).

Therefore, to investigate heterogeneity in ADHD it might be more useful to examine performance profiles over various different tasks tapping on different possible pathways, rather than to analyze single pathways independently. Accordingly, a recent study using a latent class analysis on performance on a reward sensitivity task and a time-manipulated Go/No-Go task reported three groups of children with ADHD that could either be characterized as “quick and accurate”, “having poor cognitive control”, or “being slow and variable in timing” (van Hulst, de Zeeuw, & Durston, 2014). However, these results need to be replicated in larger samples and with larger number of cognitive tasks.

In addition, the current study revealed indirect effects of sustained attention and behavioral inhibition on ADHD symptoms through risky decision-making. These indirect effects are in line with the assumption that the different pathways of sustained attention, or behavioral inhibition are not directly related to the symptoms or syndrome of ADHD. Rather, they are related to more complex behaviors like risky decision-making that in turn are related to the symptom dimensions. These higher-order, more complex behaviors might be influenced by various basal cognitive functions as risky decision-making was influenced by both sustained attention and behavioral inhibition. Considering the heterogeneous results

regarding the associations between sustained attention, behavioral inhibition, other cognitive functions and ADHD (Coghill et al., 2014; Sonuga-Barke et al., 2010; Willcutt et al., 2005), it might be fruitful to reconsider the level of analysis in favor of higher-order cognitive functions instead of solely focusing on the endophenotypes implied in multiple pathway models (Coghill et al., 2014; de Zeeuw et al., 2012; Sonuga-Barke et al., 2010).

Thus, future studies should identify other higher-order psychological functions that might potentially mediate the associations of endophenotypes on ADHD. As deficits in problem solving and planning have been observed in ADHD (Marzocchi et al., 2008; Nigg, Blaskey, Huang-Pollock, & Rappley, 2002) and given the associations of problem solving and planning with behavioral inhibition (Baughman & Cooper, 2007; Miyake et al., 2000), problem solving and planning might be candidates for higher-order cognitive functions. In addition, goal setting and goal pursuit might be cognitive-motivational higher-order candidates, as deficits in goal setting and goal pursuit have been observed in children with ADHD (Gawrilow et al., 2013; Nyman et al., 2010) on the one hand, and have been related to inhibitory processes and the allocation of attention on the other hand (Kruglanski et al., 2002; Locke & Latham, 2002).

5.3.4. The Role of Environmental Factors in Theories of ADHD

The framework presented in this thesis proposes that the inclusion of environmental factors in studies of ADHD would be useful to better understand the development of deficits in endophenotypes and to identify situations in which these deficits lead to symptoms of ADHD.

Indeed, while the current study neither addressed situation-unspecific environmental factors, nor situation specific environmental factors, knowledge about the involvement and efficacy of environmental factors could change the interpretation of the results. For instance, the current study revealed that deficits in sustained attention were related to risky decision-making and symptoms of ADHD, but no final conclusions about the underlying mechanism could be drawn. While the deficits were in line with the DMN hypothesis (Sonuga-Barke & Castellanos, 2007), effects of a non-optimal arousal could not be ruled out (Sergeant, 2002). While both theoretical accounts are not exclusive, testing which might be better in explaining the observed performance would require manipulating the arousal state of the children (e.g., by manipulating inter-trial intervals) and to assess DMN activity on task. In addition, other situation specific environmental factors like perceptual load could influence behavioral

inhibition and attention (Lavie et al., 2004; Lavie, 2010). Hence, RTV and more general deficits in sustained attention could not only been due to internal causes (e.g., arousal, DMN activity) but also external stimuli. However, if external stimuli impair cognitive performance, it is unclear whether this implies a deficit in behavioral inhibition or a deficit in reorientation of attention. For instance, do children with ADHD have problems to inhibit their attention to task-irrelevant distractors or do they have a problem to reorient their attention back to the target, once an irrelevant distractor has captured their attention (see Caseras, Garner, Bradley, & Mogg, 2007 for a similar discussion in research on depression)? To test these hypotheses it would be necessary to manipulate the level of perceptual load during behavioral inhibition tasks and to simultaneously observe the children's eye-movements and fixation times (Caseras et al., 2007).

In addition to experimental manipulations of situation specific environmental factors (e.g., arousal, perceptual load, external distractors), situation specific environmental factors could be directly observed in naturalistic settings. For instance, recent studies investigating the behavior of children with ADHD in the classroom revealed that children with ADHD paid less attention and were more off-task than unaffected children during periods of individual work and whole class group teaching, but not during small group work (Imeraj, Antrop, Sonuga-Barke et al., 2013). Moreover, children with ADHD displayed significantly more hyperactive behavior during periods when they were not actively engaged in the task or waiting for instructions (Imeraj, Antrop, Roeyers et al., 2013). These results from a naturalistic setting match the laboratory findings reported in this study, where an enhanced off-task behavior, as indicated by worse sustained attention (e.g., omission errors), was related to ADHD and to symptoms of hyperactivity in particular.

Thus, combining methods from naturalistic and experimental settings could help to identify situation specific factors associated with deficits in sustained attention, off-task behavior, and hyperactivity.

5.4. Implications for the Practice

After discussing implications for research in the previous section, this section addresses implications for clinical and educational practice. The main results of the current study revealed (1) that sustained attention or off-task behavior might be a core deficit in ADHD and (2) that there are indirect effects of basal cognitive functions (e.g., sustained attention, behavioral inhibition) on ADHD through higher cognitive functions (e.g., risky decision-making). Thus, based on these results two types of trainings or intervention programs could be suggested.

First, trainings or intervention programs could try to improve ADHD symptoms in children by treating a deficit in sustained attention. Several training programs seeking to improve sustained attention have been developed including the practice of cognitive tasks like the CPT (Rabiner, Murray, Skinner, & Malone, 2010; Shalev, Tsal, & Mevorach, 2007; Tamm, Epstein, Peugh, Nakonezny, & Hughes, 2013; for an overview see Sonuga-Barke, Brandeis, Holtmann, & Cortese, 2014), self-alerting procedures (O'Connell et al., 2008; Robertson, Tegnér, Tham, Lo, & Nimmo-Smith, 1995), and intensive meditation (Chambers, Lo, & Allen, 2008; MacLean et al., 2010; but see Chiesa, Calati, & Serretti, 2011). However, the trainings varied in in duration and intensity and only few training programs have been tested in children with ADHD (Sonuga-Barke et al., 2014). Parents reported significantly lower ratings of ADHD and inattentive symptoms in particular, after an eight-week training period (Shalev et al., 2007) or 16 bi-weekly sessions (Tamm et al., 2013) of interventions which were designed to practice sustained, selective, alternating, and divided attention through visual and auditory stimuli.

Further evidence for the effectiveness of sustained attention trainings comes from research on neurofeedback. During neurofeedback participants learn to control their brain electric activity through operant conditioning (Moriyama et al., 2012). For instance, to foster sustained attention children learned to control their theta-to-beta ratio by controlling a dolphin character on a computer screen. When the theta-to-beta ratio decreased reflecting good sustained attention, the dolphin swam down to the button of the ocean and the child received points. In contrast, when the child became distracted, the dolphin swam back to the surface of the ocean (Steiner, Frenette, Rene, Brennan, & Perrin, 2014a, 2014b). Neurofeedback has been shown to improve sustained attention (Wang & Hsieh, 2013) and several studies claimed the effectiveness of neurofeedback in ADHD (Gevensleben et al. 2009; Steiner et al., 2014a, 2014b; for reviews see Lofthouse, Arnold, Hersch, Hurt, & DeBeus, 2012; Moriyama et al.,

2012; for a meta-analysis see Arns, de Ridder, Strehl, Breteler, & Coenen, 2009). However, effectiveness is debated due to a lack of double-blind placebo-controlled studies (Vollebregt, van Dongen-Boomsma, Slaats-Willemse, & Buitelaar, 2014) suggesting that neurofeedback might not be more effective than a placebo-condition (Arnold et al., 2013).

Recently, it has been tried to implement attentional trainings (Steiner et al., 2014b; Steiner, Sheldrick, Gotthelf, & Perrin, 2011) and neurofeedback in the school context (Steiner et al., 2014a, 2014b). However, as the interventions were only implemented in the school building but not during class, teachers will need additional interventions in order to deal with inattention and off-task behavior of children with ADHD during the lessons. Interventions to improve sustained attention, on-task behavior or ADHD symptoms in the classroom include self-monitoring strategies of attention and performance (Harris, Friedlander, Saddler, Frizzelle, & Graham, 2005) and self-regulatory strategies like implementation intentions (Guderjahn et al., 2013). For instance, in a study, children selected a school-related goal that they wanted to achieve and subsequently choose an implementation intention to reach the desired goal (e.g., "If a classmate talks to me, then I will focus more intently on the lesson"). In line with the results of the current study, children most often chose goals to improve their attention and on-task behavior during class (Guderjahn et al., 2013), indicating the importance of sustained attention in ADHD. However, the effectiveness of self-regulatory strategies to improve ADHD symptoms during class is still less clear (Hodgson, Hutchinson, & Denson, 2014) and future studies need to investigate under which conditions and for which children these interventions work best.

However, whereas these trainings and interventions were specifically designed to improve sustained attention, the results of the current thesis suggest another type of intervention as indirect effects have been observed for sustained attention and behavioral inhibition through risky decision-making. The presence of indirect effects is in line with the theoretical assumption that associations between two constructs are maximal when both constructs correspond in nomothetic span (Wittmann & Klumb, 2006). If this principle is transferred to the implementation of interventions, it follows that interventions tapping on specific deficits, such as sustained attention, might be efficient in the treatment of specific deficits in ADHD, but not in reducing overall ADHD symptoms. Instead, broader unspecific interventions could have stronger effects by influencing a larger number of higher cognitive functions as for instance risky decision-making. Indeed, a recent intervention program called TEAMS (Halperin et al., 2013) aimed to improve inhibition, attention, and motor skills through games and physical exercises that adapt to the children's skill level and are fun to

perform. In addition, TEAMS acknowledges the importance of the social context by being administered both within small groups of peers and within families (Halperin et al., 2013). Preliminary results indicated that TEAMS effectively reduced ADHD symptoms according to parent and teacher ratings and the effects persisted for three months (Halperin et al., 2013).

Taken together, results from the current thesis argue for two kinds of intervention programs in ADHD. First, specific interventions (e.g., attention trainings or neurofeedback) to reduce deficits in sustained attention and off-task behavior might treat a core deficit in ADHD. Second, more general interventions might help to reduce ADHD symptoms by capitalizing on indirect effects on ADHD, as several basal functions and higher cognitive functions might be affected simultaneously.

6. Conclusion

The present thesis developed a research framework for ADHD that integrated findings from genetics, neuroscience, and psychology by extending the endophenotype concept of multiple pathway models (Coghill et al., 2014; Sonuga-Barke et al., 2010). Evidence was found for sustained attention as an endophenotypes for ADHD that could be reliably measured across several cognitive tasks. Additional indirect effects could be established for sustained attention and behavioral inhibition on ADHD symptoms through risky decision-making. Supporting the outlined research framework, theories on ADHD should incorporate sustained attention as a core deficit of ADHD. Finally, the predominant endophenotype view should be extended by adding higher-order psychological functions as potential mediators of the effect of cognitive endophenotypes on ADHD.

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Appendix

The appendix contains additional information on the GIDeCA project and in particular on the present study. Section A presents the ethical approvals of the psychological and medical ethical committees. In addition, the section includes the contact materials for the recruitment of participants. Section B comprises supplementary material on the task used in the present study, including the original instructions. Subsequently, Section C offers additional analysis not presented in the results section of this thesis, for instance, comparisons of the performance on the cognitive and behavioral tasks between children with ADHD on and off medication. Furthermore, Section D provides normative solutions for mathematically optimal decision-making in the CCT and NST. Finally, I declare the amount of my personal contribution and the contribution of my colleagues with regard to the presented thesis.

A. Ethical Approvals and Contact Materials

The approvals of the psychological and medical ethical committees are displayed in A.1. and A.2., respectively. Subsequently, the flyer distributed in schools (Section A.3.1.) and the poster presented at local child psychiatric outpatient centers (Section A.3.2.) are presented.

A.1. Psychological Ethical Committee

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Datum: 21.1.2012

Stellungnahme der Ethikkommission
des Fachbereichs 5 Psychologie und Sportwissenschaften

Studie: Differentielle Einflüsse biologischer und psychosozial-familiärer Faktoren auf Belohnungsaufschub und riskantes Entscheidungsverhalten bei Kindern mit unterschiedlichen ADHS-Symptomatiken

Bearbeitungsnummer: 2011-19_R1
Antragsteller: Prof. Dr. Caterina Gawrilow, Prof. Dr. Christine Freitag, Dr. Wolfgang Rauch

Die oben genannte Studie wurde im Regelverfahren der Ethikkommission von zwei ihrer Mitglieder auf ihre ethische Unbedenklichkeit hin begutachtet. Die Kommission ist zu der Überzeugung gelangt, dass auf der Basis der vorliegenden Unterlagen gegen die Studie

- ☐ keine Bedenken bestehen.
- ☒ keine Bedenken bestehen. Die angeführten Empfehlungen sollten jedoch berücksichtigt werden.
- ☐ keine Bedenken bestehen, wenn die angeführten Empfehlungen berücksichtigt werden.
  - ☐ Nach entsprechender Änderung der Prüfunterlagen ist eine erneute Vorlage bei der Ethikkommission nicht erforderlich.
  - ☐ Dazu sind die überarbeiteten Prüfunterlagen teilweise erneut der Ethikkommission zur Prüfung vorzulegen.
- ☐ schwere Bedenken bestehen, die im Einzelnen genannt werden. Die Ethikkommission stimmt dem Antrag nicht zu.
- ☐ die Prüfung der ethischen Unbedenklichkeit zusätzlich von einer medizinischen Ethikkommission durchgeführt werden sollte.

Studie: Differentielle Einflüsse… bei Kindern mit unterschiedlichen ADHS Symptomatiken

Hinweise, Empfehlungen und /oder Auflagen zur genannten Studie

Sehr geehrte Antragsteller,

die Gutachter fanden es angesichts des Umfangs der Testungen in dieser Studie sehr wichtig, auf Freiwilligkeit zu achten, wiederholt und vor jeder Aufgabe nachzufragen, ob das Kind weitherhin mitmachen möchte, und auf etwaige aversive Reaktionen sensibel einzugehen.

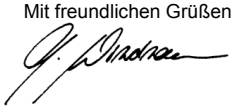
An der Stellungnahme haben mitgewirkt:

☒ Prof. Dr. Sabine Windmann Vorsitzende der Ethikkommission	☐ Prof. Dr. Regina Vollmeyer
☐ Prof. Dr. Tilmann Habermas Stellvertr. Vorsitzender der Ethikkommission	☒ Prof. Dr. Holger Horz
☐ Prof. Dr. Dirk Fabricius	☐ Prof. Dr. Caterina Gawrilow
☐ Dr. Regina Steil	☐ Dr. Wolfgang Rauch
☒ Prof. Dr. Lutz Vogt	☐ Dr. Jens Dallmeyer
☐ Cand. Psych. Rafaela Echle	☐ Cand. Psych. Wiebke Herrmann

Anmerkung:

Die Zustimmung der Ethikkommission entbindet die Studienleiter nicht von ihrer ethischen und juristischen Verantwortung für ihre Studie.

