Let's Go or Not Go!
Behavioral Investigation of the Effect of Methylphenidate on Response Inhibition in Children with Attention Deficit Hyperactivity Disorder

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ADHD is not a problem with knowing what to do;

it is a problem with doing what you know.

-Russell A. Barkley (2004)

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"Watch you wish then you will get it."

-An Lee (Taiwan), Director of movie "Life of Pi"

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ABSTRACT (ENGLISH)

The core symptoms of children with attention deficit hyperactivity disorder (ADHD) are inattention, hyperactivity, and impulsivity (American Psychiatric Association, 2000). A primary deficit in inhibitory control is proposed to account for dysfunctional behavior patterns associated with this disorder (Barkley, 1997). Early research has shown that medication (e.g., Methylphenidate) is effective when it comes to enhancing inhibitory control (e.g., Pliszka et al., 2007). Therefore, by incorporating relevant results from the previous study (Paul-Jordanov, Bechtold, & Gawrilow, 2010)[1], we explored the effect of medication on inhibition in children with ADHD in Germany and Taiwan at the same time. Children with ADHD and normally developing children in Germany and Taiwan were recruited for the current studies. In Study 1, using a Go/NoGo task, we investigated the response inhibition of children with ADHD and their healthy counterparts at both sites. Significantly, children with ADHD in Germany showed deficits in response inhibition compared to their healthy counterparts. A group difference, however, was not pronouncedly observed in the Taiwanese sample. In Study 2, the effect of medication on response inhibition was explored in children with ADHD. After the treatment of medication, the response inhibition in the ADHD group in Germany was ameliorated, while the effect of medication did not reach any significance in the Taiwanese sample. Results and implications for future studies are discussed.

Keywords: attention deficit hyperactivity disorder, cross-cultural study, executive functions, medication, response inhibition

[1] Relevant results from the previous study (Paul-Jordanov, Bechtold, & Gawrilow, 2010) are included in the current research with author's permission.

ABSTRACT (GERMAN)

Die Kernsymptome von Kindern mit einer Aufmerksamkeitsdefizit-/Hyperaktivitätsstörung (ADHS) sind Unaufmerksamkeit, Hyperaktivität und Impulsivität (American Psychiatric Association, 2000). Es wird vermutet, dass ein Kerndefizit an inhibitorischer Kontrolle, das in Verbindung mit dieser Störung steht, ursächlich ist für das dysfunktionale Verhalten (Barkley, 1997). Die frühe Forschung hat gezeigt, dass mit Medikamenten (z.B. Methylphenidat) positiv auf die inhibitorische Kontrolle eingewirkt werden kann (z.B. Pliszka et al., 2007). Daher wurde, unter Einbeziehung der einschlägigen Ergebnisse aus der vorangegangenen Studie[2] (Paul-Jordanov, Bechtold & Gawrilow, 2010), die hemmende Wirkung von Medikamenten bei Kindern mit ADHS untersucht, und zwar in Deutschland und in Taiwan parallel. An der aktuellen Studie nahmen sowohl Kinder mit ADHS als auch normal entwickelte Kinder (Kontrollgruppe) teil. In Studie 1 untersuchten wir mit einem Go/NoGo-Task die Handlungsinhibition von Kindern mit ADHS und der Kontrollgruppe an beiden Standorten. Deutlich wurden hierbei die Defizite in der Handlungsinhibition bei mit ADHS diagnostizierten Kindern in Deutschland im Vergleich zu den gesunden Kindern. Die Probanden in Taiwan wiesen diesbezüglich dagegen keinen so deutlichen Unterschied auf. In Studie 2 wurde die Wirkung der Medikamente auf die Handlungsinhibition bei Kindern mit ADHS untersucht. Nach der Behandlung der Medikamente wurde die Handlungsinhibition der ADHS-Gruppe in Deutschland gemildert, während der Effekt bei den Probanden in Taiwan nicht die gleiche Bedeutung erreichte. Ergebnisse und Implikationen für zukünftige Studien werden diskutiert.

Schlüsselwörter: Aufmerksamkeitsdefizit-/Hyperaktivitätsstörung, exekutive Funktionen, Handlungsinhibition, interkulturelle Studie, Medikamente

[2] Relevante Ergebnisse aus der vorangegangenen Studie (Paul-Jordanov, Bechtold, & Gawrilow, 2010) werden in der aktuellen Forschung mit Erlaubnis des Autors herangezogen.

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[3] German versions (excluding Appendixes F & G) are available from the author upon request.

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Children with attention deficit hyperactivity disorder (ADHD) are impulsive and have difficulty regulating their behaviors in everyday life (Nigg, 2001; Reid, Trout, & Schartz, 2005), which may arise from poor inhibitory control (Barkley, 1997). Thus, they perform more poorly than do non-ADHD children on the Go/NoGo task, a task that requires a response to one type of stimulus but the withdrawal of a prepotent response to another type of stimulus. Earlier studies have pointed to ADHD symptoms as persisting in most clinically diagnosed children into their adolescence (Barkley, 2004; Gau, Chiu, Shang, Cheng, & Soong, 2009).

Methylphenidate (MPH) is the most commonly prescribed and widely discussed stimulant medication regarding the treatment of ADHD symptoms (Goldman, Genel, Bezman, & Slanetz, 1998). MPH is effective in ameliorating relevant symptom severity (e.g., Jensen, 2009). Children with ADHD can also benefit from MPH on inhibitory control as measured by inhibition tasks, which has been demonstrated in several behavioral, electrophysiological, and neuroimaging studies (e.g., Broyd et al., 2005; Hart, Radua, Nakao, Mataix-Cols, & Rubia, 2013).

Inhibitory control is a developmental pattern; Wanless et al. (2011) noted, "…patterns of development are contextually specific and a skill such as behavioral regulation must be examined within each society in order to understand its unique properties and meaning (Cole, 1996; Shweder et al., 1998)" (p.366). Therefore, in the current research, by revisiting and incorporating the relevant results from the prior research (Paul-Jordanov, Bechtold, & Gawrilow, 2010), we explore inhibition among children with ADHD in Germany and Taiwan with the identical study design and task and further investigate the effect of MPH on response inhibition in children with ADHD at both sites.

To fulfill these purposes, in Chapter 2, we first give a brief overview on the topics relevant to ADHD, including diagnostic criteria, psychiatric comorbidity, and etiology. Chapter 3 focuses on response inhibition in ADHD by selectively reviewing behavioral, electrophysiological, and neuroimaging studies. Chapter 4 reviews the effects of medication and self-regulation strategies on response inhibition in ADHD. Chapter 5 introduces the potential impact of culture on children's behaviors—one critical motive that guides the current research. Two studies are presented in Chapters 6 and 7, respectively. We first compare response inhibition measured by the Go/NoGo task (Paul et al., 2007) between children with ADHD and their healthy counterparts in Germany and Taiwan. Second, we explore the effect of medication on response inhibition in children with ADHD at both sites. In Chapter 8, general discussion and implications for future research regarding the findings from the current research are given. Finally, Chapter 9 concludes with implications for clinical practice.

2. Attention Deficit Hyperactivity Disorder

Attention deficit hyperactivity disorder (ADHD) is a childhood-onset disorder characterized by developmentally inappropriate behaviors such as inattention, hyperactivity, and impulsivity (American Psychiatric Association, 2000) with estimated prevalence rates of 5–10% among school-age children worldwide (Faraone, Sergeant, Gillberg, & Biederman, 2003), 5.3% in Germany (Huss, Hoelling, Kurth, & Schlack, 2008), and 7.5% in Taiwan (Gau, Chong, Chen, & Cheng, 2005). Children diagnosed with ADHD frequently display difficulty sustaining their attention or regulating their behaviors in everyday life (Nigg, 2001; Reid, Trout, & Schartz, 2005). They, for instance, cannot wait for their turn patiently when playing with others, tend to interrupt others when they are not supposed to, or seem not to listen when spoken to (American Psychiatric Association, 2000). Impacts of ADHD cardinal symptoms may persist into adolescence and adulthood (Barkley, 2004, 2010; Barkley, DuPaul, & McMurray, 1990; Gau, Chiu, Shang, Cheng, & Soong, 2009; Miller, Ho, & Hinshaw, 2012).

2.1 Diagnostic Criteria

Two international classification systems are widely used by clinicians and psychiatrists for the diagnosis of ADHD: the *Diagnostic and Statistical Manual of Mental Disorders* (DSM; American Psychiatric Association, 2000, 2013) and the *International Statistical Classification of Diseases and Related Health Problems* (ICD; World Health Organization, 2008). The diagnostic criteria of ADHD in the text revision of the fourth version of the DSM (DSM-IV-TR; American Psychiatric Association, 2000) are based on two core symptom domains: inattention and hyperactivity/impulsivity (see Table 2.1). According to varying severity levels displayed on these two domains, three subtypes are outlined in ADHD: the predominantly inattentive type, the predominantly hyperactive/impulsive type, and the combined type.

Children are diagnosed with ADHD-predominantly inattentive type (ADHD-PI) when they meet criterion A in Table 2.1, while those meeting criterion B are diagnosed with ADHD-predominantly hyperactive/impulsive type (ADHD-HI), and those meeting both criteria A and B are diagnosed with ADHD-combined type (ADHD-C). Additional criteria also need to be met, including (a) the emergence of some inattentive or hyperactive/impulsive symptoms that cause impairments before the age of 7; (b) symptoms are required to cause impairments in at least two settings (e.g., both school and home); (c) evidence of clinical significant impairments is clear in social, academic, or occupational functioning, and (d) ADHD symptoms are not better explained by other mental disorders (e.g., pervasive developmental disorder, schizophrenia, and other psychotic disorders; American Psychiatric Association, 2000).

In the tenth version of the ICD (ICD-10), the term *hyperkinetic disorder* (HD) is used to describe the signs of ADHD. Unlike the DSM-IV-TR, in which three subtypes are defined, no subtypes are distinguished in the ICD-10; only when individuals simultaneously meet criteria described in each core symptom (i.e., inattention and hyperactivity/impulsivity) are they diagnosed with HD. Other diagnostic criteria in the ICD-10 are similar to those in the DSM-IV-TR. Therefore, a diagnosis of ADHD based on the DSM-IV-TR is most consistent with a diagnosis of ICD-10 HD.

Diagnostic criteria are associated with the variability of prevalence rates of ADHD/HD, which has been reported in several studies in different cultures and countries (e.g., Doepfner, Breuer, Wille, Erhart, & Ravens-Sieberer, 2008; Gau et al., 2005; Smalley et al., 2007). In general, the prevalence of ADHD based on the DSM-IV is higher than that of HD when using the ICD-10 criteria (Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007; Swanson et al., 1998). For example, Doepfner et al. (2008) reported in a national sample of children and adolescents aged 7-17 years that the prevalence rate of ADHD

according to DSM-IV criteria was 5.0%, and the rate of ICD-10 based HD was 1.0%. Other potential factors related to prevalence rates of ADHD/HD include source of information, patient's age and gender, and impairments in other functioning (Gomez, Harvey, Quick, Scharer, & Harris, 1999; Polanczyk et al., 2007).

Table 2.1

Diagnostic Criteria for Attention Deficit Hyperactivity Disorder

Either A or B:

A. Six (or more) of the following symptoms of **inattention** have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

Inattention

(a) often fails to pay close attention to detail or makes careless mistakes in schoolwork, work, or other activities

(b) often has difficulty sustaining attention in tasks or play activities

(c) often does not seem to listen when spoken to directly

(d) often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (not due to oppositional behavior or failure to understand instructions)

(e) often has difficulty organizing tasks and activities

(f) often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (such as schoolwork or homework)

(g) often loses things necessary for tasks or activities (e.g., toys, school assignments, pencils, books, or tools)

(h) is often easily distracted by extraneous stimuli

(i) is often forgetful in daily activities

B. Six (or more) of the following symptoms of **hyperactivity-impulsivity** have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

Hyperactivity

(a) often fidgets with hands or feet or squirms in seat

(b) often leaves seat in classroom or in other situations in which remaining seated is expected

(c) often runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults, may be limited to subjective feelings or restlessness)

(d) often has difficulty playing or engaging in leisure activities quietly

(e) is often "on the go" or often acts as if "driven by a motor"

(f) often talks excessively

Impulsivity

(g) often blurts out answers before questions have been completed

(h) often has difficulty awaiting turn

(i) often interrupts or intrudes on others (e.g., butts into conversations or games)

Source from DSM-IV-TR (American Psychiatric Association, 2000)

2.2 Psychiatric Comorbidity

According to Pliszka (2009, p. 2), "comorbidity can be simply defined as two or more diseases occurring in the same individual." It has been noted that approximately half of children and adolescents with ADHD are diagnosed with comorbid disorders, including oppositional defiant disorder (ODD), conduct disorder (CD), mood disorder, anxiety disorders, learning disabilities, and Tic disorder (August, Realmuto, MacDonald, Nugent, & Crosby, 1996; Gau et al., 2010; Pliszka, 1998; Spencer, 2006).

The rate of symptoms of psychiatric comorbid disorders may vary as a function of gender and/or subtype in childhood ADHD. However, findings from relevant studies are mixed. In a clinically-referred sample (Biederman et al., 2002), boys with ADHD were at significantly greater risk for comorbid externalizing problems (ODD/CD) than girls with this disorder, while the prevalence rates of ODD/CD were reported to be similar for boys and girls with ADHD in a community-based study, regardless of ADHD subtype (Levy, Hay, Bennett, & McStephen, 2005). In terms of comorbid internalizing problems, significant gender differences were observed for separation anxiety disorder (SAD) in the inattentive subtype and for generalized anxiety disorder (GAD) in the combined subtype; in both cases, higher rates of symptoms existed in girls than in boys (Levy et al., 2005).

Furthermore, several factors are predictive for comorbidities in adolescents with persistent ADHD. For instance, more severe childhood ADHD symptoms predict ODD/CD at adolescence. Older age is a higher risk for mood disorders. Comorbidity of Tic disorder is associated with a longer duration of medication treatment (Gau et al., 2010).

2.3 Etiology

ADHD is a highly multifactorial disorder, for which a number of causes have been proposed. The current view endorses the theory that genetic and neurobiological factors play a critical role in the cause of ADHD, while environmental and psychosocial influences may shape the expression of ADHD symptoms (Milberger, Biederman, Faraone, Guite, & Tsuang, 1997). A possible developmental pathway for ADHD (Mash & Wolfe, 2007) could be that genetic risk or prenatal alcohol exposure and pregnancy complications expose children to a risk of disturbances in dopamine transmission and abnormalities in the frontal lobe and basal ganglia. This then leads to a failure in adequately suppressing inappropriate responses, cognitive deficits, behavioral clinical symptoms, and impaired social functioning. Below is a selective overview on various etiologies for ADHD (see Figure 2.1).

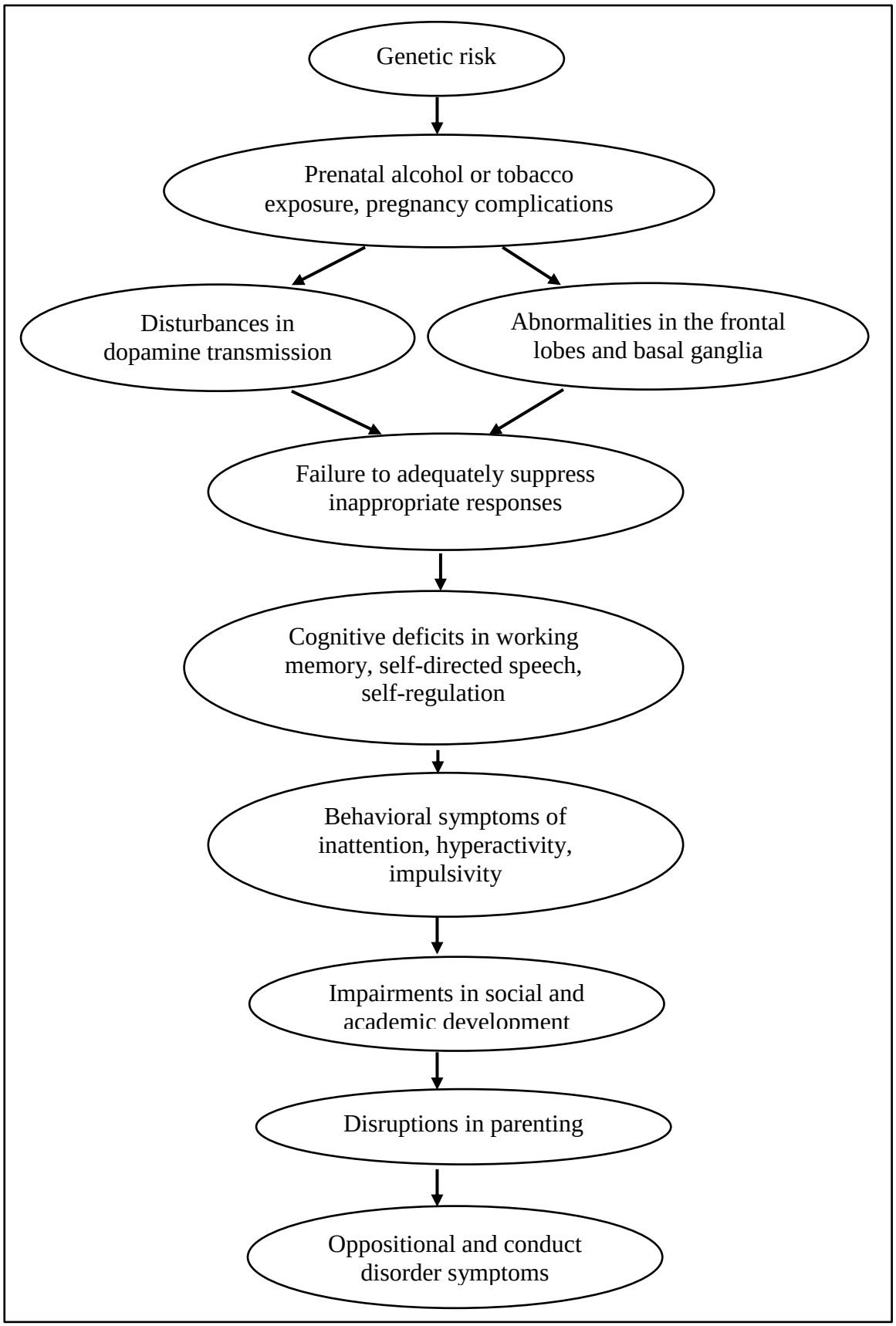

Figure 2.1 A possible developmental pathway for ADHD. Figure adapted from Mash and Wolfe (2007, p. 138).