Mit freundlichen Grüßen



(S. Windmann)

Campus Bockenheim • Senckenberganlage 31 • D-60325 Frankfurt am Main

A.2. Medical Ethical Committee

Fachbereich Medizin der
Johann Wolfgang Goethe-Universität
Frankfurt am Main

Universitätsklinikum · Theodor-Stern-Kai 7 · 60590 Frankfurt

Frau
Prof. Dr. Christine Freitag
Direktorin der Klinik für Psychiatrie und
Psychotherapie des Kindes- und Jugendalters
ZPsy

Ethik-Kommission
Der Vorsitzende

Bearbeiterin: Dr. Astrid Gießler/if
Tel.: 069/63 01-45 97
Fax: 069/63 01-83434
E-Mail: ethikkommission@kgu.de

Donnerstag, 9. Februar 2012

Geschäfts-Nr.: 38/12 (Bitte stets angeben!)
Titel: Differentielle Einflüsse biologischer und psychosozial-familiärer Faktoren auf Belohnungsaufschub und riskantes Entscheidungsverfahren bei Kindern mit unterschiedlichen ADHS-Symptomatiken

ZWEITVOTUM

Geschäftsführung der
Ethik-Kommission
Tel.: 069/63 01-4597

Sekretariat der
Ethik-Kommission
Tel.: 069/63 01-7239

http://ethik-kommission.klinik.uni-frankfurt.de

Sehr geehrter Herr Professor Freitag,

gemäß den innerbetrieblichen Anweisungen für die Ärztinnen und Ärzte des Universitätsklinikums Frankfurt a. M. über die Durchführung klinischer Studien haben Sie mit Schreiben vom 02.02.2012 Ihre Beteiligung an der o. g. multizentrischen Studie angezeigt.

Nach § 15 Abs. 1 der Hessischen Berufsordnung müssen sich die Ärztinnen und Ärzte vor Durchführung biomedizinischer Forschung am Menschen und epidemiologischen Forschungsvorhaben durch eine bei der Ärztekammer oder bei einem medizinischen Fachbereich gebildeten Ethik-Kommission über die mit dem Vorhaben verbundenen berufsethischen und berufsrechtlichen Fragen beraten lassen, sofern das betreffende Projekt nicht bereits durch eine der vorgenannten Ethik-Kommissionen beraten wurde.

Da die Ethik-Kommission des Fachbereichs Psychologie und Sportwissenschaften der J. W. Goethe-Universität Frankfurt dieses Vorhaben bereits beurteilt hat, ist eine nochmalige Beratung durch die hiesige Ethik-Kommission nicht erforderlich, jedoch satzungsgemäß eine Überprüfung möglich.

Prinzipiell schließen wir uns dem Votum an mit folgenden Hinweisen:

- Auf dem Studienprotokoll steht Frau Prof. Freitag als Studienleiterin. Auf allen anderen Dokumenten steht Frau Prof. Gawrilow als Studienleiterin. Wir gehen davon aus, dass Frau Prof. Gawrilow Studienleiterin ist, da auch das Erstvotum von der für sie zuständigen Ethikkommission erteilt wurde.
- Die Einwilligungserklärung muss von beiden Erziehungsberechtigten unterschrieben werden.

Die Ethik-Kommission des Fachbereichs Medizin hat beschlossen, nur Meldungen von schwerwiegenden oder unerwarteten unerwünschten Ereignissen zu bearbeiten, wenn sie die für den verantwortlichen Studienleiter zuständige Kommission ist oder wenn eine qualifizierte Stellungnahme des LKP's vorliegt.

Eine Information über den Abschluss der Studie wird erbeten.

Mit freundlichen Grüßen

Prof. Dr. med. Sebastian Harder
Vorsitzender der Ethik-Kommission

Die Stellungnahme der Ethik-Kommission erfolgte aufgrund folgender eingereichter Unterlagen:

Dokument:	**Version/ Nr.:**	**datiert vom:**
Protokoll	Version 1	02.02.2012
Deckblatt zur Antragstellung für Studien außerhalb des AMG		02.02.2012
Informationsbogen (UG) Eltern	Version 1	02.02.2012
Einwilligungserklärung der Eltern (UG)	Version 1	02.02.2012
Patienteninformation und Einwilligungserklärung	Version 1	02.02.2012
Votum des FB Psychologie und Sportwissensch. der Universität Frankfurt		21.01.2012
Antrag auf Prüfung eines Forschungsprojektes durch die Ethik-Kommission des Fachbereichs 5 der Goethe Universität Frankfurt inkl. Anlagen		ohne Datum

Was ist ADHS?

Etwa 5-10 % der Kinder und Jugendlichen in Deutschland sind von einer Aufmerksamkeitsdefizit-/Hyperaktivitätsstörung (ADHS) betroffen. Diese Kinder und Jugendlichen sind unaufmerksam und zappelig und haben meist eine eingeschränkte Fähigkeit zur Selbstregulation, d.h. sie können ihr Verhalten, ihre Gefühle und ihre Gedanken nicht gut kontrollieren.

Diese Schwierigkeiten führen bei Kindern und Jugendlichen mit ADHS oft dazu, dass sie in der Schule hinter anderen Schüler/innen zurückbleiben. Darüber hinaus haben sie oft Schwierigkeiten, Freundschaften zu knüpfen und aufrechtzuerhalten und im Umgang mit Eltern und Lehrkräften kann es ebenfalls zu Problemen kommen.

Haben Sie noch weitere Fragen? Dann kontaktieren Sie uns!

IDeA – Individuelle Entwicklung und Lernförderung

Ein Zentrum des DIPF (Deutsches Institut für Internationale Pädagogische Forschung) und der Goethe-Universität in Kooperation mit dem Sigmund-Freud-Institut Frankfurt am Main.

Projekt **GIDeCA**

Ansprechpartner: Dipl.-Psych. Tilman Reinelt
Dipl.-Psych. Andrea Wirth

Projektverantwortliche: Prof. Dr. Caterina Gawrilow
Prof. Dr. Christine M. Freitag
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Prof. Dr. Andreas Gold
Prof. Dr. Eckhard Klieme
Prof. Dr. Wolfgang Schneider

Gefördert durch: LOEWE
Exzellente Forschung für Hessens Zukunft

IDeA
Individuelle Entwicklung und Lernförderung

Wie Kinder lernen

Projekt **GIDeCA**

Biologische und familiäre Einflüsse auf kindliches Entscheidungsverhalten

Was ist GIDeCA?

Das Projekt GIDeCA (**G**ene by Environment **I**nteractions on **De**cision Making in **C**hildren with **A**DHD) untersucht, welche biologischen und familiären Faktoren die Entscheidungsprozesse von Kindern mit und ohne ADHS beeinflussen.

Im Rahmen des Projektes GIDeCA möchten wir Ihr Kind bitten, einfache Computerspiele, Kartenspiele und Würfelaufgaben zu bearbeiten, die das kindliche Entscheidungsverhalten erfassen. Um die biologischen Einflussfaktoren zu betrachten, bitten wir Ihr Kind darüber hinaus, eine Speichelprobe abzugeben. Außerdem möchten wir Sie bitten, verschiedene Fragebögen auszufüllen, gemeinsam mit Ihrem Kind eine Aufgabe zu bearbeiten und uns ein paar Fragen zu beantworten.

Diese Forschung kann einen wertvollen Beitrag zum Verständnis der Einflussfaktoren und der Entwicklung von ADHS liefern.

Wie groß ist der Aufwand für Sie?

Sie kommen gemeinsam mit Ihrem Kind für zwei Termine zu uns ins IDeA-Zentrum in die Räumlichkeiten der Goethe-Universität Frankfurt am Main auf den Campus Bockenheim. Die beiden Termine liegen dicht beieinander und werden persönlich mit Ihnen vereinbart.
Jeder der Termine dauert höchstens zwei Stunden.

Teilnehmen können Kinder mit und ohne ADHS im Alter von 8 bis 12 Jahren.

Ihre Daten werden selbstverständlich anonym behandelt und nur für wissenschaftliche Zwecke im Rahmen dieser Studie verwendet und nicht an Dritte weitergegeben.

Was bringt Ihnen die Teilnahme?

Über Ihre Unterstützung bei unserem Projekt freuen wir uns sehr. Da Sie uns mit Ihrem Engagement helfen, das Entscheidungsverhalten von Kindern besser zu verstehen, möchten wir uns bei Ihnen für Ihre Teilnahme bedanken:

Ihr Kind erhält eine Urkunde über die Teilnahme an der Testung sowie ein kleines Geschenk. Außerdem bekommen Sie einen Gutschein in Höhe von 20 Euro als Dankeschön für die Teilnahme.

Wir informieren Sie nach Abschluss der Studie bei Interesse gerne über die Ergebnisse des Projektes. Bitte beachten Sie jedoch, dass wir Ihnen aufgrund der Anonymisierung der Daten keine persönliche Rückmeldung geben können, sondern lediglich über die Gesamtergebnisse informieren können.

IDeA
Individuelle Entwicklung und Lernförderung

Wie Kinder lernen

DIPF

Bildungsforschung und Bildungsinformation

Projekt GIDeCA
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Was beeinflusst ADHS?

Studienteilnehmer gesucht!

Kinder mit einer Aufmerksamkeitsdefizit-/Hyperaktivitätsstörung (ADHS) sind besonders unaufmerksam und zappelig und haben dadurch oft Probleme beim Lernen und in der Schule. In unseren Forschungsprojekten versuchen wir herauszufinden, welche Faktoren ADHS beeinflussen, damit diesen Kindern bestmöglich geholfen werden kann. Insbesondere interessiert uns, wie biologischen Faktoren und Erziehungsverhalten zusammenwirken und sich wechselseitig bei der Ausprägung der Störung beeinflussen.

Wer kann teilnehmen?

- Jungen und Mädchen im Alter von **8 bis 12 Jahren mit und ohne ADHS**.

Was macht man da?

- Sie machen zwei Termine mit uns an der Universität (Bockenheimer Warte) aus.
- Kinder: bearbeiten mehrere Computeraufgaben sowie Kartenspiel- und Würfelaufgaben
- Eltern: Durchführung eines Interviews, Ausfüllen von Fragebögen, Bearbeitung einer Aufgabe gemeinsam mit dem Kind
- Außerdem: Das Kind wird gebeten eine kleine Speichelprobe abzugeben
- Dauer: **Zwei Termine à ca. 2 Stunden**.
- Belohnung: **20€** und ein kleines **Geschenk.**
- Eltern werden über die Ergebnisse der Studie informiert.
- Alle Daten werden **anonym** und **streng vertraulich** behandelt.

Bei Fragen und Interesse melden Sie sich bitte bei:

Ansprechpartner: Dipl. Psych. Tilman Reinelt & Dipl. Psych. Andrea Wirth
Tel. 069/24708-802 E-Mail: gideca@idea-frankfurt.eu

B. Supplementary Information About Tasks

This section first gives an overview of the tasks and questionnaires used in the GIDeCA project (Table B2). In addition, the order of trials in the CCT is provided (Table B1) and finally, the original German instructions for all tasks and questionnaires the children and parents receive during both experimental sessions are presented (Section B.1.).

Table B1

Order of Trials in the CCT.

Trial Number	Gain Amount	Loss Amount	Number of Loss Cards
1 & 2	1	10	1
3 & 4	1	10	2
5 & 6	5	40	1
7 & 8	5	40	2
9 & 10	5	10	1
11 & 12	5	10	2
13 & 14	1	40	1
15 & 16	1	40	2

Table B2

Overview of all Measures Used in the GIDeCA Project and Their Approximate Durations

Session I				Session II			
Children		Parents		Children		Parents	
Task/Questionnaire	Time	Task/Questionnaire	Time	Task/Questionnaire	Time	Task/Questionnaire	Time
CFT 20-R	20	Expressed emotion	5	2-CR	5	ISBI	30
MIDA	10	Kinder-DIPS	45	DRT	5	APQ	10
CCT	20	CBCL	20	Number span	5	FRQ	5
CPT	15	SDQ	5	Tapping	5	ZKE-P	5
NST	5	FBB-ADHS	5	Go/No-Go	15	CHAOS	5
Flanker	15	SCS-P	15	GDT	15	JTCI	10
Saliva sample	10	WURS-K	5	DeFT	10	SCS-C	5
BIS/BAS	10	SCL-90-R	5	Duration discrimination	10	PS	5
Geo cubes	7	Geo cubes	7	ZKE-C	10	PSI	10
Jenga tower	7	Jenga tower	7	Delay of gratification	25		

Note. 2-CR = 2-Choice Reaction Time Task; APQ = Alabama Parenting Questionnaire; BIS/BAS = Behavior Inhibition/Behavior Activation Scale; CBCL = Child Behavior Checklist; CCT = Columbia Card Task; CFT 20-R= Culture Fair Test 20-R; CHAOS = Confusion, Hubbub, and Order Scale; CPT = Continuous Performance Task; DeFT = Delay Frustration Task; DRT = Delay Reaction Time Task; FBB-ADHS = German ADHD Rating Scale; FRQ = Family Routine Rating Questionnaire; GDT = Game of Dice Task; ISBI = IDeA Social Background Inventory; JTCI = Junior Temperament and Character Inventory; MIDA = Maudsley's Index of Childhood Delay Aversion; NST = No Six Task; PS = Parenting Scale; PSI = Problem Solving Interview; SCL-90-R = Symptom Checklist 90-R; SCS-C = Self-Control Scale, child version ; SCS-P = Self-Control Scale, parent version; SDQ = Strength and Difficulties Questionnaire; WURS-K = Wender Utah Rating Scale; ZKE-C = Zurich Brief Questionnaire for the Assessment of Parental Behavior, child version; ZKE-P, parent version = Zurich Brief Questionnaire for the Assessment of Parental Behavior.

B.1.1. Instructions Children Session I

GIDeCA

Gen-Umwelt-Interaktionen auf Entscheidungsfindung bei Kindern mit und ohne ADHS

Instruktionen für Testleiter

Session I

Stand: 01.03.2013

Studienleitung:

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Begrüßung

Guten Tag Frau/Herr XY, hallo XY. Schön, dass Sie und dass du da sind. Mein Name ist XYZ und ich und ein/e Kollege/in werden diese Studie mit Ihnen durchführen. Dann gehen wir erst einmal in unsere Testräume.

Im Testraum:

Bevor wir mit der eigentlichen Testung anfangen, möchte ich mit dir dieses Informationsblatt durchgehen. Das hatten wir auch bereits deiner Mutter zugeschickt. Aber ich weiß nicht, ob Sie darüber mit dir gesprochen hat. ***Informationsblatt mit dem Kind durchgehen***

Außerdem habe ich hier eine Einverständniserklärung, die du bitte ausfüllst – wenn du damit einverstanden bist, was wir machen. ***Mit dem Kind die Einverständniserklärung durchgehen***

Vielen Dank, dass du bei unserer Studie mitmachst. Wir werden heute verschiedene Aufgaben und Spiele machen. Einige davon werden hier am Computer stattfinden, andere an dem Tisch dort drüben. Ich möchte dich bitten, dass du dich bei allen Aufgaben so viel wie möglich anstrengst. Wenn du Fragen hast oder eine Anleitung nicht gleich verstehst, kannst du mich jederzeit fragen.

Wir möchten auch gerne schauen, wie deine Bewegungen während der Aufgaben sind. Das können wir mit diesem Gerät hier messen ***(Actigraph zeigen)***. Dazu müssten wir das an deinem Hosenbund befestigen. Aber bevor wir das machen, müssen wir den erst einmal richtig einstellen.

Einstellung Actigraph

1. Actigraph an den Laptop anschließen
2. Starten von ActiLife5 (auf dem Desktop)
3. Reiter „*Devices*“ aktivieren und auf „**Initialize**“ klicken
4. Sample Rate auf 100 Hz stellen; Startzeit und Stoppzeit (Startzeit + 3h) angeben
5. *Enter Subject Info*: VP Nummer, Geschlecht, **Länge & Gewicht**, Geburtsdatum, **Limb: Waist**, Nicht-Dominanz einstellen
6. **Actigraph an der Nicht-Dominaten Seite befestigen oder von Eltern anlegen lassen**

Einführung Spielezettel

Jetzt bekommst du aber erstmal deinen Spielezettel. Darauf sind alle Aufgaben und Spiele vermerkt, die wir heute machen und du bekommst für jede geschaffte Aufgabe einen Stempel. Den Spielezettel darfst du am Ende mit nach Hause nehmen.

1. CFT-20-R Intelligenztest für Kinder

Material:

CFT-Antwortheft

Stoppuhr

Instruktion:

- Als erstes werden wir verschiedene Rätselaufgaben machen.
- Das Rätsel besteht dabei aus vier Teilen.
- Jeder Teil fängt leicht an und wird dann immer schwerer.
- Es ist ganz normal, wenn du nicht alle Aufgaben lösen kannst.
- Halte dich nicht zu lange an einer Aufgabe auf und versuche möglichst viele Aufgaben zu lösen.
- Wenn du dir bei einer Lösung nicht ganz sicher bist, dann wähl die Lösung, die dir am besten erscheint. Alle Aufgaben haben nur eine richtige Lösung.
- Für die Bearbeitung der Aufgaben steht eine begrenzte zeit zur Verfügung.
- Wenn ich “HALT” rufe, dann hör bitte sofort auf zu arbeiten und blätter bis zum nächsten Stopp-Zeichen. Dann beginnt der nächste Teil.
- Hast du noch Fragen?

Instruktion Teil 1

- Schau jetzt bitte auf die nächste Seite, wo die drei Beispiele zu Teil 1 stehen – *Kontrollieren, ob richtige Seite aufgeschlagen ist*
- Zeig mit dem Finger auf das erste Beispiel in der ersten Zeile – *wenn falsch, selber drauf zeigen*
- Kannst du sehen, dass der dicke schwarze Strich in den drei ersten Kästechen immer länger wird?

- Du sollst jetzt von den fünf Kästchen rechts danben das Kästchen angeben, das an die Stelle des leeren Kästchens gesetzt werden muss – *auf leeres Kästchen zeigen*
- Bei diesem ersten Beispiel ist a) die richtige Lösung. Es ist der ganz lange Strich im ersten Kästchen, weil dieser noch länger ist als die anderen.
- Die Lösung ist auf dem Zettel bereits angekreuzt.
- Hast du Fragen dazu?
- Dann zeig jetzt bitte auf die zweite Zeile. Man sieht, dass die kleine gebogene Linie erst nach links, dann nach rechts und dann wieder nach links gebogen ist.
- Kannst du mir sagen, wie die nächste gebogene Linie aussehen muss.
    - *Wenn falsch:* Nein, das ist nicht richtig; versuch es noch einmal.
    - *Wenn richtig:* Ja, sie muss nach rechts gebogen sein. Kreuz jetzt bitte die richtige Lösung auf dem Zettel an. Welche Lösung ist richtig?
        - *Wenn falsch:* Nein das ist nicht richtig, versuch es noch einmal.
        - *Wenn richtig:* Ja, Buchstabe c) ist richtig.
- Schau jetzt bitte auf die dritte Zeile. Stell dir vor, dass sich dieses kleine schwarze Dreieck wie der Zeiger einer Uhr dreht. Es fängt oben an und bewegt sich immer weiter runter.
- Such jetzt nach der richtigen Antwort und kreuz sie an. Welche Antwort ist richtig?
    - *Wenn falsch:* Nein das ist nicht richtig, versuch es noch einmal.
    - *Wenn richtig:* Ja, richtig. Kästchen a).

- Die Aufgaben, die du nun lösen sollst, stehen auf den nächsten beiden Seiten – *NICHT umblättern lassen*

- In jeder Reihe soll immer diejenige der fünf Figuren von der rechten Seite ausgewählt werden, die zu den drei Figuren auf der linken Seite am besten passt.
- Auf den beiden nächsten Seiten findest du 15 Aufgaben, die genauso gelöst werden sollen, wie wir das eben geübt haben.

- Bitte noch nicht umblättern, ich muss erst noch etwas erklären
- Wenn du die Antwort, die du angekreuzt hast, verbessern willst, so streich das Kreuz durch und kreuze die richtige Antwort an.
- Du wirst wahrscheinlich nicht genug Zeit haben, alle Aufgaben zu bearbeiten. Arbeite aber bitte so schnell und so sorgfältig wie möglich.
- Wenn eine Aufgabe zu schwierig ist, so kannst du sie auch überspringen und gleich zur

nächsten Aufgabe weitergehen.

- Du kannst deine Antworten so lange berichtigen, bis ich "HALT" sage. Dann ist Schluss.
- Hast du noch Fragen? – *auf Fragen dürfen nur Teile der obigen Anweisungen wiederholt werden; außer es geht gar nicht anders*
- Dann blätter jetzt bitte um und fang mit den Aufgaben an. Wenn du mit den Aufgaben auf der Seite fertig bist, machst du auf der nächsten Seite weiter, bis ich "HALT" sage.
- Los geht's – ***Hinter die Trennwand zurückziehen und Kind durch die Kamera beobachten!***
- *– Kind gegebenenfalls daran erinnern, dass der Test zwei Seiten hat*
- ***Nach genau 4 Minuten****:* HALT. Mach bitte einen Strich unter die Aufgabe, die du gerade bearbeitet hast, leg deinen Stift zur Seite und blätter bis zum nächsten Stoppzeichen *kontrollieren*.
- Du hast viele Aufgaben gelöst. Nun kommen wir zum zweiten Teil.

- Blätter nun bitte um. Auf der nächsten Seite findest du die Übungsbeispiele zum zweiten Teil.
- Zeig bitte mit dem Finger auf die oberste Zeile.
- Du siehst, dass vier Figuren in irgendeiner Weise gleich sind, eine unterscheidet sich dagegen von den anderen.
- In diesem Teil geht es also darum herauszufinden, welches Kästchen sich in irgendeiner Weise von den anderen unterscheidet.
- Im ersten Beispiel ist die Antwort schon gegeben. Welche ist es? *Antwort abwarten*
- Ja, es ist das vierte Kästchen. Und warum? *Antwort abwarten*
- Genau, weil der Balken aufrecht steht und die anderen nicht.

- Nun, wie ist es bei der zweiten Zeile?
- Welche Figur unterscheidet sich hier von den anderen? *Antwort abwarten*
- Ja, es ist die erste. Sie ist schwarz, während alle anderen weiß sind. Die anderen sind zwar auch verschieden groß, sie sind aber alle weiß und so kann man von ihnen keine besonders herausheben.
- Du musst also Antwort a) ankreuzen. *Kontrollieren*
- In jeder Reihe soll man also herausfinden, welche der Figuren in den fünf Kästchen sich von den anderen vier Figuren in irgendeiner Weise unterscheidet, also nicht zu den anderen Figuren passt.

- Es kann immer nur ein Kästchen in Frage kommen.
- Bitte beachte, dass die Aufgaben wieder auf zwei Seiten stehen. Wenn du mit der ersten Seite fertig bist, dann arbeite gleich auf der nächsten Seite weiter.
- Arbeite bitte zügig und sorgfältig und versuche so viele Aufgaben wie möglich zu lösen, bis ich HALT sage.
- Hast du noch Fragen?
- Dann blätter jetzt bitte um und fang mit den Aufgaben an.
- ***Nach genau 4 Minuten****:* HALT. Mach bitte einen Strich unter die Aufgabe, die du gerade bearbeitet hast, leg deinen Stift zur Seite und blätter bis zum nächsten Stoppzeichen *kontrollieren*.
- Du hast viele Aufgaben gelöst. Nun kommen wir zum dritten Teil.

- Blätter nun bitte um. Auf der nächsten Seite findest du die Übungsbeispiele zum dritten Teil.
- Zeig bitte mit dem Finger auf das große Viereck, in dem die großen schwarzen Punkte sind. *Kontrollieren*
- Du siehst, ein Kästchen im Viereck ist leer geblieben. Welches der fünf Kästchen rechts daneben gehört hier hinein? *Antwort abwarten*
- Ja, es ist das dritte, weil dieses Kästchen am besten in das große Viereck hineinpasst und es richtig vervollständigt.
- Nun sieh auf das zweite Beispiel.
- Welches Kästchen gehört hier in das leere, damit das Viereck vollständig ist? *Antwort abwarten*
- Ja, es ist das erste.
- Nun nehmen wir uns das dritte Beispiel vor. Welches ist hier die richtige Antwort? *Antwort abwarten*
- Richtig, die vierte, also d).
- Bei jeder Aufgabe soll also rechts ein Kästchen mit der Zeichnung ausgewählt werden, die in das leere Kästchen links am besten hineinpasst, um den Kasten richtig zu vervollständigen.
- Diesmal sind es wieder 15 Aufgaben, die auf den drei nächsten Seiten auf diese Wiese gelöst werden sollen.
- Versuch möglichst viele davon zu lösen.
- Hast du noch Fragen?

- Dann blätter jetzt bitte um und fang mit den Aufgaben an – *aufpassen, dass Aufgaben dieses Mal auf drei Seiten stehen*
- ***Nach genau 3 Minuten****:* HALT. Mach bitte einen Strich unter die Aufgabe, die du gerade bearbeitet hast, leg deinen Stift zur Seite und blätter bis zum nächsten Stoppzeichen *kontrollieren*.
- Du hast viele Aufgaben gelöst. Nun kommen wir zum vierten Teil.

- Blätter nun bitte um. Auf der nächsten Seite findest du die Übungsbeispiele zum vierten Teil.
- Du siehst hier im obersten Kästchen links einen Kreis, einen Punkt und ein Viereck.
- Der Punkt ist im Kreis aber außerhalb des Vierecks
- Nun sieh in den Kästchen rechts daneben nach, wo du einen Punkt in einen Kreis hineinsetzen kannst, ohne dass er gleichzeitig im Viereck liegt.
- Wie steht es mit dem ersten Kästchen a)? *Pause*
- Hier geht es nicht, weil ein Punkt im Kreis auch gleichzeitig im Viereck liegen würde.
- Würde es beim zweiten gehen? *Pause*
- Auch nicht, ein Punkt im Kreis würde auch hier wieder im Viereck liegen.
- Und beim dritten Kästchen c)? *Pause*
- Richtig, hier ist der Punkt im Kreis, aber nicht im Viereck. Zeichne den Punkt bitte nun ein, damit man ihn sehen kann.
- Bei diesem ersten Beispiel ist also c) die richtige Lösung, weil du nur hier den Punkt in den Kreis setzen kannst, ohne dass er gleichzeitig im Viereck liegt
- Bei den folgenden Beispielen und Aufgaben sollst du aber wieder nur die richtige Lösung ankreuzen.
- Das große viereckige Kästchen hat keine Bedeutung. Es ist nur der Rahmen, der bei allen Aufgaben gleich ist.

- So, nun sieh dir in der zweiten Zeile das Kästchen genau an.
- Hier ist der Punkt im Ei, aber unter der Linie.
- Wir müssen jetzt ein Kästchen suchen, in dem ein Punkt auch im Ei, aber runter der Linie liegen würde. Welches ist es? *Antwort abwarten*
- Ja, das zweite, also b). Und das ist das einzig richtige.

- Nun die dritte Zeile – Diesmal muss der Punkt in beiden Vierecken zugleich liegen, aber außerhalb des Kreises. - *Pause*

- Im ersten Bild bei a) könnte man den Punkt zwar zugleich in beide Vierecke setzen, aber dann würde er auch im Kreis liegen – *Pause*
- Im Kästchen b) könnte man den Punkt zwar auch zugleich in beide Vierecke setzen, aber auch hier würde der Punkt im Kreis liegen und das soll er ja nicht.
- Und das Kästchen c)? – *Pause*
- Ja, das ist das einzig richtige, bei dem man den Punkt gleichzeitig in beide Vierecke, aber nicht in den Kreis setzen könnte.
- Kreuz also Kästchen c) an
- Bei jeder Aufgabe soll man also zunächst genau hinsehen, wo der Punkt liegt und dann unter den 5 Auswahlfiguren diejenigen heraussuchen, in welcher der Punkt genauso liegen könnte.
- Bei einigen Aufgaben sind jedoch 2 oder 3 Punkte vorhanden.
- Diesmal sind nur 11 Aufgaben auf den beiden nächsten Seiten zu bearbeiten.
- Hast du noch Fragen?
- Dann blätter jetzt bitte um und fang mit den Aufgaben an
- ***Nach genau 3 Minuten****:* HALT. Mach bitte einen Strich unter die Aufgabe, die du gerade bearbeitet hast und leg deinen Stift zur Seite
- Du hast diesen Teil geschafft.
- Als nächstes spielen wir ein Spiel am Computer.

2. Maudsley Index of Childhood Delay Aversion (MIDA)

Material:

Computer

Computermaus

Starten des Experimentes

- Öffne aus dem GIDeCA-Ordner den Unterordner „MIDA“ und starte die Aufgabe durch Doppelklick auf das Icon „Delay Aversion“
- Klicke auf SETUP und wähle Edit Setup
- Checke, ob der Haken bei „No Post Delay“ gesetzt ist
- Gib in Number of Trials per session ein: **2**
- Drücke auf weiter

Instruktion

- Bei diesem Spiel bist du der Kapitän eines Raumschiffes, das von feindlichen Raumschiffen angegriffen wird.
- Für jedes feindliche Raumschiff, das du abschießt, erhältst du Punkte
- Dein Raumschiff bewegt sich von links nach rechts über den Bildschirm
- Wenn sich die Box „Zielbereich“ rot färbt, kannst du die feindlichen Raumschiffe abschießen. Dafür musst du die linke Maustaste drücken
- Pro Spieldurchgang hast du nur einmal die Möglichkeit zu schießen
- Wenn du auf das einzelne feindliche Raumschiff ganz links schießt, erhältst du einen Punkt und die Runde ist danach beendet.
- Wenn du das einzelne feindliche Raumschiff nicht abschießt und wartest, bis du die beiden anderen feindlichen Raumschiffe ganz rechts zusammen abschießen kannst, erhältst du zwei Punkte
- Wenn du in einer Spielrunde nicht schießt, erhältst du keinen Punkt.
- Die Anzahl der in der aktuellen Runde gesammelten Punkte wird dir unter Punkte angezeigt
- Die gesammelten Punkte aus allen Spielrunden siehst du unter Gesamtpunkte
- Wir machen jetzt einen Probedurchgang - *Gib nun ID = 0 ein*
- Schieße nun auf das einzelne feindliche Raumschiff, sobald die Box rot wird
- Dieses Mal schießt du nicht auf das einzelne feindliche Raumschiff, sondern wartest

bis du die beiden anderen zusammen abschießen kannst.

- Wie viele Punkte bekommst du, wenn du das erste Raumschiff abschießt?
- Wie viele Punkte bekommst du, wenn du die beiden Raumschiffe abschießt?
- Musst du länger warten mit dem Abschießen, wenn du einen oder zwei Punkte erhalten möchtest?
- *Wenn das Kind die Fragen falsch beantwortet, dann klären und gegebenenfalls den Probedurchgang noch einmal starten*
- Ich starte gleich das Spiel. Es gibt einen Unterschied zum Probedurchgang: Dieses Mal kannst du in jeder Spielrunde selbst entscheiden, ob du schießt, wenn die Box das erste Mal rot wird, oder wenn sie das zweite Mal rot wird.
- Ziel des Spiels ist es, so viele Punkte wie möglich zu sammeln.
- Während des Spiels gehe ich hinter die Trennwand und werde nicht mit dir reden.
- Hast du noch Fragen?
- Dann starte ich nun das Spiel
- *Klicke auf SETUP und wähle Edit Setup*
- *Checke, ob der Haken bei „No Post Delay“ gesetzt ist*
- *Gib in Number of Trials per session ein:* ***20***
- *Drücke auf weiter und gib die ID des Kindes ein*

3. Columbia Card Task (CCT – hot Version)

Material

Vorgemischte SkipBo-Karten (d.h. Verlustkarte à 10 Minuspunkte und 11 Gewinnkarten à 1 Punkt, Verlustkarte liegt oben auf dem verdeckten Stapel)
Kartenmischmaschine
Infotafel
Laminierte Zettel (16 Stück)
abwischbaren Stift
Ergebnisbogen

Instruktion

- Als Nächstes spielen wir ein Kartenspiel. Auch hier kannst du wieder Punkte gewinnen.
- Als Startguthaben erhältst du 400 Punkte.
- In jeder Runde werde ich 12 Karten auf den Tisch legen – *die 12 Karten verdeckt austeilen, 3 Reihen à 4 Karten* -. Du darfst dann einzelne Karten umdrehen– *einzelne Karten umdrehen* -. Jede Karte ist entweder eine Gewinn- oder eine Verlustkarte, d.h. entweder du gewinnst dabei Punkte oder du verlierst welche.
- Jedes Mal wenn du eine Gewinnkarte aufdeckst, erhältst du so viele Pluspunkte wie die Zahl auf der Karte und du darfst danach eine weitere Karte aufdecken. Musst du aber nicht.
- Das heißt: Nach jeder aufgedeckten Karte kannst du dich entscheiden, ob du aufhören willst um die Punkte sicher zu bekommen oder ob du weiterzuspielen möchtest und so vielleicht noch mehr Punkte gewinnen kannst.
- Wenn du dann aber eine Verlustkarte aufdeckst, werden dir Punkte abgezogen und die Runde ist beendet.
- Insgesamt spielen wir 16 Runden.
- Alle zwei Runden werden wir jedoch etwas an den Bedingungen verändern. Das zeigt dir immer diese Tafel hier an – *auf die Tafel zeigen.*
- Hier siehst du die Gewinnkarten: Die Gewinnkarten *(VL zeigt auf die Gewinnkarten)* können entweder einen oder 5 Punkte wert sein.
- Hier siehst du die Minuspunkte: Wenn du eine Verlustkarte *(VL zeigt auf die Verlustkarten)* ziehst, kannst du entweder 10 oder 40 Punkte verlieren.