2.3.1 Genetic Factors

Evidence from twin and adoption studies shows that ADHD is a familial and heritable disorder with a mean heritability estimate of 0.76 (Faraone et al., 2005). Several candidate genes involving the etiology of ADHD are implicated in molecular genetic studies (Faraone et al., 2005). Among these, genes encoding the dopamine transporter and receptors have been extensively studied, e.g., the dopamine receptor D4 and D5 (DRD4 and DRD5) and the dopamine transporter (DAT1/SLC6A3) genes (Faraone, Doyle, Mick, & Biederman, 2001). A meta-analysis (Faraone et al., 2001) of case-control and family-based studies examining the association between ADHD and the DRD4 gene demonstrated a small but statistically significant association between ADHD and the DRD4 7-repeat allele in each analysis. The combined estimates of the odds ratios (ORs) were 1.9 in control studies (95% confidence interval [CI] 1.4-2.2) and 1.4 in family studies (95% CI 1.1-1.6). A more recent analysis (Li, Sham, Owen, & He, 2006) also showed that the 7-repeat and 5-repeat alleles of the DRD4 gene, as well as the 148-bp allele of the DRD5 gene, conferred increased risk for ADHD. Similarly, in a sample of Taiwanese children and adolescents with DSM-IV-diagnosed ADHD, Chen et al. (2003) found that the DAT1 gene increased susceptibility to ADHD (OR = 2.9). Moreover, variations of the DAT1 gene may primarily have an impact on the ADHD-predominantly inattentive subtype, not the other two subtypes (Shang, Gau, Liu, & Hwu, 2011). By far, evidence supports a critical dopamine component in the pathogenesis of ADHD. Other candidate genes have been of considerable interest to researchers, such as those related to the noradrenergic (NET/SLC6A2, ADRA2A, ADRA2C) and serotonergic (5-HTT/SLC6A4, HTR1B, HTR2A, TPH2) systems (for a review of this literature, see Banaschewski, Becker, Scherag, Franke, & Coghill, 2010; Faraone et al., 2005).

2.3.2 Pregnancy and Birth Complications

Biological adversity may contribute to the etiology of ADHD in addition to genetic influences. Other discussed biological factors include pregnancy, delivery, and infancy complications, and maternal smoking and alcohol-drinking during pregnancy. A positive association exists between childhood ADHD or associated impaired cognitive functioning and pregnancy, delivery, and infancy complications (Milberger et al., 1997). More specifically, using linear and logistic regression models, Milberger et al. (1997) revealed that risk factors such as maternal illness/infection, neonatal medical problems, maternal substance use/family problems, and maternal emotional problems/difficult infant were significantly associated with higher rates of ADHD, as well as with cognitive impairments in children. It was these specific complications reflecting chronic exposures that accounted for the association. Evidence from studies with a population-based sample (Thapar et al., 2003) or a clinical sample (Milberger, Biederman, Faraone, Chen, & Jones, 1996) demonstrated that maternal smoking during pregnancy was positively associated with ADHD in children, and the association remained significant even after other potential confounds were taken into consideration.

2.3.3 Brain Function

Frontal lobe dysfunction has been an active focus of ADHD research over the past several decades, based on the observation that experimental animals or human patients with frontal lesions sometimes produce ADHD-like symptoms of hyperactivity or impulsivity alone or in combination (Fuster, 1993).

Brain volume and structure. In a landmark neuroimaging study, Castellanos et al. (2002) compared brain volumes in the cerebrum, cerebellum, gray and white matter for the four major lobes (i.e., frontal, temporal, parietal, and occipital lobes), and caudate nucleus in approximately 150 children and adolescents with ADHD and 140 age- and sex- matched

healthy controls. Their primary findings were (a) brain volumes in all regions are significantly smaller in those with ADHD than in controls, regardless of sex; (b) group differences on the white matter for all lobes pronouncedly exist between unmedicated individuals with ADHD and controls, but not between medicated individuals with ADHD and controls, and (c) developmental trajectories of brain volume abnormalities (except caudate) that are independent of medical status remain stable and parallel for individuals with ADHD and healthy controls during childhood and adolescence. These findings suggest that influences of genes and/or early environment on brain development in ADHD are fixed, nonprogressive, and not related to stimulant treatment.

With respect to brain structures, anatomical measures of frontostriatal circuitry (specifically, the prefrontal cortex, caudate nucleus, and globus pallidus) are abnormal in children with ADHD. Casey et al. (1997) demonstrated that these frontostriatal regions were correlated with performance on response inhibition tasks, and that significant correlations between task performance and anatomic measures of the prefrontal cortex and caudate nuclei existed predominantly in the right hemisphere, supporting the important role of the right frontostriatal structures in response inhibition. Other brain regions, such as the corpus callosum (Giedd et al., 1994), basal ganglia (Aylward et al., 1996), and anterior cingulate cortex (Bush et al., 1999) appear to be abnormal in individuals with ADHD.

Neurotransmitters in the brain. Symptomatology of ADHD is associated with the dysregulation (i.e., too much or too little) of neurochemical systems in the brain, including norepinephrine, epinephrine, dopamine, and serotonin systems. A multistage model (Pliszka, McCracken, & Maas, 1996) is hypothesized to describe the underlying mechanisms in the pathophysiology of ADHD by focusing on the interaction of norepinephrine, epinephrine, and dopamine in modulation of attention and impulsive control. In this hypothesis, disruptions of neurotransmitter transmission at different stages are comprehensively

implicated. A higher level of norepinephrine or lower level of epinephrine, which often exists in children with ADHD, may be associated with inattention, as the imbalanced levels of neurotransmitters indirectly lead to a dysfunction in the posterior norepinephrine-mediated attention system. The posterior attention system would then fail to efficiently separate from old stimuli, shift attention to the new ones, and read them out to the anterior dopamine-mediated system. Disruptions in the brain mechanism (i.e., the brainstem) or the dopamine system in the anterior lobe (e.g., the dorsolateral or the orbital medial frontal cortex) may result in individuals being unable to readily make responses, as dopamine plays a critical role in the execution of behaviors. This indicates that insufficient dopamine in the brain is correlated with impulse control.

Moreover, the severity of childhood aggression and parental aggression is associated with low serotonergic function in children with comorbid ADHD and disruptive behavior disorders, indicating that low serotonin may put these children at greater risk for poor long-term outcomes, adolescence, or adulthood aggression (Halperin et al., 1997).

2.3.4 Psychopathology

Deficits in executive functioning. According to Welsh and Pennington (1988), executive function is defined in a developmental neuropsychological way:

> ….as the ability to maintain an appropriate problem-solving set for attainment of a future goal (Bianchi, 1992; Luria, 1966). This set can involve one or more of the following: (a) an intention to inhibit a response or to defer it to a later more appropriate time, (b) a strategic plan of action sequences, and (c) a mental representation of the task, including the relevant stimulus information encoded into memory and the desired future goal-state. (pp. 201–202)

By definition, executive functions include set-shifting, inhibition, working memory, planning, contextual memory, and fluency (Pennington & Ozonoff, 1996). The concept of

executive function is characterized by “context-specific action selection, especially in the face of strongly competing, but context-inappropriate, responses,” and “maximal constraint satisfaction in action selection, which requires the integration of constraints from a variety of other domains, such as perception, memory, affect and motivation” (Pennington & Ozonoff, 1996, p. 55).

Impaired executive functioning is reported in ADHD. Significantly, children with ADHD exhibit more inferior performance than do healthy controls on one or more tasks measuring the above-mentioned executive functions (Pennington & Ozonoff, 1996). Gawrilow, Gollwitzer, and Oettingen (2011), using a modified version of the Wisconsin Card Sorting Test (WCST), found that children with ADHD had higher perseverative errors, which were strongly associated with poorer set-shifting performance, than did children without ADHD. In a typical WCST, participants are instructed to match a series of cards to stimulus cards according to three categories (i.e., color, form, and number). The examiner only tells them if the card has been placed correctly or not, but they have to infer the sorting strategy from the feedback offered. Once 10 consecutive cards have been sorted correctly, the sorting principle is changed, unbeknownst to participants. Derived from the WCST is a measure of preservation, which is obtained by counting the number of times a participant adheres to a previously correct rule despite the negative feedback provided by the examiner (Heaton, Chelune, Talley, Kay, & Curtiss, 1993). The WCST preservation has proven to be the most widely-used and sensitive variable for investigating executive function deficits (Greve, Stickle, Love, Bianchini, & Stanford, 2005).

Numerous studies with tasks tapping different executive functions (e.g., working memory, and inhibition) demonstrated that healthy controls displayed better performance than individuals with ADHD (e.g., Gawrilow & Gollwitzer, 2008; Gawrilow et al., 2011; Paul et al., 2007), which then led to the speculation that ADHD may have a mix of specific

and general deficits: a core executive function deficit (probably in inhibition), and another cognitive inefficiency. This conjecture, however, requires future studies to dissect executive function deficits and investigate their specificity in ADHD (Pennington & Ozonoff, 1996).