- Hier siehst du, wie viele Verlustkarten im Spiel sind: In einigen ist genau eine der Karten eine Verlustkarte. In anderen Runden sind zwei der Karten Verlustkarten.
- Wir werden das jetzt an einem Beispiel durchgehen:
- *Die Karten in 3 Reihen à 4 Karten austeilen. Dabei sind eine Verlustkarte mit 10 Minuspunkten und 11 Gewinnkarten mit 1 Pluspunkt im Stapel. Die Verlustkarte wird aus Sicht des Versuchsleiters in die linke obere Ecke gelegt (-> Karten müssen vorher entsprechend gemischt sein, dass oberste Karte des Stapels die Verlustkarte ist!!!)*
- *Auf die Infotafel zeigen* – Im ersten Beispiel siehst du, dass **eine** der 12 Karten eine Verlustkarte ist. Die Gewinnkarte ist **einen** Punkt wert. Das Aufdecken der Verlustkarte bedeutet in dieser Runde, dass du **10** Punkte verlierst.
- Du würdest jetzt anfangen, Karten aufzudecken. Lass uns mal schauen was passiert! *Erste Karte aufdecken* – Jetzt hättest du also einen Punkt gewonnen. Du könntest dich jetzt entscheiden, ob du weiterspielen und eine weitere Karte aufdecken möchtest oder ob du lieber aufhörst und deine Punkte sicherst. Wenn du weiterspielst, darfst du eine weitere Karte aufdecken.
- *Zweite Karte aufdecken.* Jetzt hättest du zwei Punkte. Jetzt musst du dich wieder entscheiden: Willst du weiterspielen oder willst du aufhören und deine Punkte sichern? Wenn du weiterspielen willst, darfst du die nächste Karte aufdecken.
- *Dritte Karte aufdecken.* Jetzt hättest du drei Punkte. Und wieder müsstest du dich jetzt entscheiden: Weiterspielen oder aufhören und die Punkte sichern. Ich spiele noch einmal weiter.
- *Nun die Verlustkarte aufdecken.* Mist, dass ist die Verlustkarte. Dir werden also 10 Punkte abgezogen. Das heißt: Du hattest drei Punkte und jetzt werden 10 Punkte abgezogen. Insgesamt verlierst du also 7 Punkte.
- Das waren jetzt ganz schön viele Regeln. Deshalb spielen wir erst einmal eine Proberunde
- *Karten austeilen: 1 Verlustkarte mit 10 Minuspunkte, 11 Gewinnkarten mit 5 Punkte*
- *auf die Infotafel zeigen:* Achte bitte auf das, was hier steht. Jede Gewinnkarte bringt dir also **5** Punkte, es gibt **eine** Verlustkarte und diese Verlustkarte zählt **10** Minuspunkte.
- Du darfst jetzt so viele Karten aufdecken wie Du möchtest. Du kannst die Runde jederzeit beenden und deine Punkte sichern. Wenn du aber eine Verlustkarte aufdeckst, werden dir die entsprechenden Punkte abgezogen und die Runde ist beendet. Los geht's!“

- *Kind deckt die Karten auf, am Ende das Ergebnis mitteilen.*
- Bevor wir mit dem eigentlichen Spiel beginnen noch ein paar kurze Fragen: *letzte Infotafel aufdecken.* Guck einmal auf diese Tafel: Wie wären jetzt die Bedingungen? *Wenn Kind nicht korrekt antwortet, dann nochmals erklären.*
- Nehmen wir an, du hast sehr viel Glück und du schaffst es, alle Gewinnkarten aufzudecken, ohne eine Verlustkarte zu erwischen, wie viele Gewinnkarten würdest du dann aufdecken?“
- *Kind antwortet korrekt (11 Karten)* ➔ *VL:* Genau
- *Kind zögert* ➔ *VL erklärt nochmals die Infotafel, stellt die Frage erneut und antwortet wenn die richtige Antwort kommt:* Genau
- Angenommen du hättest in dieser Runde einige Karten aufdecken wollen, aber gleich die erste Karte war eine Verlustkarte.Wie viele Karten wären dann aufgedeckt worden? Was würde dann passieren?
- *Kind antwortet korrekt (nur eine Karte/ Es gibt Minuspunkte und die Runde ist beendet)* ➔ *VL:* Genau.
- *Kind zögert* ➔ *VL erklärt nochmals die Regel, stellt die Frage erneut und antwortet:* Richtig
- Wie viele Karten darfst du jede Runde umdrehen? - (*so viele wie ich will*)
- Wann kannst du immer aufhören, Karten umzudrehen? (wann es will)
- Hast du noch Fragen?
- Dann beginnen wir jetzt das Spiel.
- Denk daran, dass du die Runde jederzeit beenden kannst und deine gesammelten Punkte sichern kannst.
- *VL legt die Karten entsprechenden der jeweiligen Angaben auf der Infotafel aus. Kind deckt die Karten auf, VL teilt ihm am Ende jeder Runde das Ergebnis mit und trägt es auf dem Protokollbogen ein*
- ***Zu Beginn jeder neuen Infotafel: Ich drehe die Infotafel jetzt um. Das heißt die Gewinnkarten zählen jetzt X Punkte, es gibt Y Verlustkarte, und die Z Punkte zählen Denk daran, dass du jeder Zeit aufhören und deine Punkte sichern kannst.***

4. CPT (Tier oder Fahrzeug)

Material

Computer

Response Box

Starten des Experimentes

- Starte directRT
- Klicke auf File – Select and run input file
- Wähle unter Eigene Dateien den Ordner GIDeCA aus – Session I – CPT_GIDeCA
- wähle die Datei CPT_GIDeCA.csv
- Trage die Versuchspersonennummer ein und starte das Experiment

Instruktion

- Bei dieser Aufgabe siehst du auf dem Bildschirm immer zuerst ein kleines Kreuz. Das zeigt dir an, wo du hinschauen sollst.
- Danach werden dir einzelne Bilder von Tieren oder Fahrzeugen gezeigt.
- Die Tiere sehen so aus.
- Die Fahrzeuge sehen so aus.
- Sobald ein Bild auf dem Bildschirm erscheint, musst du mit den farbigen Tasten entscheiden, ob es sich um ein Tier oder um ein Fahrzeug handelt.
- Ist auf dem Bild ein Tier, drückst du die blaue Taste. – Ist auf dem Bild ein Fahrzeug, drückst du die rote Taste
- Hier noch einmal zwei Beispiele. Welche Taste würdest du hier drücken?
- Du machst gleich einen Übungsdurchgang.
- Wenn du richtig gedrückt hast, wird der Bildschirm grün.
- Wenn du falsch gedrückt hast, wird der Bildschirm rot.
- Und wenn du zu langsam warst, wird der Bildschirm gelb.
- Leg nun deine Finger auf die beiden Tasten. Bitte lass sie während der gesamten Aufgabe darauf liegen, damit du besser drücken kannst
- Bist du bereit?
- *Nach dem Übungsdurchgang.* Das war der Übungsdurchgang. Hast du noch Fragen?
- Gleich geht's richtig los!

- Du bekommst keine Rückmeldung mehr, ob du richtig, falsch oder zu langsam gedrückt hast.
- **Versuche so schnell wie möglich zu drücken und so wenige Fehler wie möglich zu machen!**
- Bitte, lass deine Finger während der gesamten Aufgabe auf den beiden Tasten liegen.
- Bist du bereit?

5. Würfelspiel klassischer Stil („Nur keine 6“)

Material

Würfelbecher mit Würfel

Ersatzwürfel

Ergebnisbogen

Instruktion

- Das nächste Spiel ist ein Würfelspiel. Dabei kannst du wieder Punkte sammeln.
- Du erhältst einen Würfel und darfst damit so oft würfeln, wie du willst. Die Zahlen werden immer zusammengezählt. Du sollst dabei versuchen, so viele Punkte wie möglich zu sammeln.
- Würfelst du im ersten Wurf eine 2 und im zweiten Wurf eine 3 hättest du also insgesamt schon 5 Punkte und dürftest noch weiterwürfeln.
- Ein Hindernis gibt es aber doch: Wenn du eine 6 würfelst, dann werden alle Punkte gestrichen und du landest wieder bei 0 Punkten.
- Es geht also darum, viele Punkte zu sammeln und möglichst keine 6 zu würfeln. Du selbst entscheidest, wie oft du würfeln möchtest. Wenn du die Runde beenden möchtest, dann sagst du „Stopp“ und ich schreibe die Punkte auf.“
- Weißt du noch, bei welchen Zahlen die Punkte zusammengezählt werden? *Antwort des Kindes abwarten, ggf. noch mal wiederholen, dass die Zahlen von 1 bis 5 zusammengezählt werden.*
- Und bei welcher Zahl werden alle Punkte gestrichen? *Antwort des Kindes abwarten, ggf. noch mal wiederholen, dass bei einer 6 alle bereits erspielten Punkte gestrichen werden.*
- Was sagst du, wenn du aufhören willst zu würfeln? Was passiert dann?

- Zunächst wollen wir einen Probedurchgang spielen. Hier hast du den Würfel und den Würfelbecher. Du darfst sofort beginnen. *VL übergibt den Würfel an das Kind und teilt ihm jeweils das Ergebnis des Probedurchgangs mit.*
- *Ist gleich die erste Zahl eine 6:* Dieser Durchgang zählt jetzt im Probelauf noch nicht, im richtigen Spiel würdest du allerdings für diese Runde keine Punkte erhalten.
- Insgesamt spielen wir 10 Durchgänge, in denen du möglichst viele Punkte sammeln

sollst.

- Hast du noch Fragen?
- Dann geht's jetzt los mit der ersten Runde! *(VL übergibt den Würfel an das Kind und lässt das Kind würfeln, bis es die Runde beenden möchte bzw. bis eine 6 fällt)*."
- *VL nennt dem Kind nach jedem Wurf, die Gesamtzahl der bisher erspielten Punkte. Bsp: Kind würfelt 3, dann 5 - VL sagt: „3 Punkte +5 Punkte sind 8 Punkte", Kind würfelt 2 - VL sagt: „8 Punkte +2 Punkte sind 10 Punkte" usw. Möchte das Kind nicht mehr würfeln sagt der VL: „In dieser Runde hast du xy Punkte gewonnen."*
- *die gewürfelten Augen und das Ergebnis der Runde in der beigefügten Tabelle notieren*

Rückmeldungen:

- *Kind verliert mehrmals nacheinander und ist kurz vor dem Aufgeben oder hat keine Lust mehr. VL:* „Du hast noch xy Durchgänge vor dir. Sicher hast du das nächste Mal mehr Glück!

6. Flanker

Material

Computer

Response Box

Starten des Experimentes

- Starte directRT
- Klicke auf File – Select and run input file
- Wähle unter Eigene Dateien den Ordner GIDeCA aus – Session I – Flanker_GIDeCA
- wähle die Datei Flanker_GIDeCA.csv
- Trage die Versuchspersonennummer ein und starte das Experiment

Instruktion

- Bei dieser Aufgabe siehst du auf dem Bildschirm immer zuerst ein kleines Kreuz. Das zeigt dir an, wo du hinschauen sollst.
- Danach werden dir einzelne Reihen mit jeweils 5 Pfeilen angezeigt. Die Pfeile sehen so aus – *auf dem Bildschirm zeigen*
- Sobald die Pfeile auf dem Bildschirm erscheinen, musst du mit den farbigen Tasten anzeigen, in welche Richtung der jeweils mittlere Pfeil zeigt.
- Zeigt der Mittlere Pfeil nach links, drückst du die blaue Taste.
- Zeigt der mittlere Pfeil nach rechts, drückst du die rote Taste.
- Du siehst nun zwei Beispielaufgaben. Welche Taste würdest du hier drücken?
- *Wenn richtig* --> Super!
- *Wenn falsch* --> Das war leider nicht ganz richtig, achte auf den mittleren Pfeil.
- Du machst gleich einen Übungsdurchgang.
- Wenn du richtig gedrückt hast, wird der Bildschirm grün.
- Wenn du falsch gedrückt hast, wird der Bildschirm rot.
- Und wenn du zu langsam gedrückt hast, wird der Bildschirm gelb.
- Leg nun deine Finger auf die beiden Tasten. Bitte lass sie während der gesamten Aufgabe darauf liegen, damit du besser drücken kannst
- Bist du bereit?
- *Nach dem Übungsdurchgang.* Das war der Übungsdurchgang. Hast du noch Fragen?

- Gleich geht's richtig los!
- Du bekommst keine Rückmeldung mehr, ob du richtig, falsch oder zu langsam gedrückt hast.
- **Versuche so schnell wie möglich zu drücken und so wenige Fehler wie möglich zu machen!**
- Bitte lass deine Finger während der gesamten Aufgabe auf den beiden Tasten liegen.
- Bist du bereit?

7. Speichelprobe

Material

Speichelset

Handschuhe

BIS/BAS bereit halten

Glas/Tasse und Wasserflasche

Instruktion

- ***Speichelröhrchen beschriften mit Family-ID (XXX-001)***
- ***Handschuhe anziehen***
- Wir machen jetzt eine Speichelprobe. Dafür musst du gleich in dieses Röhrchen spucken. *–Röhrchen zeigen.* – Damit du auch genug Speichel in das Röhrchen spucken kannst, ist es wichtig, dass deine Wangen möglichst entspannt sind. Du kannst dafür deine Wangen so ganz sanft reiben. *Vormachen: Wangen sanft reiben.– ungefähr 30 Sekunden.*
- Du musst soviel Spucke in den Trichter rein spucken, bis die Füllhöhe erreicht ist.
- Versuch so wenige Luftbläschen wie möglich im Speichel zu haben.
- Und bitte fass nicht oben auf das Röhrchen, weil es dann passieren kann, dass deine Probe unbrauchbar wird.
- *Wenn noch nicht genug Speichel im Röhrchen – wiederholen. Bei Schwierigkeiten bzw. zu vielen Luftbläschen BIS/BAS Fragebogen zwischendurch machen, wenn gar nichts geht: Kind etwas Wasser trinken lassen* – Ich glaube, du musst erst einmal wieder etwas Spucke sammeln. Daher machen wir jetzt erstmal etwas anderes und danach machen wir hier mit dem Speichel weiter.