Deficits in motivation. Children with ADHD prefer to choose an immediate small reward rather than a large one after an interval of time. Conducting two experiments in one study, Sonuga-Barke, Taylor, Sembi, and Smith (1992) demonstrated that children with ADHD and controls equally preferred the large reward during the *no post delay (i.e., the next choice comes immediately after the reward is delivered)* and *post delay (i.e., the next choice appears a period of time after the reward is delivered) conditions* in Experiment 1. In Experiment 2, neither group differed in percentage in choosing the large reward under the *time constraint condition (i.e., children are instructed to gain more points during a certain period of time),* but children with ADHD tended to choose the large reward less frequently than did healthy children under *the trail constraint condition (i.e., children are instructed to gain more points in a limited number of choices).* These results suggest that children with ADHD are delay-averse, resulting in their choosing rewards in a way that minimizes the overall delay.

Furthermore, a more elaborate model of ADHD is proposed to describe a distinction between executive function and motivation control deficits in children with ADHD. According to the dual pathway model of behavior and cognition introduced by Sonuga-Barke (2002), ADHD may not only pertain to a dysregulation of thought and action pathway (DTAP), but also to a motivational-style pathway (MSP). Both children with ADHD DTAP and ADHD MSP meet criteria for the ADHD-combined subtype even though they are characterized by distinct symptoms, development, etiology, and cognitive profiles, as described below.

The first pathway (ADHD DTAP) is manifested in a primary inhibitory dysfunction that is mediated by secondary cognitive and behavioral dysfunctions, which in turn lead to faulty task-engagement (e.g., deficits of set-shifting, working memory) and to symptomatic behaviors (e.g., inattentiveness, hyperactivity). ADHD DTAP seems further etiologically caused by neurobiological risk factors. Indeed, several studies and meta-analyses observed severe cognitive impairments, executive function deficits, and especially inhibition deficits in children with ADHD (Oosterlaan, Logan, & Sergeant, 1998; Oosterlaan & Sergeant, 1996). The second pathway (ADHD MSP) is characterized by a dysregulation of reward mechanisms, leading to a higher preference for immediate rewards by children with ADHD. As associative learning plays an important role in the development of ADHD MSP, it is linked to environmental instead of neurobiological risk factors. ADHD MSP relates empirically to researchers observing delay-aversion and delay-of-gratification deficits in children with ADHD (Sonuga-Barke et al., 1992; Sonuga-Barke, Williams, Hall, & Saxton, 1996).

Deficits in behavioral inhibition. A number of prominent theories emphasizing disinhibition as the core deficit have emerged to elucidate the inappropriate behaviors observed in children and adolescents with ADHD (e.g., Barkley, 1997; Quay, 1997). Built on rigorous evidence from behavioral and neuropsychological studies, two biologically-based systems are hypothesized in Quay's (1997) theory: the behavior activation system (BAS) and behavior inhibition system (BIS). The BAS is sensitive to conditioned stimuli for reward, while the BIS responds to those for punishment and non-reward, leading individuals to passively avoid them and withhold responses. The BIS, however, is underactive in those with ADHD; thus they fail to inhibit their behaviors efficiently.

Barkley (1997) proposes a neuropsychological model of executive functions by integrating the concepts of executive functions, inhibition, and self-regulation to account for

clinical symptoms of children with ADHD—specifically, those with combined subtype or predominantly hyperactive/impulsive subtype (American Psychiatric Association, 2000). According to this model, ADHD primarily arises from a deficit in behavioral inhibition, which refers to three abilities: (a) inhibition of prepotent responses to events, (b) ceasing of ongoing responses, and (c) the protection of responses during the delay from being interrupted by irrelevant events (interference control).

Deficient behavioral inhibition leads to secondary impairments in four executive functions sub-served by the prefrontal lobe, including working memory, self-regulation of affect/motivation/arousal, internalization of speech, and reconstitution. To be executed efficiently, these four executive functions are dependent upon behavioral inhibition. Both primary and secondary deficits have direct or indirect impacts on the development of motor control. Abnormal executive functioning and motor control then develop to dysfunctional behaviors associated with ADHD (see Figure 2.2).

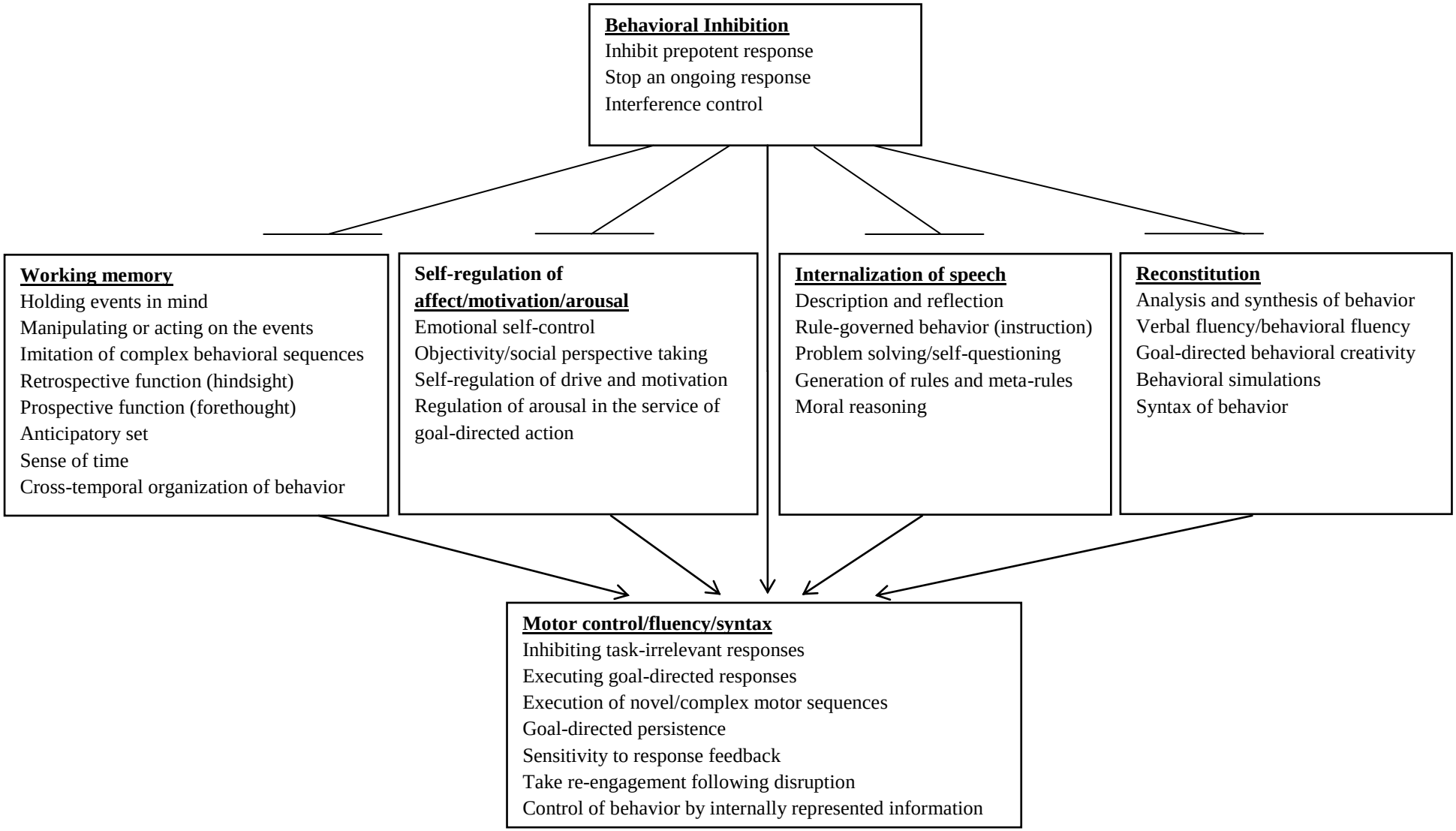

Figure 2.2 Barkley's (1997) neuropsychological model of executive functions. Figure adapted from Barkley (1997).

2.3.5 Psychosocial Factors

As mentioned earlier, psychosocial factors may not be the direct causes of ADHD. However, they have an important impact on the prognosis of this disorder and the development of comorbid disorders (Milberger et al., 1997). A variety of psychosocial risk factors may be influential in comorbid disorders and functional impairments in ADHD (Deault, 2010). For example, among children with ADHD, a lack of positive parenting, parental psychopathology, parenting stress and family conflict, and less social support are associated with comorbid externalizing disorders (e.g., oppositional defiant disorder and conduct disorder). Maternal anxiety and depression, and inconsistent parenting styles, are risk factors for the occurrence of comorbid internalizing disorders (e.g., anxiety and depression); better parenting skills are predictive of later positive outcomes regarding children's academic achievement (Deault, 2010).

Compared with parents of healthy children, those of children with hyperactivity exhibit poorer parenting and greater use of harsh and aggressive discipline methods toward their child—even after the confounding effects of child conduct problems and parental mental health are considered (Woodward, Taylor, & Dowdney, 1998). Early positive parenting, particularly maternal responsiveness, serves as a protective factor against the development course of conduct problems, aggression, and oppositional defiant behaviors, while maternal depression is predictive of the occurrence of conduct problems in young children with ADHD (Chronis et al., 2007; Seipp & Johnston, 2005). Maternal negativity is also a predictive factor of a child's stealing (Anderson, Hinshaw, & Simmel, 1994).