Für den Versuchsleiter

Kein Kontakt mit den bloßen Händen zum Röhrchenende

1. Röhrchen nach Abgabe senkrecht in der Hand halten und den Deckel drücken bis er hörbar einrastet.
2. Halten Sie das Röhrchen. Schrauben Sie das Röhrchen vom Trichter ab.
3. Die kleine Kappe für das Röhrchen in die Hand nehmen und fest damit verschließen.
4. Schütteln Sie das verschlossene Röhrchen 5 Sekunden lang. Entsorgen oder recyceln Sie den Trichter.

8. BIS/BAS

Material

Fragebogen

Instruktion

- Jetzt machen wir kurz was Anderes!
- Wir haben hier einen Fragebogen, den wir jetzt gemeinsam ausfüllen werden.
- Der Fragebogen enthält eine Reihe von Feststellungen, mit denen man sich selbst beschreiben kann. Diese Feststellungen können genau auf dich zutreffen, eher zutreffen, eher nicht oder gar nicht auf dich zutreffen. – *auf Antwortfolie zeigen*
- Bitte beantworte jede Feststellung, auch wenn du einmal nicht sicher bist, welche Antwort für dich zutrifft.
- Ich werde dir die Fragen jetzt vorlesen und du zeigst mir hier auf dem Zettel deine Antwort an.

- **Gegebenenfalls Speichelprobe fortsetzen**

9. Eltern-Kind-Interaktion

Material

Nikitin Geowürfel

Laminierte Spielkarten

Stoppuhr

Ergebnisbogen

Instruktion

- Als nächstes wollen wir ein Spiel spielen – dieses Mal aber nicht alleine, sondern mit deiner Mutter/ deinem Vater zusammen.
- Dafür werde ich jetzt einmal deine Mutter/deinen Vater rüberholen
- *Zum Kind:* Du sollst gleich mit diesen Steinen verschiedene Figuren nachbauen. Bevor wir damit aber anfangen, muss ich wissen, ob du dieses Spiel schon kennst.
- *Zum Kind:* Kennst du dieses Spiel? *Wenn ja* – wie oft spielst du dieses Spiel?
- *Zur Mutter/Vater:* Und Sie? Kennen Sie dieses Spiel? *Wenn ja* – Wie oft spielen Sie dieses Spiel? *Auf Ergebnisbogen eintragen.*
- Dann erkläre ich jetzt die Regeln.
- *Zum Kind:* Du baust ganz schnell verschiedene Figuren nach den Kartenvorlagen – *Beispielkarten zeigen. Beispiele: ein buntes, ein graues.*
- Wenn die Bausteine auf der Karte farbig sind, musst du auch diese Steine benutzen. Wenn die Bausteine auf der Karte grau sind, dann ist die Farbe der Steine egal und du kannst die Steine verwenden, die du möchtest.
- Die Figuren sollen genau so aussehen, wie auf der Karte abgebildet.
- Achte bitte darauf! Es ist wichtig, sonst kann die Figur nicht gewertet werden. .
- Insgesamt hast du 7 Minuten Zeit. Wenn die Zeit um ist, rufe ich „STOPP“ und dann musst du sofort aufhören.
- Versuch so viele Figuren nachzubauen wie möglich.
- Wenn dir eine Figur zu schwer ist, dann kannst du die Karte zur Seite legen und eine andere Figur bearbeiten.
- Für jede Figur, die du richtig gelöst hast, bekommst du 5 Punkte.
- Bitte leg alle Karten, die du bearbeitet hast auf einzelne Stapel ab. Ein Stapel mit allen gelösten Karten – und ein Stapel mit allen Karten, die du sonst noch versucht hast.
- *Zur Mutter/Vater:* Sie dürfen Ihrem Kind helfen, indem Sie Tipps geben oder es anfeuern oder Ähnliches. Sie dürfen aber nicht selber mitbauen.

- Gibt es noch Fragen?
- Gut: Dann fangen wir an. Auf die Plätze – fertig – los!
- *Aus dem Raum gehen– nach 7 Minuten wieder hereinkommen:* Und Stopp.
- Das wäre geschafft. Dann wollen wir einmal zählen, wie viele Figuren du geschafft hast.
- Das wären dann XY Punkte. Super!

Diesen Teil der Instruktion nicht vorlesen!!! ***Sollte möglichst echt/realistisch vorgebracht werden…***

- Super! Du hast alle Aufgaben für heute geschafft.
- Ich werde jetzt gleich noch einmal in den Raum nebenan gehen, um deine genauen Punkte auszurechnen. Außerdem bekommst du von mir noch eine Urkunde.
- Leider kann das manchmal etwas länger dauern. Daher hab ich dir und deiner Mutter/ deinem Vater ein Spiel für die Zwischenzeit mitgebracht: Den Jenga-Turm *aufbauen*

10. Jenga-Turm

Material

Jenga-Turm aufgebaut

Außerdem im anderen Raum bereithalten:

- *Urkunde*
- *Trinkflasche*

Intruktion

- Kennt ihr den Jenga-Turm?
- Beim Jenga-Turm müssen du und deine Mutter/dein Vater immer abwechselnd einen Holzklotz ganz vorsichtig aus dem Turm ziehen und oben wieder drauflegen.
- Auf diese Weise wird der Turm immer höher – aber auch immer wackeliger.
- Ihr könnt jeden Stein aus dem Turm ziehen, den ihr wollt – außer aus den obersten drei Reihen. Außerdem dürft ihr zum Rausziehen nur eine Hand benutzen.
- Ok? Ich versuch auch mich zu beeilen. *Den Raum verlassen, die Punkte ausrechnen, Namen in Urkunde eintragen und nach genau 7 Minuten wieder in den Raum kommen.*
- ***Wenn Eltern oder Kind darauf hinweisen, dass die Kamera noch an ist, dann die Kamera ausschalten. Wenn die nicht spielen wollen, dann ist das ok, Turm einfach zu denen auf den Tisch stellen und den Raum verlassen.***

Beim Wiederkommen:

- Ok – du hast also insgesamt XYZ Punkte bisher eingespielt. Das war echt super. Beim nächsten Mal kannst du noch weitere Punkte dazugewinnen.
- Außerdem habe ich hier schon einmal eine Urkunde und eine Trinkflasche für dich.

Actigraphen auslesen und Daten speichern

- Actigraphen anschließen und ActiLife öffnen
- Download
- VP-Nummer und Session Beginn auswählen
- Auf 1 Sekunde aggregieren

B.1.2. Instructions Children Session II

GIDeCA Studie

Gen-Umwelt-Interaktionen auf Entscheidungsfindung bei Kindern mit und ohne ADHS

Instruktionen für Testleiter

Session II

Stand: 01.03.2013

Studienleitung:

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Begrüßung

Guten Tag Frau/Herr XY, hallo XY. Schön, dass Sie und dass du da sind. Heute findet der zweite Teil unserer Studie statt. (*Falls beim ersten Termin andere Testleiter:* Mein Name ist XYZ und ich und ein/e Kollege/in werden diese Studie mit Ihnen durchführen.) Dann gehen wir jetzt wieder in unsere Testräume.

Im Testraum:

Vielen Dank, dass du heute wieder gekommen bist. Wir werden heute wieder verschiedene Aufgaben und Spiele machen. Einige davon werden wie auch beim letzten Mal am Computer stattfinden, andere am Tisch. Ich möchte dich wieder bitten, dass du dich bei allen Aufgaben so gut wie möglich anstrengst. Wenn du Fragen hast oder eine Anleitung nicht gleich verstehst, kannst du mich jederzeit fragen.
Wie schon beim letzten Mal möchten wir gerne schauen, wie deine Bewegungen während der Aufgaben sind. Dafür bringen wir wieder den ActiGraph an deiner Hose an.

Einstellung Actigraph

7. Actigraph an den Laptop anschließen
8. Starten von ActiLife5 (auf dem Desktop)
9. Reiter „*Devices*“ aktivieren und auf „**Initialize**“ klicken
10. Sample Rate auf 100 Hz stellen; Startzeit und Stoppzeit (Startzeit + 3h) angeben
11. *Enter Subject Info*: VP Nummer, Geschlecht, **Länge & Gewicht**, Geburtsdatum, **Limb: Waist**, Nicht-Dominanz einstellen
12. **Actigraph an der Nicht-Dominaten Seite befestigen oder von Eltern anlegen lassen**

Spielezettel

Und auch heute bekommst du wieder einen Spielezettel, auf dem du Stempel für die Aufgaben und Spiele sammeln kannst. Spielezettel ausgeben. *Spielezettel ausgeben.*

1. 2CR + DRT

Two Choice Delay Task (2CR)

Material

Computer (Im Unterordner GIDeCA auf dem Desktop)

Tastatur

Starten des Experimentes

- Öffne den Ordner GIDeCA
- Unterordner „Session 2“, Unterordner „1. 2CR“ öffnen und Experiment „2CR-MR green mouse.ebs2 starten
- Trage die VP-Nummer ein

Instruktion

Bei dieser Aufgabe wird dir auf dem Bildschirm immer ein grüner Pfeil angezeigt. Dieser Pfeil zeigt dabei immer entweder nach links oder nach rechts.

Wenn der Pfeil nach links zeigt, dann musst du die linke, blaue Taste *(auf der Computertastatur zeigen)* drücken. Wenn der Pfeil nach rechts zeigt, dann musst du die rechte, rote Taste drücken.

Bitte versuch so schnell wie möglich zu drücken und so wenig Fehler wie möglich zu machen.

Als erstes wirst du ein paar Probedurchgänge machen. *Probedurchgänge starten.*

Nach Beendigung der Probedurchgänge: Das waren die Probedurchgänge. Hast du noch Fragen?

Wann musst du welche Taste drücken?

Während des Spiels darf ich nicht mit dir sprechen. Ich werde hinter die Trennwand gehen. Wenn du fertig bist, kannst du mich zurückrufen.

Los geht’s!

Delay Reaction Time Task (DRT)

Material

Computer (Im Ordner GIDeCA auf dem Desktop)

Tastatur

Starten des Experimentes

- Öffne den Ordner GIDeCA
- Unterordner „Session 2“; Unterordner „DRT“ öffnen und Experiment „delayedRT-MR mouse.ebs2“ starten
- Trage die VP-Nummer ein
- Nächste Instruktionsfolie immer mit beliebiger Tastaturtaste anzeigen

Instruktion

- Diese Aufgabe ist so ähnlich wie die Aufgabe, die du gerade gemacht hast.
- Das heißt, auch in dieser Aufgabe wird dir auf dem Bildschirm immer ein grüner Pfeil angezeigt, der entweder nach links oder nach rechts zeigt.
- Wie eben drückst du die linke, blaue Taste *(auf der Computertastatur zeigen)*, wenn der Pfeil nach links zeigt und die rechte, rote Taste, wenn der Pfeil nach rechts zeigt.
- Aber: Dieses Mal sollst du immer erst dann drücken, wenn der Pfeil verschwunden ist.
- Jeder Durchgang beginnt dabei mit einem Ton. Anschließend wird dir der Pfeil angezeigt. Sobald dieser Pfeil verschwindet, drückst du die entsprechende Taste. Danach wird dir der Pfeil zur Kontrolle noch einmal angezeigt. Dann musst du aber nicht drücken. Anschließend hörst du einen Ton und der nächste Durchgang beginnt.
- Lass uns zuerst einige Probedurchgänge spielen – *Probedurchgänge starten*
- Hast du noch Fragen? Was ist anders als bei der Aufgabe von eben?
- Ok, dann beginnen wir jetzt mit dem eigentlichen Spiel
- Während des Spiels darf ich nicht mit dir sprechen. Ich werde wieder hinter die Trennwand gehen und wenn du fertig bist, rufst du mich. Los geht’s. *Spiel starten*

2. Zahlenspanne

Material

Protokollbogen

Instruktion

Zahlenspanne vorwärts

- Ich spreche dir jetzt ein paar Zahlen vor. Höre aufmerksam zu.
- Wenn ich fertig bin, sollst du die Zahlen wiederholen. Sag einfach, was ich gesagt habe.
- *Immer beide Versuche vorlesen – Kind kann entsprechend 0,1 oder 2 Punkte pro Länge der Zahlenspannen erreichen.*
- ***Die Versuche so lange durchführen, bis Abbruchkriterium – beide Versuche einer Aufgabe nicht gelöst – erreicht.***

Zahlenspanne rückwärts

- Jetzt spreche ich dir noch weitere Zahlen vor. Doch dieses Mal sollst du die Zahlen rückwärts wiederholen.
- Wenn ich sage 8 – 2, was musst du dann sagen?
- *Wenn richtig:* Das ist richtig. *Zum nächsten Beispiel übergehen*
- *Wenn falsch:* Das ist nicht ganz richtig. Ich habe gesagt: 8 – 2. Wenn du die Zahlen rückwärts wiederholst, musst du sagen 2 – 8. Versuchen wir es noch einmal: 8 – 2
- Versuchen wir es mit folgenden Zahlen. Denk daran: Du sollst sie rückwärts wiederholen: 5 – 6
- *Wenn richtig:* Das ist richtig. *Zur Aufgabe übergehen*
- *Wenn falsch:* Das ist nicht ganz richtig. Ich habe gesagt: 6 – 5. Wenn du die Zahlen rückwärts wiederholst, musst du sagen 5 – 6.
- *Wenn richtig:* Das ist richtig.
- *Wenn falsch:* Das ist nicht ganz richtig. Ich habe gesagt: 5 – 6. Wenn du die Zahlen rückwärts wiederholst, musst du sagen 6 – 5. Versuchen wir es noch einmal: 5 – 6
- *Die Versuche so lange durchführen, bis Abbruchkriterium* ***– beide Versuche einer Aufgabe nicht gelöst –*** *erreicht.*

3. Tapping

Material

Computer

Response Box

Starten des Experimentes

- Starte directRT – File – Select and run input file
- Öffne den Ordner Eigene Dateien – GIDeCA – Session II – Tapping
- Wähle tapping.csv
- Trage die VP-Nummer ein

Instruktion

5. Mit dieser Aufgabe wollen wir untersuchen, wie gut du in einem bestimmten Rhythmus tippen kannst
6. In dieser Aufgabe wirst du Töne hören, die immer im gleichen Abstand erklingen.
7. Deine Aufgabe ist es zusammen mit jedem Ton die rote Taste *(auf der Response Box zeigen)* zu drücken.
8. Irgendwann wirst du keine Töne mehr hören.
9. Deine Aufgabe ist es dann, die Taste immer weiter zu drücken, bis der Computer STOPP! sagt
10. Wir machen jetzt einen kurzen Übungsdurchgang. *Wenn es zu viele Fehler waren oder die Aufgabe nicht klar ist noch einen Übungsdurchgang*
11. Hast du noch Fragen?
12. Dann geht’s jetzt los!

4.Go-NoGo

Material

Computer

Response Box

Starten des Experimentes

- Starte DirectRT
- Wähle: “File” – “select and run input file”
- Klicke dich durch die Ordnerstruktur (GIDeCA – Session II – GoNoGo)
- Und wähle GoNoGo_GIDeCA.csv

Instruktion

- Diese Aufgabe heißt Drücken – Nicht-Drücken
- Bei dieser Aufgabe siehst du auf dem Bildschirm immer zuerst ein kleines Kreuz. Das zeigt dir an, wo du hinschauen sollst.
- Danach werden dir immer Tierbilder gezeigt. Deine Aufgabe ist es, bei jedem Tierbild die rote Taste *(auf der Response Box zeigen)* zu drücken – außer du siehst eine Gans. Dann drückst du gar keine Taste.
- Hier siehst du die 4 Bilder bei denen du drücken sollst und unten siehst du die Gans, bei der du nicht drücken darfst.
- Du machst jetzt einen kleinen Übungsdurchgang.
- Lege nun deine Finger auf die rote Taste. Bitte lass sie während der gesamten Aufgabe drauf liegen damit du besser drücken kannst.
- Nach jedem Durchgang wird dir angezeigt, ob du richtig gedrückt hast.
- Bist du bereit?
- *Ende des Übungsdurchganges*
- Hast du noch Fragen?
- Gleich geht’s richtig los! Du bekommst keine Rückmeldung mehr darüber, ob du richtig falsch oder zu langsam gedrückt hast.
- **Versuche so schnell wie möglich zu drücken und so wenige Fehler wie möglich zu machen!**
- Bitte, lass deinen Finger während der gesamten Aufgabe auf der roten Taste liegen.
- Bist du bereit?