Parent-child interactions are associated with externalizing behaviors that frequently accompany children with ADHD. For example, boys who have a more negative interaction with their mothers are more likely to engage in externalizing behaviors. Similarly, with a Taiwanese sample of 375 medicated children with ADHD and 750 healthy controls, Gau

(2007) demonstrated that compared with families of controls, those of children with ADHD had more inappropriate parenting strategies, impaired family relationships, and increased parent–child conflicts. Mothers of children with ADHD reported that they had less parental care and were more overprotective toward their children. Findings from this study (Gau, 2007) showed that children with ADHD and their families had difficulties interacting with each other, even though they were undergoing treatment. These findings, therefore, suggest that the parental approach should be incorporated in the medication treatment of ADHD.

Taken together, although the current literature is supportive of the view that genetic and neurobiological factors play a critical role in the cause of ADHD, environmental and psychosocial factors are influential in the development of ADHD associated comorbid disorders and functioning impairments (Milberger et al., 1997).

3. Response Inhibition in Attention Deficit Hyperactivity Disorder

According to Barkley's (1997) model, behavioral inhibition refers to three interrelated processes: (a) inhibition of prepotent responses to events (motor response inhibition), (b) ceasing of ongoing responses, and (c) the protection of responses during the delay from being interrupted by irrelevant events (interference inhibition). For the issue of interest and the purpose of brevity, however, the current research is narrowly focused on one process, which is inhibiting prepotent responses to events. Therefore, in the sections that follow, the terms *inhibition*, *response inhibition*, and *inhibitory control* are used interchangeably to refer to this process. We mainly focus on studies of inhibition measured by the Go/NoGo or stop task—but do not focus on those studies using Stroop, Simon, or Flanker task, as these two groups of inhibition tasks are considered to tap into different cognitive constructs of inhibition (Hart et al., 2013), which are motor response inhibition (i.e., measured by the Go/NoGo or stop task) and interference inhibition (i.e., measured by Stroop, Simon, or Flanker task), respectively.

3.1 Behavioral Correlates of Response Inhibition

Impaired inhibitory control causes individuals with ADHD to perform more poorly than controls on tasks that require inhibition. Examples include the stop signal task (SST) (Pliszka, Borcherding, Spratley, Leon, & Irick, 1997; Pliszka, Liotti, & Woldorff, 2000; Rubia, Oosterlaan, Sergeant, Brandeis, & v. Leeuwen, 1998), Go/NoGo task (Paul-Jordanov et al., 2010), and the Continuous Performance Task (CPT), a version of the Go/NoGo task (Banaschewski et al., 2004; Doehnert, Brandeis, Imhof, Drechsler, & Steinhausen, 2010; Fallgatter et al., 2004; Seifert, Scheuerpflug, Zillessen, Fallgatter, & Warnke, 2003; Valko et al., 2009). Studies employing neuropsychological techniques support the literature by demonstrating abnormal activation in the prefrontal lobe and surrounding brain areas such as the cingulate cortex, where the inhibitory process is majorly governed (Bush, 2011; Bush

et al., 1999; Cubillo, Halari, Smith, Taylor, & Rubia, 2012; Pliszka et al., 2000; Rubia et al., 1999).

3.1.1 Stop Signal Task

The SST is a reliable measure of response inhibition across the lifespan (Williams, Ponesse, Schachar, Logan, & Tannock, 1999). It has been extensively used with ADHD populations (e.g., MacLaren, Taukulis, & Best, 2007; Pliszka et al., 1997; Pliszka et al., 2000). In a typical SST, there are a proportion of the Go stimuli (e.g., 25% of the total trials), followed by stop signals with randomized intervals (Logan, Cowan, & Davis, 1984; Pliszka et al., 1997). Participants are instructed to respond to Go stimuli by pressing corresponding buttons but to withhold their already-initiated responses when stop signals are presented. According to the Horse-Race Model (Logan et al., 1984), the probability of successful inhibitions is dependent on a race between the mean reaction time to the Go stimulus (MRT; the Go process) and the Stop Signal Reaction Time (SSRT; the Stop process), which is assumed to be an index reflecting an internal attempt to inhibit the response to stop signals.

The SSRT is estimated by subtracting the delay between the Go stimulus and the stop signal (stop signal delay; SSD) from the MRT (i.e., SSRT = MRT – SSD). When the Go process finishes earlier than the Stop process (i.e., MRT < SSRT + SSD), the response is executed, resulting in a failed inhibition. In contrast, the response is successfully inhibited when the Stop process finishes before the Go process (i.e., MRT > SSRT + SSD). It is noted, however, that the skewedness of the reaction time (RT) distribution and gradual slowing response latencies may bias the SSRT estimates (Verbruggen, Chambers, & Logan, 2013).

People who are more impulsive have longer SSRT than healthy controls (Logan, Schachar, & Tannock, 1997). Meta-analytic reviews (Alderson, Rapport, & Kofler, 2007; Lijffijt, Kenemans, Verbaten, & van Engeland, 2005) showed that children with ADHD had slower and more variable mean reaction times to Go stimuli than did healthy controls.

According to Alderson et al. (2007), effect size estimates were 0.45 for MRT and 0.72 for SDRT. Similarly, the SSRT was found to be significantly longer for the ADHD group than for the control group (mean effect size = 0.61; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). These substantial findings point to abnormalities in response inhibition among children with ADHD.

3.1.2 Go/NoGo Task

Inhibition is also assessed with the Go/NoGo task (Broyd et al., 2005; Groom et al., 2008; Groom, Scerif, et al., 2010; Johnstone, Barry, Markovska, Dimoska, & Clarke, 2009; J. L. Smith, Johnstone, & Barry, 2004). This is a task requiring a response to a type of stimulus (the Go stimulus; e.g., letters except X) and the withdrawal of a prepared but not yet initiated response to another type of stimulus (the NoGo stimulus; e.g., X).

Compared to healthy controls, children with ADHD have been observed to display slower and more variable mean RTs (Epstein et al., 2011; Uebel et al., 2010) and exhibit more omission (i.e., people do not respond to Go stimuli to which they are required to respond) and commission errors (i.e., false alarm: an error occurring when people respond to NoGo stimuli to which they are not supposed to respond) (Broyd et al., 2005; Paul-Jordanov et al., 2010). Several factors may modulate the behavioral performance of children with ADHD (e.g., mean RT, RT variability, and commission errors) on the Go/NoGo task (Epstein et al., 2011; Uebel et al., 2010). For example, children with ADHD tend to respond more quickly and accurately and less variably when rewarded immediately (Uebel et al., 2010). A meta-analytic study (Metin, Roeyers, Wiersema, van der Meere, & Sonuga-Barke, 2012) showed that the rate at which stimuli were presented (i.e., event rate [ER]) had an impact on mean RT (MRT) and errors of commission (EOC). Shorter MRTs and more EOCs with increasing ERs were observed both in the ADHD and control groups. However, the difference between the variables with slow ER and fast ER in the ADHD group was

disproportionate to that of healthy controls, resulting in significant group by event rate interactions (MRT: effect size [ES] between 0.22 and 0.26, *p* between .004 and .003; EOC: ES between -0.17 and -0.18, *p* between .006 and .001). No significance on the effect of ER on RT variability was observed in this meta-analysis (*p* for ES $_{\text{group x event rate}}$ between 0.03 and 0.11).

3.1.3 Mechanisms Underlying the Go/NoGo and Stop Signal Tasks

Although the Go/NoGo task and SST are used interchangeably in measuring inhibition in ADHD, behavioral and neurological substrates for the two tasks may be distinct. According to Schachar et al. (2007), two forms of response inhibition are distinguished: action restraint and action cancellation. Action restraint is used to describe that inhibition of a pre-planned motor response occurs before the response has been initiated, while action cancellation describes that inhibition occurs while the motor response is executed. Action restraint and action cancellation refer to the inhibition measured with the Go/NoGo task and SST, respectively.

The amount of time required for the inhibition of a motor response also differs between the two tasks. In the Go/NoGo task, the time in processing the NoGo stimulus includes response selection and response inhibition; in the stop task, participants withdraw their responses as quickly as they can upon the occurrence of the stop stimulus. They do so without selecting responses, as the stop stimulus occurs after the go process is already initiated (Rubia et al., 2001). Several brain regions are commonly activated in both tasks. However, the regions tend to be activated bilaterally for the Go/NoGo task and are more right-hemisphere-dominant for the stop task (Rubia et al., 2001). Moreover, with respect to the pharmacology of inhibition, serotonin plays a critical role in action restraint during the Go/NoGo task, while action cancellation for the stop task is related to noradrenaline (Eagle, Bari, & Robbins, 2008).

3.2 Electrophysiological Correlates of Response Inhibition

Over the past decade, considerable studies have been conducted to obtain electrophysiological data when participants with or without ADHD are performing inhibition-related tasks (especially with the visual/auditory stop tasks) to enhance the understanding on the pathophysiology of ADHD and the neural mechanisms underlying inhibitory control in ADHD (Albrecht, Banaschewski, Brandeis, Heinrich, & Rothenberger, 2005; Dimoska, Johnstone, Barry, & Clarke, 2003; Johnstone, Barry, & Clarke, 2013; Johnstone et al., 2007; Liotti, Pliszka, Higgins, Perez, & Semrud-Clikeman, 2010; Liotti et al., 2007; Overtoom et al., 2002; Senderecka, Grabowska, Szewczyk, Gerc, & Chmylak, 2012; Shen, Tsai, & Duann, 2011).