5. Game of Dice

Material

Würfelbecher
Würfel
Setzstein
Spielfolie
Taschenrechner
Laminierte Zettel (18 Stück)
abwischbaren Stift
Ergebnisbogen

Instruktion

- Als nächstes spielen wir ein Würfelspiel. Dabei bekommst du von uns 180 Punkte als Startguthaben. *Auf abwischbaren Zettel schreiben*
- In jeder Runde des Spiels kannst du entweder Punkte dazugewinnen oder aber Punkte verlieren.
- Ziel des Spiels ist es erneut so viele Punkte wie möglich zu sammeln.
- *auf die Würfeltafel zeigen* - In jeder Runde des Spiels kannst du auf eine oder mehrere Zahlen setzen. Danach würfelst du *(Würfel und Würfelbecher zeigen)*
- Du hast die Möglichkeit auf eine, zwei, drei oder vier Zahlen zu setzen, wobei du unterschiedlich viele Punkte gewinnen oder verlieren kannst – nämlich 10 Punkte, 5 Punkte, 2 Punkte oder einen Punkt.
- Zum Beispiel kannst du auf vier Zahlen gleichzeitig setzten, d.h. du glaubst, dass im nächsten Durchgang eine dieser vier Zahlen gewürfelt wird. Wenn du z.B. auf 1 bis 4 setzt, dann gewinnst du einen Punkt, wenn du eine 1, eine 2, eine, 3 oder eine 4 würfelst.
- Wenn du aber eine andere Zahl würfelst, z.B. eine 5 oder eine 6, dann verlierst du einen Punkt.
- Du könntest auch auf nur zwei Zahlen setzen, z.B. auf die Zahlen 3 und 4. In diesem Fall gewinnst du fünf Punkte, wenn du entweder eine 3 oder eine 4 würfelst. Wenn aber eine andere Zahl fällt, dann verlierst du fünf Punkte.
- Natürlich kannst du auch auf drei Zahlen gleichzeitig setzen oder bloß auf eine einzige Zahl.

- Wir machen einfach einmal einen Probedurchgang.
- Worauf möchtest du setzen? Setze deinen Setzstein darauf.
- Wie viele Punkte würdest du dabei gewinnen bzw. verlieren? *Gegebenenfalls noch einmal erklären*
- Welche Zahl musst du würfeln, um zu gewinnen? Bei welchen Zahlen würdest du verlieren?
- Dann darfst du jetzt würfeln.
- *Ergebnis des Probedurchganges mitteilen*: Eine *x*. Das bedeutet: Du hättest jetzt *y* Punkte gewonnen/verloren
- Hast du noch Fragen?
- Dann beginnen wir jetzt mit dem richtigen Spiel. Insgesamt spielen wir 18 Runden.
- Worauf möchtest du setzen? – Dann darfst du jetzt würfeln – Du hattest auf x gesetzt, jetzt hast du y gewürfelt, d.h. du gewinnst/verlierst z Punkte.

Rückmeldungen ?!:

- Kind verliert mehrmals nacheinander und ist kurz vor dem Aufgeben oder hat keine Lust mehr ➔ VL: „Manchmal hat man ein wenig Pech (???), aber du hast noch xy Durchgänge vor Dir. Bestimmt klappt es beim nächsten Mal schon besser!“
  Kind glaubt die Würfel sind gezinkt ➔ VL: „Ich habe hier noch weitere Würfel, du darfst gern einen anderen ausprobieren.“ Kind darf sich einen neuen Würfel aussuchen und mit diesem weiterspielen

6. Delay Frustration Task (DeFT)

Material

Computer

Response Box

Erklärungsfolie

Starten des Experimentes

- Öffne aus dem GIDeCA-Ordner den Unterordner „DeFT“ und starte die DeFT.exe-Datei.
- Trage die VT-Nummer ein
- Ist der Haken bei „use keyboard“ gesetzt?
- Klicke auf „load configuration file“
- Wähle aus dem Ordner GIDeCA\DeFT den File “child.txt“ aus
- Trage Alter und Geschlecht des Kindes ein und drücke ok

Instruktion

Die gleichen Instruktionen stehen auch auf dem Bildschirm, sodass das Kind diese mitlesen kann. Allerdings konnte das Beispiel nicht übersetzt werden und ist noch dazu falsch!!!. Daher dem Kind sagen, dass es bitte auf unsere Beispielfolie gucken soll.

- Bei dieser Aufgabe wirst du auf dem Bildschirm einfache Matheaufgaben sehen – so wie diese – *auf der Folie zeigen*
- Unterhalb der Aufgaben werden dir vier Lösungsmöglichkeiten angezeigt: A, B, C und D. Aber nur eine der Lösungsmöglichkeiten ist auch richtig.
- Deine Aufgabe ist es, zu entscheiden, welche der vier Lösungen richtig ist, und die entsprechende Taste zu drücken.- *auf der Response Box zeigen.*
- Wenn du meinst, dass die Lösung A richtig ist, dann Taste A drücken. Wenn du meinst, dass Lösung B richtig ist, dann Taste B usw.
- Sobald du dich entschieden und die Taste gedrückt hast, zeigt dir der Computer normalerweise die nächste Aufgabe an.
- Da das Programm aber sehr rechenaufwendig ist, kann es sein, dass der Computer manchmal etwas „hängt“ und du die Taste mehrmals drücken musst.
- Wenn es dir hilft, kannst du zum Rechnen deine Finger benutzen.

- Hast du noch Fragen?
- Ich werde jetzt das Spiel starten und möchte, dass du dein Bestes gibt und so schnell wie möglich antwortest.
- Während des Spiels darf ich nicht mit dir sprechen.
- Los geht's!
- ***Spiel starten, indem auf den „Ok"-Button geklickt wird. Achtung: Dieser ist halb unter der Abbildung (oberhalb) versteckt!!***

7. Duration Discrimination

Material

Computer

Response Box

Starten des Experimentes

- Starte directRT
- "File" – "Select and run input file"
- Klicke dich durch die Ordnerstruktur: Eigne Dateien – GIDeCA – Session II – Duration Discrimination
- Wähle Duration Discrimination.csv

Instruktion

- Mit dieser Aufgabe wollen wir untersuchen, wie gut du zwischen zwei ähnlich langen Zeitabständen unterscheiden kannst.
- In dieser Aufgabe werden dir immer zwei Zeitabstände dargeboten.
- Jeder Zeitabstand beginnt und endet mit einem Ton.
- Deine Aufgabe ist es, zu sagen, welcher Zeitabstand länger ist.
- Wenn der erste Zeitabstand länger ist, dann drückst du die blaue Taste *(auf der Response Box zeigen).*
- Wenn der zweite Zeitabstand länger ist drückst du die rote Taste *(auf der Response Box zeigen).*
- Hast du noch Fragen?
- Wir beginnen mit vier Probedurchgängen.
- *Probedurchgang starten*
- *Nach dem Probedurchgang* - Hast du noch Fragen?
- Dann starte ich jetzt die Aufgabe

8. ZKE-Fragebogen

Material

Fragebogen

Antworttafel für das Kind

Instruktion

- Jetzt machen wir mal etwas Anderes!
- Wir haben hier einen Fragebogen, den wir jetzt gemeinsam ausfüllen werden
- Ich werde dir ein paar Sätze vorlesen, in denen beschrieben wird, wie sich Eltern manchmal verhalten.
- Hör dir bitte jeden Satz in Ruhe an und zeig mir dann auf dieser Antworttafel - *Antworttafel zeigen* –, ob der Satz für deine Mutter stimmt und ob der Satz für deinen Vater stimmt.
- Du kannst jeweils wählen zwischen „Stimmt nicht“ (0) – „stimmt wenig“ (1) – „stimmt ziemlich“ (2) – oder „stimmt völlig“ (3) – *auf Antworttafel dem Kind zeigen.*
- Wohnst du mit deiner Mutter oder deinem Vater allein zuhause?
- *Wenn ja:* In diesem Fall beantwortest du die Fragen nur für XY.
- Hast du noch Fragen?
- Ok, dann legen wir los.

9. Delay of Gratification

Material

Schokoriegel

- Bounty
- Lion
- Mars
- Snickers

Teller

Fragebögen (2 Versionen)

Glocke

Instruktion

- Ich habe hier verschiedene Schokoriegel. *Vor das Kind die einzelnen Riegel hinlegen* – Welchen von diesen Schokoriegeln magst du denn am liebsten?
- *Den Riegel den das gibt angibt auf dem Teller liegen lassen, alle anderen wieder einsammeln.*
- Das ist der Riegel, den du am liebsten magst. Bitte pack diesen Riegel aus und leg ihn vor dir auf den Teller – aber noch nicht essen.
- Ich werde gleich wieder hinter die Trennwand gehen. Du kannst dich entscheiden, ob du den Schokoriegel sofort essen möchtest oder ob du wartest, bis ich zurückkomme. Dann würdest du zwei Schokoriegel bekommen. *Den zweiten Schokoriegel auf den Tisch hinter den Teller legen (aus Sicht des Kindes).*
- Du kannst zu jeder Zeit den Schokoriegel essen. Dann ist das Spiel aber vorbei, und du bekommst keinen zweiten Schokoriegel. Wenn du nicht länger warten möchtest und dich entscheidest den Schokoriegel zu essen, drückst du auf die Glocke. Dann werde ich sofort zu dir zurückkommen.
- Nochmals zur Erinnerung:
    - Du hast die Wahl: einen Schokoriegel sofort zu bekommen, oder zu warten bis ich wieder da bin und dann zwei Schokoriegel zu bekommen
    - Du kannst jederzeit das Spiel beenden und den Schokoriegel essen.
    - Wenn du den Riegel essen möchtest, dann drückst du auf die Glocke und ich komme zurück.
- Hast du noch Fragen?

- Hast du eine Uhr oder ein Handy? *Wenn ja* – Würdest du mir die bitte geben, solange wir das Spiel spielen? Es ist nämlich ganz wichtig, dass du auf keinen Fall auf deine Uhr guckst.
- Dann geht es jetzt los!

*Nach **25!** Minuten wieder kommen (außer natürlich das Kind, klingelt)*

- Nun habe ich noch ein paar Fragen an dich. *FB-Fragen an Kind stellen und FB ausfüllen.*

Abschluss

Material

Urkunde, Bleistift

- Super! Du hast alle Aufgaben geschafft.
- Dann hole ich mal deine Mutter/deinen Vater rüber.
- Hier bekommst du noch deine Urkunde für den heutigen Termin.
- Vielen Dank nochmal für deine Teilnahme…

B.1.3. Instructions Parents Session I

GIDeCA

Gen-Umwelt-Interaktionen auf Entscheidungsfindung bei Kindern mit und ohne ADHS

Instruktionen für Testleiter

Session I - Elternversion

Stand: 01.03.2013

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Begrüßung

Guten Tag Frau/Herr XY, hallo XY. Schön, dass Sie und dass du da sind. Mein Name ist XYZ und ich und ein/e Kollege/in werden diese Studie mit Ihnen durchführen. Dafür müssen wir aber erst einmal in unsere Testräume gehen.

Im Testraum:

Bevor wir mit der eigentlichen Studie beginnen, möchte ich Sie bitten sich dieses Informationsblatt durchzulesen – es ist das gleiche, das Ihnen auch bereits zugeschickt worden ist. Außerdem habe ich eine Einverständniserklärung hier, die Sie bitte ausfüllen mögen, sofern Sie einverstanden sind, mit dem was wir in dieser Studie machen wollen.

Vielen Dank, dass Sie an unserer Studie teilnehmen. Der Termin heute wird sich in vier Teile untergliedern. Im ersten Teil werden wir mit Ihnen ein Interview durchführen, in dem wir Ihnen mehrere Fragen zu Ihrem Kind stellen werden. Im zweiten Teil werden Sie dann gebeten verschiedene Fragebögen zu Verhaltensweisen Ihres Kindes auszufüllen, bevor es im dritten Teil dann um Sie selbst geht und Sie Fragebögen zu Ihrer eigenen Person beantworten sollen. Der abschließende vierte Teil besteht aus einer Aufgabe, in der Sie gemeinsam mit Ihrem Kind Aufgaben lösen sollen.

Wie auch bei allen Aufgaben, die Ihr Kind bearbeiten wird, gilt: Ihre Teilnahme ist absolut freiwillig und alle Daten werden anonymisiert und nur zu wissenschaftlichen Zwecken im Rahmen dieser Untersuchung verwendet. Es ist nicht möglich persönliche Rückschlüsse zu ziehen.

Haben Sie noch generelle Fragen? Ansonsten beginnen wir mit dem ersten Teil.

1. Expressed Emotion

Material:

Diktiergerät

Stoppuhr

Instruktion:

Bevor wir mit dem eigentlichen Interview beginnen, würde ich gerne von Ihnen Ihre Gedanken und Gefühle bezüglich Ihres Sohnes XY/Ihrer Tochter YZ hören – in Ihren eigenen Worten. Ich möchte Sie bitten fünf Minuten lang zu erzählen, was für eine Person XY ist und wie Sie miteinander auskommen. Ich werde Sie dabei weder unterbrechen noch irgendwelche Fragen stellen oder Kommentare abgeben. Nachdem Sie begonnen haben zu erzählen, werde ich also keine Fragen beantworten, bis die 5 Minuten um sind. Ich werde Sie dabei auch nicht ansehen. Bitte lassen Sie sich hiervon nicht irritieren. Bevor wir beginnen: Haben Sie irgendwelche Fragen dazu?
Während des Sprachsamples keine Nachfragen stellen; die Person nicht angucken, keine Kommentare/Geräusche (z.B. hmm) machen!!!

- **Antworten auf Fragen *vor* dem Sprachsample**
  - Was genau soll ich denn erzählen/ Wollen Sie, dass ich beginne als XY so und so alt war?
    - Bitte erzählen Sie, was Sie denken, was wichtig ist über XY und wie sie miteinander auskommen.
- **Umgang mit Fragen *während* des Sprachsamples**
  - Mach ich das so richtig?
    - *Mit Kopf nicken*
  - Wie viel Zeit habe ich noch?
    - Noch ein paar Minuten/ Noch eine Minute – *auf keinen Fall die exakte Zeit sagen*
  - Wollen Sie, dass ich weiter über diese Sache spreche?
    - Erzählen Sie bitte weiter – noch für ein paar Minuten.
- **Was wenn die Person vor den 5 Minuten aufhört zu reden**
  - *Wenn nötig:* Bitte erzählen Sie mir noch irgendetwas über XY für die restlichen paar Minuten.
  - *Wenn Person dennoch nicht weiterspricht: 5 Minuten ablaufen lassen*

2. Anamnesebogen und Kinder-DIPS

Material:

Anamnesebogen

Kinder-DIPS Bogen

Instruktion:

- Als nächstes werde ich mit Ihnen ein längeres Interview führen.
- Das Interview dient dazu, einen Überblick über mögliche Schwierigkeiten Ihres Kindes zu gewinnen. Deswegen werden verschiedene Bereiche angesprochen, in denen generell Probleme und Schwierigkeiten bei Kindern auftreten können. Möglicherweise wird nicht jeder oder vielleicht auch gar kein Bereich auf Ihr Kind zutreffen.
- Einige Fragen werden Ihnen vielleicht auch seltsam oder sogar unpassend für Ihr Kind vorkommen.
- Für eine sorgfältige Diagnostik ist es aber wichtig, dass alle Bereiche angesprochen und abgeklärt werden.
- Während des Interviews werde ich mich an einem Leitfaden orientieren und Ihre Antworten entsprechend festhalten. So kann ich sicherstellen, dass ich keine Frage vergesse.
- Aufgrund der vielen Informationen, die ich abfragen werde, und weil die Zeit begrenzt ist, kann es passieren, dass ich Sie in Ihren Antworten unterbreche und mit der nächsten Frage weiter mache. Ich bitte Sie, das zu entschuldigen.
- Bei einem solchen Interview gibt es keine richtigen oder falschen Antworten. Es interessiert uns vielmehr, wie ihr Kind bestimmte Dinge erlebt und wie es sich in bestimmten Situationen fühlt oder verhält.
- Haben Sie noch Fragen, die das Interview betreffen?