3.2.1 Stimulus-Related Event-Related Potentials

In event-related potential (ERP) studies (e.g., Broyd et al., 2005; Pliszka et al., 2007), continuous electroencephalogram (EEG) data are collected when the participant is performing a cognitive task in which different types of stimuli/trials are repeatedly presented. Recorded EEG data are then averaged across many trials. This aims to cancel out the noise that may result from random brain activity, physiological signals such as electrooculography (EOG) and electrocardiography (EKG), as well as interference from the external environment (e.g., electronic noise). After that, a waveform is produced, representing brain activity in response to a type of stimulus in the task. Typically, two stimulus-locked ERP components—N2 and P3—are elicited when response inhibition tasks are administered. Both N2 and P3 are stable and heritable electrophysiological markers involved in the inhibitory process (Dimoska et al., 2003; Falkenstein, Hoormann, & Hohnsbein, 1999; Jodo & Kayama, 1992; McLoughlin et al., 2009; Overtoom et al., 2002; Overtoom, Verbaten, Kemner, Kenemans, van Engeland, et al., 1998; Pliszka et al., 2000; Seifert et al., 2003).

The N2 component. The N2, which is associated with conflict monitoring, is a negative-going shift with a peak at approximately 200 ms after stimulus onset. Functional responses of the N2 are mostly interpreted as the initiation of inhibitory processing. However, some studies adopting auditory stop task paradigms may complicate the inhibitory hypothesis by reporting the absence of the N2 effect in healthy controls (Falkenstein et al., 1999) or in the ADHD group (Fisher, Aharon-Peretz, & Pratt, 2011). One study (Dimoska et al., 2003) suggests that the N2 may be an indicator of the activation level of the inhibitory process. In accordance with the former view (i.e., the N2 represents the initiation of inhibition), amplitudes of the NoGo N2 are found to be larger over frontal-central brain regions than those of the Go N2. This indicates that inhibitory processing is manipulated to withhold an ongoing but inappropriate response in NoGo trials (Broyd et al., 2005; Falkenstein et al., 1999; Pliszka et al., 2007). With respect to the NoGo N2, Falkenstein et al. (1999) found in a Go/NoGo task that, relative to healthy controls, children with ADHD displayed a significant correlation between reduced N2 amplitudes for NoGo stimuli and higher commission errors, indicating a weakness in successfully inhibiting responses to NoGo stimuli in ADHD. Moreover, in the SST, Pliszka et al. (2007) demonstrated that children with ADHD exhibited a strongly diminished NoGo N2 in the right frontal lobe, a brain region found to be impaired in the inhibitory process (Rubia et al., 1999; Rubia, Smith, Brammer, Toone, & Taylor, 2005). This corresponds to the observation that inhibition is executed with a right-hemisphere dominance (Garavan, Ross, & Stein, 1999).

The P3 component. Following the N2, the P3 deflects positively, peaking at around 300 ms after stimulus onset. This component is closely linked to selective attention and allocation of efforts (Picton, 1992). In Go/NoGo studies, greater amplitudes and longer latencies are observed in NoGo trials than in Go trials, indicating greater demand on

resources in processing NoGo trials (Fallgatter & Strik, 1999; Overtoom, Verbaten, Kemner, Kenemans, van Engeland, et al., 1998; Picton, 1992; Pliszka et al., 2007). The NoGo P3 is observed to be diminished in ADHD, which results in P3 amplitude differences between NoGo and Go trials being significantly smaller in children with ADHD relative to normally developing children (Paul-Jordanov et al., 2010) and reflects poor attention and impaired response control in the ADHD group.

The NoGo P3 has higher frontal-central/anterior topographical distribution than does the Go P3 (Bokura, Yamaguchi, & Kobayashi, 2001; Fallgatter et al., 2004; Picton, 1992; Tekok-Kilic, Shucard, & Shucard, 2001). Distinct scalp distributions suggest that different neural cortical generators underlie the Go and NoGo P3 (Tekok-Kilic et al., 2001). The Go P3 is recorded maximally at parietal electrode sites (Fallgatter et al., 2004). The topographical distribution of the NoGo P3 is accounted for by dipoles located in the anterior cingulate cortex (ACC) (Paul et al., 2007), a central brain area consistently reported to be dysfunctional in the ADHD group from electrophysiological and neuroimaging studies (Bush et al., 1999; Fallgatter et al., 2004).

Similarly, studies using other paradigm tasks found abnormal P3 response in ADHD. For instance, Strandburg et al. (1996) found reduced and delayed visual P3, and diminished late frontal negativity but normal neuronal activity related to the early stages of stimulus processing in children with ADHD. This suggests deficits in post-decisional processing. Jonkman et al. (1997) reported that children with ADHD were more likely than healthy controls to exhibit smaller P3 amplitudes toward non-target stimuli, both in auditory and visually selective attention tasks. They hypothesized that the activation of the P3 process was impaired in ADHD. Likewise, Overtoom et al. (1998) reported that significantly attenuated P3 amplitudes toward targets were observed in the ADHD group during a visual continuous performance task as compared to normal controls, while no group significance

regarding amplitudes of the inhibition-related negative waves existed. This indicates a deficit in attention but not in impulsivity (or inhibition).

Prox, Dietrich, Zhang, Emrich, and Ohlmeier (2007) demonstrated that the P3 had a link with decreased activity in adults with ADHD. Together with other findings that early ERP components related to attention mechanisms were enhanced in ADHD, Prox et al. (2007) suggested that adults with ADHD might manage to achieve the same task performance as controls and compensate for their deficits by paying more attention than healthy adults. Using an oddball auditory task, Itagaki et al. (2011) found that the P3 was significantly decreased in amplitude, but not prolonged in latency, among ADHD adults relative to the control group.

Associations between age and the N2 and P3. Differential response patterns of the N2 and P3 components on inhibition are modulated by age difference (Johnstone, Pleffer, Barry, Clarke, & Smith, 2005). For instance, using the auditory Go/NoGo task, Johnstone et al. (2005) found that in healthy subjects, the N2 and P3 increased in amplitude and decreased in latency across developmental ages of childhood, and in young and older adults. The effect of the NoGo N2 being larger than the Go N2 in amplitude—particularly over the frontal scalp—diminished with age, with the largest and most reduced effects observed in children and young adults, respectively. However, a minor reversed difference in older adults was found, in contrast to the previous finding that no significant age effect existed on the NoGo N2 with visual Go/NoGo tasks (e.g., Jonkman, Lansbergen, & Stauder, 2003). Other studies found that this effect was only restricted to the younger children in the ADHD group, while a Go > NoGo N2 effect was observed in older children with ADHD (e.g., Broyd et al., 2005).

Taken together, these mixed findings may indicate that differential mechanisms in processing stimuli are adopted between age groups or between ADHD and control groups

(Broyd et al., 2005). For the P3, the NoGo P3 has a more anterior scalp distribution relative to the Go P3 across age in the control and ADHD groups (Broyd et al., 2005; Falkenstein et al., 1999). The NoGo P3 effect is more pronounced in the older age group than the younger age group, both in visual and auditory modality (Broyd et al., 2005; Johnstone et al., 2005; Jonkman et al., 2003).

Some researchers attribute their results to a general deficit in attention rather than inhibition; they found that a significant diminished P3 exists in the adult ADHD group in comparison to healthy controls (Itagaki et al., 2011; Prox et al., 2007; Rodriguez & Baylis, 2007; Sawaki & Katayama, 2006). However, a number of studies with different tasks and methodologies tested on adults with ADHD have substantially demonstrated that the child and adolescent ADHD patterns characterizing deficient inhibitory control persist into adulthood, implying that impaired inhibition may be still critical to clinical ADHD symptomatology (Aron, Dowson, Sahakian, & Robbins, 2003; Boonstra, Kooij, Oosterlaan, Sergeant, & Buitelaar, 2005, 2010; Epstein, Johnson, Varia, & Conners, 2001; Fallgatter et al., 2005; Lijffijt et al., 2005; MacLaren et al., 2007; Murphy, 2002; Nigg, Butler, Huang-Pollock, & Henderson, 2002; Ossmann & Mulligan, 2003; Schneider et al., 2007; Wodushek & Neumann, 2003).

For example, regarding the behavioral performance on the stop task, a meta-analysis of 29 studies (Lijffijt et al., 2005) revealed that the indices of deficient inhibitory control, such as the prolongation of SSRT, existed significantly in the ADHD group relative to controls robustly across the groups' lifespan. On measures of inattention, such as longer RTs and variable standard deviation RT (i.e., SDRT), a pronounced difference between groups was observed among the child population (see also Alderson et al., 2007), but not seen in the adult population. Within the adult ADHD group, SSRT can vary according to the level of ADHD symptoms. Individuals with high levels of ADHD symptoms, who are

supposed to experience more difficulties inhibiting their responses, are reported to have longer SSRT than those with low levels of ADHD symptoms (Wodushek & Neumann, 2003).