3. Fragebögen zum Kind: ADHS-Symptomatik

Material

Fragebögen zum Kind: ADHS-Screening

Instruktion

- Nach dem Interview möchte ich Sie bitten verschiedene Fragebögen auszufüllen, die sich spezifisch mit dem Verhalten und der Entwicklung Ihres Kindes beschäftigen und zusätzlich zu dem Interview eine wichtige Datenquelle darstellen.
- Alle Fragebögen sind bereits vielfach von anderen Wissenschaftlern eingesetzt worden, sodass wir die Fragen nicht verändern können.
- Wir bitten Sie aber, alle Fragen zu beantworten, auch wenn Ihnen einige Fragen nicht angemessen erscheinen oder auf Ihr Kind nur wenig zutreffen.
- Wenn Sie Probleme mit einzelnen Fragen haben, können Sie sich aber immer an mich wenden.
- Haben Sie noch Fragen bezüglich der Fragebögen?

4. Fragebögen zum Elternteil: Psychopathologie

Material:

Fragebögen WURS & SCL-90-R (Psychopathologie des Elternteils)

Instruktion

- In den folgenden Fragebögen geht es nun nicht mehr um Ihr Kind, sondern um Sie selber.
- Im ersten Fragebogen werden Sie gebeten, sich in Ihre Kindheit zurückzuversetzen und zu beschreiben, wie Sie im Alter von 8-10 Jahren waren.
- Im zweiten Fragebogen finden Sie eine Liste von Problemen und Beschwerden, die man manchmal hat und Ihre Aufgabe ist es zu entscheiden, wie stark sie **während der vergangenen sieben Tage bis heute** durch diese Beschwerden gestört oder bedrängt worden sind.
- Sollte es Unklarheiten bei einzelnen Fragen geben, können Sie mich wie bei den Fragebögen eben gerne Fragen.
- Haben Sie noch Fragen zu diesem Fragebogen?

5. Eltern-Kind-Interaktion

Instruktion:

- Die nächste Aufgabe werden Sie gemeinsam mit Ihrem Kind bearbeiten.
- Wir warten jetzt, bis die/der Kollegin/Kollege von nebenan uns Bescheid gibt, dass sie soweit sind. Dann gehen wir rüber in den anderen Raum, wo die/der Kollegin/Kollege dann erzählen wird, worin die genaue Aufgabe besteht.

B.1.4. Instructions Parents Session II

GIDeCA

Gen-Umwelt-Interaktionen auf Entscheidungsfindung bei Kindern mit und ohne ADHS

Instruktionen für Testleiter

Session II - Elternversion

Stand: 01.03.2013

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Begrüßung

Guten Tag Frau/Herr XY, hallo XY. Schön, dass Sie und dass du da sind. Heute findet der zweite Teil unserer Studie statt. (*Falls beim ersten Termin andere Testleiter:* Mein Name ist XYZ und ich und ein/e Kollege/in werden diese Studie mit Ihnen durchführen.) Dann gehen wir jetzt wieder in unsere Testräume.

Im Testraum:

Vielen Dank, dass Sie heute erneut gekommen sind. Der Termin heute wird sich in drei Teile untergliedern. Im ersten Teil werden wir mit Ihnen ein Interview durchführen, in dem wir Ihnen mehrere Fragen zu Ihrem demographischen Hintergrund stellen werden. Im zweiten Teil werden Sie dann gebeten verschiedene Fragebögen auszufüllen, die sich mit der Situation bei Ihnen zuhause, Erziehung und der Persönlichkeit Ihres Kindes beschäftigen. Beim abschließenden dritten Teil handelt es sich dann erneut um ein kurzes Interview, in dem Sie gebeten werden, ihr Verhalten in verschiedenen hypothetischen Situationen zu beschreiben.

Wie beim letzten Mal, so gilt auch dieses Mal für alle Aufgaben und Fragen, die Sie oder ihr Kind bearbeiten: Ihre Teilnahme ist absolut freiwillig und alle Daten werden anonymisiert und nur zu wissenschaftlichen Zwecken im Rahmen dieser Untersuchung verwendet. Es ist nicht möglich persönliche Rückschlüsse zu ziehen.
Haben Sie noch generelle Fragen? Ansonsten beginnen wir mit dem ersten Teil.

1. Fragebogen ISBI

Material:

ISBI Fragebogen

Laminierte Antworttafeln

Instruktion:

- Im Folgenden werde ich Ihnen verschiedene Fragen zu Ihrer Herkunft, der Sprache, die in Ihrer Familie gesprochen wird, aber auch zu sozialen Aspekten stellen.
- Alle Antworten sind hierbei freiwillig und es gibt keine richtigen und falschen Antworten.
- Wenn Sie bereit sind und keine Fragen haben, dann beginne ich jetzt das Interview.

2. Fragebögen Erziehungsverhalten/JTCI

Material:

Fragebogenpaket Session II Eltern

Instruktion

- Die folgenden Fragebögen untersuchen Erziehungsverhalten, Familienorganisation und das Temperament Ihres Kindes.
- Das Beantworten aller Fragen wird etwa 45 Minuten dauern.
- Bitte beantworten Sie möglichst alle Fragen, auch wenn Ihnen einzelne Fragen merkwürdig erscheinen.
- Es gibt dabei keine richtigen und falschen Antworten. Daher antworten Sie möglichst spontan, was Ihnen als erstes einfällt.

3. Problemlöseinterview

Material

Diktiergerät

Szenarien

Instruktion

- Im letzten Teil des heutigen Tages möchte ich gerne erfahren, wie Sie Probleme lösen.
- Dafür werde ich Ihnen verschiedene Situationen schildern. Jede dieser Situationen enthält ein Problem.
- Bitte stellen Sie sich vor, dass Sie selbst in dieser Situation sind.
- Nachdem ich Ihnen die einzelnen Situationen vorgelesen habe, werde ich Ihnen jeweils die folgenden drei Fragen stellen:
    1. Nennen Sie mir alle Arten, auf die man das Problem lösen KÖNNTE.
    2. Nennen Sie mir die Lösung, die Sie in dieser Situation wählen würden.
    3. Schildern Sie mir bitte genau, wie Sie die Lösung umsetzen würden.
- Es soll also zunächst darum gehen, dass Sie alle Lösungsmöglichkeiten, die Ihnen einfallen aufzählen, unabhängig davon, ob Sie auch so reagieren würden. Im zweiten Schritt sollen Sie die für Sie passende Lösung nennen und diese dann im dritten Schritt genau und detailliert erläutern.
- Ich werde Ihre Antworten mit diesem Diktiergerät aufnehmen, sodass ich sie mir hinterher für die Auswertung erneut anhören kann.
- Ich glaube, diese Aufgabe könnte Ihnen gefallen.
- Haben Sie noch Fragen zum Ablauf? Ansonsten beginne ich mit der ersten Situation.

Hinweise für den Interviewer

- wenn die Person abschweift: zurück zur Aufgabe führen und sagen, dass hinterher Zeit ist, sich darüber zu unterhalten
- Sicherstellen, dass jede der drei Fragen bei jedem Problem gestellt wird
- Wenn Person auf Frage eins mehrere Lösungen nennt und dann verstummt – nachfrage: „Noch etwas“, wenn Person das verneint, dann zur zweiten Frage übergehen
- Wenn Person nähere Details haben möchte, antworten, dass es keine weiteren Details

gibt und das sie die Aufgabe so gut es geht mit den vorhanden Informationen bearbeiten solle

- Nicht unterschiedlich auf die einzelnen Lösungen reagieren, sondern auf alle in einer konstant positiven Art (Nicken, Ok, gut, in Ordnung)
- Wenn Person fragt, ob sie das gut mache, antworten: Es gibt keine richtigen oder falschen Antworten. Ich möchte bloß wissen, wie Sie eine solche Situation händeln würden.

Situation I

Ihr Kind kommt mir einem Blauen Brief nach Hause. In zwei Fächern hat es eine 5 und in drei weiteren Fächern sind die Noten ebenfalls schlecht. Sie wussten nicht, dass Ihr Kind schlecht in der Schule ist und sind besorgt.

1. Nennen Sie mir alle Arten, auf die man das Problem lösen KÖNNTE.
2. Nennen Sie mir die Lösung, die Sie in dieser Situation wählen würden.
3. Schildern Sie mir bitte genau, wie Sie die Lösung umsetzen würden.

Situation II

Der Lehrer Ihres Kindes ruft Sie an und teilt Ihnen mit, dass sich Ihr Kind in der Schule schlecht benimmt. Ihr Kind ärgert andere Kinder, stört den Unterricht und prügelt sich auf dem Schulhof. Der Lehrer ist sehr erregt und sagt, dass Sie etwas tun müssten.

1. Nennen Sie mir alle Arten, auf die man das Problem lösen KÖNNTE.
2. Nennen Sie mir die Lösung, die Sie in dieser Situation wählen würden.
3. Schildern Sie mir bitte genau, wie Sie die Lösung umsetzen würden.

Situation III

Zwei achtjährige Nachbarskinder ärgern, jagen und schlagen sogar Ihr sechsjähriges Kind. Ihr Kind kommt oft traurig und weinend nach Hause.

1. Nennen Sie mir alle Arten, auf die man das Problem lösen KÖNNTE.
2. Nennen Sie mir die Lösung, die Sie in dieser Situation wählen würden.
3. Schildern Sie mir bitte genau, wie Sie die Lösung umsetzen würden.

C. Supplementary Analyses

This section includes additional analyses, which have not been presented in the main thesis, but supplement the results. Table C1 displays comparisons of children with ADHD on and off medication on measures of sustained attention, behavioral inhibition, conflict-monitoring, and risky decision-making. Table C2 shows comparisons of children with ADHD and control children on several single measures of sustained attention derived from the cognitive tasks used in the project, while Table C3 contains correlations between measures of conflict-monitoring and the sustained attention factor score. The sustained attention factor score significantly correlated with the amount of PCE and the size of sequential congruency effects for response times and errors in the Flanker. Higher values on the sustained attention factor score were associated with reduced error rates after incongruent trials and higher sequential congruency effects for response times and errors, respectively (see Table C3). Furthermore, Section C.3. reports on associations of the cognitive and behavioral measures of sustained attention, behavioral inhibition, conflict-monitoring, and risky decision-making with continuous ADHD symptoms derived from the SDQ (Section C.3.1.), the CBCL (Section C.3.2.), and the FBB-ADHS (Section C.3.3.) total scale. Finally, Section C.4. gives the bootstrapping results for indirect effects of conflict-monitoring on ADHD symptoms through risky-decision making, when the total sample of children is considered.

Table C1

Comparisons of Children With ADHD on and of Medication on Sustained Attention, Behavioral Inhibition, Conflict-Monitoring, and Risky Decision-Making

Measure	ADHD on medication			ADHD off-medication				
	n	*M*	*SD*	*n*	*M*	*SD*	*F*	*p*
Sustained Attention								
Factor score	16	0.36	0,88	12	0.43	1.53	0.02	.904
Behavioral Inhibition								
Go/No-Go IE, %	19	.06	.04	16	.07	.04	0.50	.483
Flanker IFE, %	17	.15	.11	16	.10	.11	0.89	.353
Conflict Monitoring,								
Flanker PCS, ms	17	9	22	16	-8	34	1.75	.196
Flanker PCE, %	17	.01	.04	16	.00	.06	0.00	.998
Flanker SCRT, ms	17	-12	54	16	-12	66	0.02	.887
Flanker SCE, %	17	.07	.09	16	.05	.13	0.08	.929
Risky Decision-Making								
GDT risky decision, n	20	11	4.03	20	9.85	5.12	0.25	.623
CCT, n cards	20	4.50	0.85	19	4.81	1.32	0.04	.839
NST, n dices	20	37.50	12.71	20	38.15	15.00	0.26	.617

Note. All analyses controlled for age. IE = inhibition errors; IFE = interference for errors; OE = omission errors; OE ≥ 2 = at least two omission errors in a row; PCE = post-conflict errors; PCS = post-conflict slowing; RTV = response time variability; SCE = sequential congruency effect for errors; SCRT = sequential congruency effect for response times.

Table C2

Comparison of Children With and Without ADHD on Several Measures of Sustained Attention

	ADHD			Control					
Measure	*n*	*M*	*SD*	*n*	*M*	*SD*	*F*	*p*	η_p^2
CPT OE, %	36	4.31	5.00	50	2.85	4.71	2.36	.128	.028
Go/No-Go OE, %	35	6.39	7.19	47	3.54	5.41	4.19	.044	.050
Flanker OE, %	33	7.87	9.61	48	5.45	9.24	2.21	.141	.028
CPT OE ≥ 2, n	36	1.67	2.88	50	0.54	1.25	7.33	.008	.081
Go/No-Go OE ≥ 2, n	35	3.06	4.09	47	1.23	2.61	6.59	.012	.077
Flanker OE ≥ 2, n	33	3.76	5.81	48	2.06	4.55	2.69	.105	.033
CPT RTV	36	0.41	0.16	50	0.34	0.11	6.22	.125	.070
Go/No-Go RTV	35	0.33	0.10	47	0.26	0.07	9.53	.108	.108
Flanker RTV	33	0.46	0.19	48	0.36	0.17	6.94	.010	.082

Note. All analyses controlled for age. OE = omission errors; OE ≥ 2 = at least two omission errors in a row; PCE = post-conflict errors; PCS = post-conflict slowing; RTV = response time variability.

Table C3

Correlations Between Measures of Conflict-Monitoring and Sustained Attention

Measure	1	2	3	4
1. Flanker PCS	-			
2. Flanker PCE	-.18	-		
3. Flanker SCRT	.26*	-.08	-	
4. Flanker SCE	-.13	-.21†	-.03	-
5. Sustained attention factor score	.06	.41*	-.28*	-.39*

Note. †$p < .10$; *$p < .05$; **$p < .01$. PCE = Post-Conflict Error Rate; PCS = Post-Conflict Slowing; SCE = Sequential Congruency Effect for Errors; SCRT = Sequential Congruency Effect for Response Times.

C.3.1. Associations Between Cognitive and Behavioral Performance Measures and the SDQ Hyperactivity Scale

This section examines the association between measures of sustained attention, behavioral inhibition, conflict monitoring, risky decision-making and the SDQ hyperactivity scale. All analyses controlled for age but not for medication, as medication did not improve task performance.

Sustained Attention. A GLM was calculated with the sustained attention deficit factor score as a dependent variable and the SDQ hyperactivity scale and age as centered predictors. A significant effect was revealed for ADHD symptoms, $F(1, 67) = 16.78$, $p = .001$, $\eta_p^2 = .200$, indicating worse sustained attention in younger children and children with higher scores on the SDQ hyperactivity scale. No effects for age were observed.

Go/No-Go. A GLM was calculated with the inhibition error rate as a dependent variable and the SDQ hyperactivity scale and age as centered predictors. No significant effects for age or scores of the SDQ hyperactivity scale were found.

Flanker. To simultaneously investigate whether children with an ADHD diagnosis exhibited a larger Flanker interference effect or a reduction in conflict-monitoring as compared to unaffected children response times and error rates were separately entered into a 2 x 2 repeated measures ANCOVA with the within factors Flanker congruency (congruent vs. incongruent) and Flanker congruency in the preceding trial (congruent vs. incongruent), and age and the SDQ hyperactivity scale as centered covariates.