From the perspective of electrophysiology, one study (Valko et al., 2009) showed that the inhibition-related component (i.e., NoGo P3) was attenuated in children and adults with ADHD relative to their healthy counterparts; impacts yielded by ADHD symptoms on electrophysiological performance were evident despite developmental effects. Another study (MacLaren et al., 2007) applying the stop task also demonstrated a diminished N2/P3 complex in adults with ADHD in the absence of a significant difference on the behavioral measures.

Inconsistent with the notion of deficient inhibitory control in ADHD, one study (Wiersema, van der Meere, Antrop, & Roeyers, 2006) adopted the Go/NoGo paradigm and found no pronounced group differences in terms of the N2 response. As noted by Wiersema et al. (2006), however, this study may be limited by its relatively small size. Meanwhile, some potential confounding variables that may lead to inconsistent results/conclusions need to be taken into serious consideration—for example, the level of the task demand and the composition of the participant population.

Associations between cognitive functioning and the P3. In addition to age effect, cognitive functioning is associated with the P3 response (e.g., Dichter, van der Stelt, Boch, & Belger, 2006; O'Donnell, Friedman, Swearer, & Drachman, 1992; Pelosi et al., 1992; van der Stelt, Frye, Lieberman, & Belger, 2004). However, evidence from the ADHD group is more limited compared to that from the nonclinical and schizophrenia samples. Investigations of correlations between neuropsychological functioning (e.g., intelligence and working memory) and the P3 response have been reported with component latencies and amplitudes among clinical patients (Dichter et al., 2006; Egan et al., 1994; van der Stelt

et al., 2004) or healthy individuals (O'Donnell et al., 1992; Pelosi et al., 1992). Several lines of evidence have indicated that neuropsychological functioning is associated positively with P3 amplitude, but negatively with P3 latency (e.g., Dichter et al., 2006; Jaušovec & Jaušovec, 2000; Polich, Howard, & Starr, 1983). For example, P3 latency was reported to be inversely correlated with digit-span performance in normal individuals. As digit-span performance was considered as a measure of working memory, relatively short P3 latencies were hypothesized to reflect decreased memory function (Polich et al., 1983).

Walhovd and Fjell (2002) found that the P3 latency-digit span correlation was weaker for backward spans than for forward spans and total spans, while the P3 amplitude-digit span correlation was not dependent on the type of digit span. Additionally, with auditory and visual oddball task, Jaušovec and Jaušovec (2000) demonstrated that highly intelligent people were more likely than those with less intelligence to display reduced P3 latencies and increased P3 amplitudes. Given that the P3 amplitude reflects cognitive resources allocated to process the stimuli (Kok, 2001) and the P3 latency indexes the time of stimulus evaluation (Kutas, McCarthy, & Donchin, 1977), Jaušovec and Jaušovec (2000) suggested a simultaneous activation of more specific neural networks in highly intelligent individuals during the cognitive tasks. Similarly, O'Donnell et al. (1992) reported a significant negative relationship of P3 latency with intelligence (-0.44) and with concentration (-0.33).

After controlling for measures of executive functioning, Dichter et al. (2006) found a positive relationship between the estimated intelligence score and P3 amplitude in the control group, but a negative relationship in schizophrenic patients. They demonstrated that shorter P3 latencies were associated with higher estimated intelligence in healthy controls, but not in the schizophrenia group. These findings suggest that the association between P3 response and intelligence may differ between diagnostic groups, indicating that measures of

executive functioning should be included in investigations aimed at exploring the P3-intelligence relationship.

3.2.2 Failed Response Inhibition-Related Event-Related Potentials

Two error-related components have received increasing attention and have been reported in several inhibition-related studies using the flanker task (Herrmann et al., 2010), and the Go/NoGo task (Groom, Cahill, et al., 2010; O'Connell et al., 2009; Wiersema, van der Meere, & Roeyers, 2009) or the SST (Liotti, Pliszka, Perez, Kothmann, & Woldorff, 2005; Senderecka et al., 2012; Shen et al., 2011). After the onset of incorrect button presses, the error-related negativity (ERN), a negative-going component with a frontal-central scalp distribution, is evoked at approximately 100 ms, while the error positivity (Pe) peaks at 300 ms over the central-parietal scalp (Senderecka et al., 2012). Both components, which appear to be generated in the ACC, represent different aspects associated with error processing (Garavan, Ross, Murphy, Roche, & Stein, 2002). More specifically, the ERN may be elicited unconsciously and reflect the detection of an error resulting from a conflict between the intended and actual responses; the Pe occurs only when the participant is aware of an erroneous response (Shen et al., 2011; Wiersema et al., 2009).

Deficits in error processing have been reported in ADHD children (Senderecka et al., 2012; Shen et al., 2011), adolescents (Groom, Cahill, et al., 2010), and adults (O'Connell et al., 2009; Wiersema et al., 2009), which may partially be associated with poor performance observed during inhibition tasks. Inconsistent findings on the ERN amplitude are evident in a number of studies. Studies using the SST either in visual (Liotti et al., 2005) or auditory modality (Senderecka et al., 2012) demonstrated that the ERN was markedly diminished in the ADHD group relative to healthy children. Some studies found that larger ERNs were observed in the ADHD group (Burgio-Murphy et al., 2007); other studies with the Go/NoGo task found no significant group differences on the ERN in child (Wiersema, van

der Meere, & Roeyers, 2005) and adult populations (Wiersema et al., 2009). By contrast, the Pe is found to be consistently smaller in the ADHD group than in controls regardless of developmental age (Groom, Cahill, et al., 2010; O'Connell et al., 2009; Senderecka et al., 2012; Wiersema et al., 2005; Zhang, Wang, Cai, & Yan, 2009). Therefore, these findings imply that—rather than error detection—abnormal neural activity in the late stage of error processing (i.e., conscious evaluation of an error), which is indexed by reduced Pe, appears to serve as a lifelong trait marker in ADHD (Wiersema et al., 2009; Zhang et al., 2009).

3.3 Evidence from Neuroimaging Studies on Response Inhibition

The right inferior prefrontal cortex serves as the crucial brain region for response inhibition. The mesial frontal cortex, including the anterior cingulate cortex and bilateral inferior parietal lobes, is responsible for error detection or error monitoring (Rodrigo et al., 2014; Rubia, Smith, Brammer, & Taylor, 2003). Abnormal brain activity in these regions has been reported in individuals with ADHD across their lifespans (Cubillo et al., 2010; A. Smith, Taylor, Brammer, Toone, & Rubia, 2006), and certain brain dysfunction (i.e., the right inferior prefrontal cortex) is reportedly disorder-specific to ADHD (Rubia et al., 2010).

However, findings from neuroimaging or event-related potential studies are mixed. In a functional magnetic resonance imaging (fMRI) study with the SST, Pliszka et al. (2006) found that children with ADHD were more likely than healthy controls to show decreased activation in the anterior cingulate cortex and left ventrolateral prefrontal cortex for unsuccessful inhibitions, but increased activation in the right inferior performance cortex for successful inhibitions. The finding of enhanced right inferior prefrontal cortex activation was in line with earlier reports of increased activation of the right ventrolateral prefrontal cortex in adolescents with childhood ADHD during the Go/NoGo task (Schulz et al., 2004). However, they were inconsistent with a previous study (Rubia et al., 2005) that found reduced activation in the right inferior prefrontal cortex in medication-naïve adolescents

with ADHD performing the stop task. These discrepant results could be explained by different experimental designs or techniques in the studies (Pliszka et al., 2006).

A recent meta-analysis of fMRI studies (Hart et al., 2013) showed that individuals with ADHD, compared to controls, exhibited significantly reduced activation in the right inferior frontal cortex (IFG) and insula, the right supplemental motor area (SMA) and ACC, the right thalamus, left caudate, and the right occipital lobe. Moreover, studies found that frontal-striatal and parietal dysfunction during motor inhibition, which were observed in children (Rubia et al., 2010), adolescents (A. Smith et al., 2006), and adults (Cubillo et al., 2010) with ADHD, were unrelated to long-term stimulant medication exposure (Pliszka et al., 2006; A. Smith et al., 2006). These dysfunction similarities between age groups may therefore suggest a continuation of frontostriatal inhibitory network deficits from childhood to young adulthood in ADHD (Cubillo et al., 2010).

Furthermore, Cubillo et al. (2010) observed reduced functional interconnectivity between critical brain areas involving inhibition (i.e., between the right inferior prefrontal cortex and other dysfunction regions such as left inferior prefrontal cortex, striatum, parietal lobe, and anterior and posterior cingulate), indicating that the dysfunctions in ADHD may affect not only isolated brain regions but also the inter-regional connectivity between these affected regions. This finding and evidence from other studies (e.g., Shang, Wu, Gau, & Tseng, 2013; Wolf et al., 2009) extend the functional deficit findings on motor inhibitory control in ADHD to the neural network level.

3.4 Brief Summary

Taken together, evidence from behavioral, event-related potential, or neuroimaging studies supports that individuals with ADHD are impaired in inhibitory control to some degree. Although altered response patterns of the stimulus-related N2 and P3, and error-related ERN/Pe during the inhibitory processing (e.g., amplitudes, latencies, and topographical distributions) have been consistently demonstrated in ADHD, findings on the electrophysiological performances of adults with ADHD are relatively mixed due to limited literature in comparison with those on children and adolescents with ADHD. As a result, future studies with different paradigms are needed to confirm the previous findings and to further characterize the electrophysiological patterns and facilitate clear investigations in light of inhibition and pathophysiology in ADHD across the human lifespan.