For response times significant main effects were observed for congruency, $F(1, 78) = 36.83$, $p < .001$, $\eta_p^2 = .321$, and age, $F(1, 78) = 14.71$, $p < .001$, $\eta_p^2 = .159$, indicating slower response times for incongruent trials and younger children. In addition, a marginally significant interaction was found for ADHD symptoms and congruency in the preceding trial, $F(1, 78) = 3.94$, $p = .051$, $\eta_p^2 = .048$, indicating less post-conflict slowing for children with more or stronger ADHD symptoms. No other main effects or interactions reached significance.

For error rates significant main effects were observed for congruency, $F(1, 78) = 133.44$, $p < .001$, $\eta_p^2 = .631$, congruency in the preceding trial, $F(1, 78) = 6.95$, $p = .010$, $\eta_p^2 = .082$, and the SDQ hyperactivity scale, $F(1, 78) = 11.14$, $p = .001$, $\eta_p^2 = .125$, indicating higher error rates for incongruent trials, a reduced error rate after incongruent trials, and a generally pronounced error rate in children with higher scores in the SDQ hyperactivity scale. A significant interactions effects were found for congruency and congruency in the preceding trial, $F(1, 78) = 45.26$, $p < .001$, $\eta_p^2 = .367$, demonstrating a sequential congruency effect. Additionally, marginally significant three-way interactions between congruency, congruency in the preceding trial and age, $F(1, 78) = 3.62$, $p = .061$, $\eta_p^2 = .044$, as well as congruency, congruency in the preceding trial, and SDQ hyperactivity scores, $F(1, 78) = 3.40$, $p = .069$, $\eta_p^2 = .042$, indicating that the sequential congruency effect was less pronounced in younger children and children with higher scores on the SDQ hyperactivity scale. No other main effects or interactions were found.

GDT. To analyze whether children with higher scores on the SDQ hyperactivity scale played more risky in the GDT, a GLM was calculated with the number of risky decisions as dependent variable and SDQ hyperactivity scale and age as centered predictors. Results yielded a significant effect of SDQ scores only, $F(1, 78) = 13.90$, $p < .001$, $\eta_p^2 = .135$, with stronger affected children making a higher number of risky decisions

CCT. To analyze whether children with higher scores on the SDQ hyperactivity scale played more risky in the CCT, a GLM was calculated with the average number of cards turned over as the dependent variable and the scores on the SDQ hyperactivity scale and age as centered predictors. No significant effects for age or ADHD symptoms were found.

NST. To analyze whether children with higher scores on the SDQ hyperactivity scale played more risky in the NST, a GLM was calculated with the number of dice throws in the NST as the dependent variable and scores on the SDQ hyperactivity scale and age as centered predictors. No significant effects for age or ADHD symptoms were found.

C.3.2. Associations Between Cognitive and Behavioral Performance Measures and the CBCL Attention Problem Scale

This section examines the association between measures of sustained attention, behavioral inhibition, conflict monitoring, risky decision-making and the CBCL attention problem scale. All analyses controlled for age but not for medication, as medication did not improve task performance.

Sustained Attention. GLM was calculated with the sustained attention deficit factor score as a dependent variable and the CBCL attention problem scale and age as centered predictors. A significant effect was revealed for ADHD symptoms only, $F(1, 67) = 19.50$, $p < .001$, $\eta_p^2 = .225$, indicating worse sustained attention in children with higher scores on the CBCL attention problem scale.

Go/No-Go. A GLM was calculated with the inhibition error rate as a dependent variable and the SDQ hyperactivity scale and age as centered predictors. No significant effects for age or CBCL attention problems were found.

Flanker. To simultaneously investigate whether children with higher scores on the CBCL attention problem scale exhibited a larger Flanker interference effect or a reduction in conflict-monitoring as compared to unaffected children response times and error rates were separately entered into a 2 x 2 repeated measures ANCOVA with the within factors Flanker congruency (congruent vs. incongruent) and Flanker congruency in the preceding trial (congruent vs. incongruent), and age and the CBCL attention problem scale as centered covariates.

For response times significant main effects were observed for congruency, $F(1, 78) = 37.34$, $p < .001$, $\eta_p^2 = .324$, and age, $F(1, 78) = 15.36$, $p < .001$, $\eta_p^2 = .164$, indicating slower response times for incongruent trials and younger children. No other main effects or interactions reached significance.

For error rates significant main effects were observed for congruency, $F(1, 78) = 136.11$, $p < .001$, $\eta_p^2 = .636$, congruency in the preceding trial, $F(1, 78) = 7.41$, $p = .008$, $\eta_p^2 = .087$, and the CBCL attention problem scale, $F(1, 78) = 8.65$, $p = .004$, $\eta_p^2 = .100$, indicating higher error rates for incongruent trials, a reduced error rate after incongruent trials, and a generally pronounced error rate in children with higher scores on the CBCL attention problem scale. In addition, a significant interactions effect was found for congruency and congruency in the preceding trial, $F(1, 78) = 46.23$, $p < .001$, $\eta_p^2 = .372$, revealing a sequential congruency effect. As indicated by marginally significant three-way interactions between congruency, congruency in the preceding trial and age, $F(1, 78) = 3.90$, $p = .052$,

η_p^2 = .048, as well as congruency, congruency in the preceding trial, and CBCL attention problem scores, $F(1, 78) = 3.06$, $p = .084$, $\eta_p^2 = .038$, this sequential congruency effect was less pronounced in younger children or children with higher scores on the CBCL attention problem scale. Furthermore, a marginally significant interaction between congruency in the preceding trial and the CBCL attention problem scale, $F(1, 78) = 2.94$, $p = .090$, $\eta_p^2 = .036$, indicated a smaller error reduction after incongruent trials for children with stronger attention problems. No other main effects or interaction effects were found.

GDT. To analyze whether children with higher scores on the CBCL attention problem scale played more risky in the GDT, a GLM was calculated with the number of risky decisions as dependent variable and the CBCL attention problem scale and age as centered predictors. Results yielded a significant effect of CBCL scores only, $F(1, 78) = 5.84$, $p = .018$, $\eta_p^2 = .062$, with stronger affected children making a higher number of risky decisions

CCT. To analyze whether children with higher scores on the CBCL attention problem scale played more risky in the CCT, a GLM was calculated with the average number of cards turned over as the dependent variable and the scores on the CBCL attention problem scale and age as centered predictors. No significant effects for age or ADHD symptoms were found.

NST. To analyze whether children with higher scores on the CBCL attention problem scale played more risky in the NST, a GLM was calculated with the number of dice throws in the NST as the dependent variable and scores on the CBCL attention problem scale and age as centered predictors. No significant effects for age or ADHD symptoms were found.

C.3.3. Associations Between Cognitive and Behavioral Performance Measures and the FBB-ADHS Total Scale

This section examines the association between measures of sustained attention, behavioral inhibition, conflict monitoring, risky decision-making and the FBB-ADHS total scale. All analyses controlled for age but not for medication, as medication did not improve task performance.

Sustained Attention. To analyze whether children with higher scores on the FBB-ADHS total scale had stronger deficits in sustained attention, a GLM was calculated with the sustained attention deficit factor score as a dependent variable and the FBB-ADHS total score and age as centered predictors. A significant effect was revealed for ADHD symptoms only, $F(1, 67) = 11.49$, $p = .001$, $\eta_p^2 = .146$, indicating worse sustained attention in children with higher scores on the FBB-ADHS total scale.

Go/No-Go. To analyze whether children with higher scores on the FBB-ADHS total scale had stronger deficits in withholding of responses, a GLM was calculated with the inhibition error rate as a dependent variable and the FBB-ADHS total score and age as centered predictors. No significant effects for the FBB-ADHS total scale age, or age were found.

Flanker. To simultaneously investigate whether children with higher scores on the FBB-ADHS total scale exhibited a larger Flanker interference effect or a reduction in conflict-monitoring as compared to unaffected children response times and error rates were separately entered into a 2 x 2 repeated measures ANCOVA with the within factors Flanker congruency (congruent vs. incongruent) and Flanker congruency in the preceding trial (congruent vs. incongruent), and age and the FBB-ADHS total scale as centered covariates.

With regard to response times, significant main effects were observed for congruency, $F(1, 78) = 36.70$, $p < .001$, $\eta_p^2 = .320$, and age, $F(1, 78) = 15.78$, $p < .001$, $\eta_p^2 = .168$, indicating slower response times for incongruent trials and younger children. In addition, a marginally significant interaction between congruency in the preceding trial and the FBB-ADHS total scale, $F(1, 78) = 3.20$, $p = .078$, $\eta_p^2 = .039$, revealed less post-conflict slowing in children with more or stronger ADHD symptoms. No other main effects or interactions reached significance.

With regard to error rates, significant main effects were observed for congruency, $F(1, 78) = 134.04$, $p < .001$, $\eta_p^2 = .632$, congruency in the preceding trial, $F(1, 78) = 6.82$, $p = .011$, $\eta_p^2 = .080$, and the FBB-ADHS total scale, $F(1, 78) = 6.09$, $p = .016$, $\eta_p^2 = .072$, indicating higher error rates for incongruent trials, a reduced error rate after incongruent trials, and a generally pronounced error rate in children with higher scores on the FBB-ADHS total scale. In addition, a significant interactions effect was found for congruency and congruency in the preceding trial, $F(1, 78) = 43.95$, $p < .001$, $\eta_p^2 = .360$, revealing a sequential congruency effect. No other main effects or interaction effects were found.

GDT. To analyze whether children with higher scores on the FBB-ADHS total scale played more risky in the GDT, a GLM was calculated with the number of risky decisions as dependent variable and the FBB-ADHS total score and age as centered predictors. Significant effects were observed for the FBB-ADHS total scale, $F(1, 89) = 7.75$, $p = .007$, $\eta_p^2 = .080$. Children with more or stronger ADHD symptoms made a higher number of risky decisions. No effects of age were observed.

CCT. To analyze whether children with higher scores on the FBB-ADHS total scale played more risky in the CCT, a GLM was calculated with the number of cards turned over as

dependent variable and the FBB-ADHS total score and age as centered predictors. No significant effects for the FBB-ADHS total scale or age were found.

NST. To analyze whether children with higher scores on the FBB-ADHS total scale played more risky in the NST, a GLM was calculated with the number of dice throws as dependent variable and the FBB-ADHS total score and age as centered predictors. No significant effects for the FBB-ADHS total scale or age were found.

C.4. Indirect Effect of Conflict-Monitoring on ADHD Symptoms

While no indirect effects of conflict-monitoring on ADHD symptoms through risky decision making were observed in the subsample used for the analyses in the current thesis (see Section, 4.4.), the following analysis included all children, regardless whether they had properly worked on the task or not (see criteria for elimination of participants in section 3.5.2.). When effects of age were controlled, conflict-monitoring, assessed by the sequential congruency effect for errors in the Flanker task, predicted risky decision making, b = -10.89, $t(84)$ = -2.10, p = .039, but not ADHD symptoms, b = -2.48, $t(84)$ = -0.98, p = .328. Children with a smaller sequential congruency effect for errors made a higher number of risky decisions. When ADHD symptoms were predicted simultaneously by conflict-monitoring and risky decision-making, risky decision-making remained significant, b = 0.14, $t(84)$ = 2.78, p = .004, whereas conflict-monitoring was not a significant predictor, b = -2.48, $t(84)$ = -0.98, p = .328. A bias-corrected bootstrap 95% CI based on 20.000 bootstrap samples indicated an indirect effect of conflict-monitoring through risky decision-making, b = -1.56, 95%
CI: [-4.02, -0.11].

D. Normative Solutions for Risky Decision-Making Tasks

D.1. Normative Solution for the CCT

The mathematically optimal decision-making strategy in the CCT entails turning over the number of cards that maximizes the expected outcome, given the specific levels of gain amounts, loss amounts and number of loss cards. The following section illustrates the mathematical structure and the normative solution of this problem.

Let n_{cards} be the number of loss cards not yet turned over ($0 \leq n_{\text{cards}} \leq 16$); let n_{loss} be the number of hidden loss cards in the deck (one or two); let g be the gain amount per winning card (one or five); let l be the loss amount (10 or 40) and let EV_{n} be the expectancy value for turning over the nth card.

If p_{win} is the probability that the next card to be turned over is a win card, it can be computed as:

$$p_{win} = \frac{n_{cards} - n_{loss}}{n_{cards}} . \quad (1)$$

In similar, if p_{loss} is the probability that the next card to be turned over is a loss card, it can be computed as

$$p_{loss} = \frac{n_{loss}}{n_{cards}} . \quad (2)$$

The expectancy value for the outcome of turning over the nth card, is given by

$$EV_n = p_{win} \cdot g - p_{loss} \cdot l . \quad (3)$$

Substituting (1) and (2) in expression (3), we obtain

$$EV_n = \frac{n_{cards} - n_{loss}}{n_{cards}} \cdot g - \frac{n_{loss}}{n_{cards}} \cdot l . \quad (4)$$

The normative solution postulates that no further card should be turned over if $EV_n < 0$, thus if

$$\frac{n_{cards} - n_{loss}}{n_{cards}} \cdot g - \frac{n_{loss}}{n_{cards}} \cdot l < 0 . \quad (5)$$

Solving (5) for n_{cards}, we obtain

$$(n_{cards} - n_{loss}) \cdot g - n_{loss} \cdot l < 0 \quad (6)$$

$$\Leftrightarrow g \cdot n_{cards} - g \cdot n_{loss} - n_{loss} \cdot l < 0 \quad (7)$$

$$\Leftrightarrow -g \cdot n_{loss} - n_{loss} \cdot l < -g \cdot n_{cards} \quad (8)$$

$$\Leftrightarrow g \cdot n_{loss} + n_{loss} \cdot l > g \cdot n_{cards} \quad (9)$$

$$\Leftrightarrow n_{loss}(g + l) > g \cdot n_{cards} \quad (10)$$

$$\Leftrightarrow \frac{n_{loss}(g + l)}{g} > n_{cards}. \quad (11)$$

According to Equation (11) a child should stop turning over cards if the remaining number of cards, n_{cards}, becomes smaller than the left hand side of the equation, which depends on the gain amount g, the loss amount l, the number of loss cards in the deck, n_{loss}. Hence, children should turn over the most cards if the gain amount g was five, the loss amount l was 10, and the number of loss cards in the deck n_{loss} was one. Here, children should stop when two cards remain on the table. In contrast, if the gain amount g was one, the loss amount l was 40, and the number of loss cards in the deck n_{loss} was two, children should not turn over a single card. However, as children are forced to turn over at least one card, they should stop after the first card they turned over.

D.2. Normative Solution for the NST

The mathematically optimal decision-making strategy in the NST gives a decision criterion when to stop rolling the dice, given the sum of the already accumulated points. The following section illustrates the mathematical structure and the normative solution of this problem.

Let PA_{n} be the sum of the already accumulated points after the dice was rolled for n times; let EV_{win} be the expectancy value for each time the dice is thrown under the condition that a winning number will be rolled and let p_{loss} be the probability of rolling a six.

The expectancy value EV_{win} can be computed by the following formula:

$$EV_{win} = \frac{1}{6}\sum_{i=1}^{5} i = \frac{1}{6} \cdot 15 = 2.5. \quad (12)$$

The expectancy value of rolling the dice for the $n+1$th time, $EV_{\text{n+1}}$, is given by

$$EV_{n+1} = EV_{win} - p_{loss} \cdot PA_n . \quad (13)$$

Substituting (12) in (13) and with $p_{\text{loss}} = ⅙$ we obtain

$$EV_{n+1} = 2.5 - \frac{1}{6} \cdot PA_n . \quad (14)$$

The normative solution postulates that no further dice should be rolled if $EV_{\text{n+1}} < 0$, thus if

$$2.5 - \frac{1}{6} \cdot PA_n < 0 . \quad (15)$$

Solving (15) for PA_{n}, we obtain

$$15 < PA_n . \quad (16)$$

Hence, a child should stop further rolling the dice, if she or he has already accumulated more than 15 points.