4. Effects of Medication and Self-Regulation Strategies on Response Inhibition

4.1 Medication

Stimulant treatment. Methylphenidate (MPH), a central nervous system (CNS) stimulant, has been widely discussed in studies on the treatment of ADHD (e.g., Broyd et al., 2005; Goldman, Genel, Bezman, & Slanetz, 1998; Paul-Jordanov et al., 2010). Two formulations of MPH are the most commonly prescribed medications in clinical practice: immediate-release-MPH (IR-MPH) for thrice-daily administration, and long-acting osmotic-release oral system-MPH (OROS-MPH) for once-daily administration. OROS-MPH is found to have better treatment effects relative to IR-MPH (Gau, Shen, Soong, & Gau, 2006), as individuals with ADHD have higher adherence rates to OROS-MPH than IR-MPH (Tzang et al., 2012). In the U.S., the rate of individuals aged 18 years and below using stimulants rose from 0.6% in 1987 to 4.8% in 2002 (Zuvekas, Vitiello, & Norquist, 2006). Approximately 48.6 million daily doses of MPH in Germany were prescribed in 1990; this number increased to 1036 million daily doses in 2000 (Schubert, Selke, Osswald-Huang, Schroeder, & Nink, 2002). In Taiwan, MPH is the first-line medication to treat ADHD. Its immediate-release formulation (i.e., IR-MPH) has been used for decades, and as of October 2003 (Gau et al., 2006), osmotic-release MPH is accepted as an alternative for ADHD treatment. Even though the rates of using stimulant treatment differ in different populations, these trends show that an increasing number of children are treated with psychotropic drugs.

Effects are exerted by MPH on two catecholamine systems: norepinephrine and dopamine systems. Both are critical for the operation of executive functioning but are found to be dysfunctional in ADHD studies on humans (Pliszka et al., 1996) and animals (Grund, Lehmann, Bock, Rothenberger, & Teuchert-Noodt, 2006). MPH restores the levels of norepinephrine and dopamine in synaptic clefts in individuals with ADHD by binding to

corresponding transporters on the presynaptic neuron membrane, and then preventing the reuptake of neurotransmitters in brain areas related to attention or motor control (Jensen, 2009; Scahill, Carroll, & Burke, 2004).

A body of empirical studies and meta-analyses has documented that MPH has short-term or long-term efficacy in improving most clinical symptoms in children with ADHD (e.g., Abikoff et al., 2004; Schachter, Pham, King, Langford, & Moher, 2001; Van der Oord, Prins, Oosterlaan, & Emmelkamp, 2008). This documentation is based not only on subjective ratings by teachers, parents, and psychiatrists (Abikoff et al., 2004; Van der Oord et al., 2008) but also on objective measurements combined with the infrared motion system (Heiser et al., 2004). Similarly, MPH is effective in ameliorating impaired response inhibition measured by inhibition tasks, which is reflected on electrophysiological or behavioral measures and has been demonstrated in child (Tannock, Schachar, Carr, Chajczyk, & Logan, 1989; Tannock, Schachar, Carr, & Logan, 1989), adolescent (Barkley, 2004), and adult (Murphy, 2002) populations. Aron et al. (2003) showed that SSRT was significantly faster in adults between 18 and 41 years of age when off medication than on medication. However, no significant differences were observed between the two medication conditions in terms of reaction times to Go stimuli and choice errors. Overall, individuals with ADHD exhibited more error rates than did their healthy counterparts prior to medication; after the treatment with MPH, the error rates were significantly reduced in the ADHD group (Aron et al., 2003).

Regarding evidence from electrophysiological studies, the NoGo N2 and P3 amplitudes are smaller in the unmedicated ADHD group relative to the control group. But after the treatment of MPH, amplitudes of the N2 and P3 do not differ between groups (Broyd et al., 2005; Paul-Jordanov et al., 2010). Using a stop signal task, Pliszka et al. (2007) demonstrated the differential effects of MPH on the N2 and P3 by comparing

amplitudes of both components in children and adolescents with ADHD when off and on medication. Independent of gender and age, increased NoGo N2 amplitudes were observed after the treatment of MPH over the right inferior frontal lobe on successful inhibition trials, while the enhancements of the ACC-originated NoGo P3 were exclusively limited to failed inhibition trials. This indicates that different brain regions might be responsible for different cognitive functioning (i.e., the right frontal lobe for inhibition, while ACC for attention re-allocation after an error occurs) during the inhibitory process (Pliszka et al., 2007). Neuroimaging studies showed that MPH enhances the activation of the right inferior frontal cortex (IFG)/insula, brain regions which are critical for inhibitory control but dysfunctional in ADHD (Rubia et al., 2014). MPH ameliorates response inhibition by exerting its effect on attention networks associated with response control requirements (Pauls et al., 2012).

Nonstimulant treatment. Another alternative medication treatment used for ADHD is nonstimulant medication such as atomoxetine. Atomoxetine is a selective norepinephrine reuptake inhibitor. The efficacy of atomoxetine in the management of ADHD has been reported in clinical studies from different countries, e.g., the U.S. (Kelsey et al., 2004), Russia (Martenyi et al., 2010), Germany (Wehmeier et al., 2012), and Taiwan (Gau et al., 2007). Atomoxetine is effective in reducing not only the core symptoms of ADHD (Gau et al., 2007; Martenyi et al., 2010; Wehmeier et al., 2012) but also oppositional symptoms (Gau et al., 2007; Michelson et al., 2001). For instance, with a large sample of 106 patients, Gau et al. (2007) demonstrated that atomoxetine is an effective, well-tolerated, and safe treatment for children and adolescents with ADHD in Taiwan. In addition, atomoxetine improves inhibitory control in healthy adults (Chamberlain et al., 2009) and adults with ADHD (Chamberlain et al., 2007).

Atomoxetine exerts its beneficial effects on inhibition by modulating right inferior frontal functioning (Chamberlain et al., 2009). In general, the efficacy of atomoxetine is

comparable with that of MPH in the treatment of ADHD (Hanwella, Senanayake, & de Silva, 2011; Hazell et al., 2011). However, some meta-analytic studies point out that OROS-MPH could be more effective than atomoxetine and may be considered as the first-line treatment in children and adolescents with ADHD (Hanwella et al., 2011).

4.2 Self-Regulation Strategies

Recently, a growing number of papers reveal that the self-regulation strategy of implementation intentions has an effective impact regarding the support of inhibition tasks in the ADHD group (Gawrilow & Gollwitzer, 2008; Gawrilow et al., 2011; Paul-Jordanov et al., 2010). An implementation intention (Gollwitzer, 1993, 1999), which is a specific form of a plan, emphasizes an anticipated critical situation (i.e., the if-component) and a goal-directed response (i.e., the then-component). It is important to recognize that implementation intentions differ from goal intentions. Goal intentions merely specify an anticipated outcome and have the format of "I intend to achieve Z." When people form an implementation intention, a mental link is made between the two components ("If situation X arises, then I will initiate goal-directed response Y"). "Implementation intentions are subordinate to goal intentions because, whereas a goal intention specifies *what* one will do, an implementation intention spells out the *when*, *where*, and *how* of what one will do" (Sheeran, Webb, & Gollwitzer, 2006, p. 123).

As forming an if-then plan (see Figure 4.1) implies the selection of a future situation, the mental representation of this situation becomes highly activated, and therefore more easily accessible. Additionally, this critical cue automatically triggers the intended (and pre-planned) response and/or behavior: it is enacted immediately, efficiently, and without conscious intent (e.g., Bayer, Achtziger, Gollwitzer, & Moskowitz, 2009). Both mechanisms—the heightened accessibility of the cue and the automatic activation of the intended behavior—produce a perceptual and behavioral readiness that accounts for if-then

plan effects on goal attainment (e.g., Webb & Sheeran, 2008). In this way, implementation intentions help people initiate their actions more automatically, thereby enhancing the self-regulation of goal striving and the attainment of self-set or assigned task goals effectively. If-then plans have been proven to have benefits above mere goal intentions: A meta-analysis by Gollwitzer and Sheeran (2006) revealed an effect size of d = 0.65; a medium-to-large effect size (Cohen, 1992) representing the additional facilitation size of this effect is remarkable, because goal intentions commonly have a facilitating effect on behavior enactment in and of themselves (Webb & Sheeran, 2006).

A study conducted by Gawrilow and Gollwitzer (2008) showed that children with ADHD benefited from forming if-then plans for a modified Go/NoGo task. This task required participants to classify stimuli that were presented on a computer screen by pressing a pre-specified key on the computer keyboard, and then inhibit this classification in response to a NoGo signal. Children with ADHD were randomly assigned to one of two groups. Children in the goal intention group formed a goal to inhibit a classification response for marked stimuli. Children in the implementation intention group, in addition to forming this goal intention, formed an if-then plan. In their first study, Gawrilow and Gollwitzer (2008) compared the performance of children with and without ADHD. Children without ADHD performed at a high level, no matter whether they had formed a goal intention or an implementation intention. Children with ADHD, however, reached the high performance level only when they had complemented the goal intention with an if-then plan; children with ADHD in the goal intention-only condition showed a significantly lower performance level.

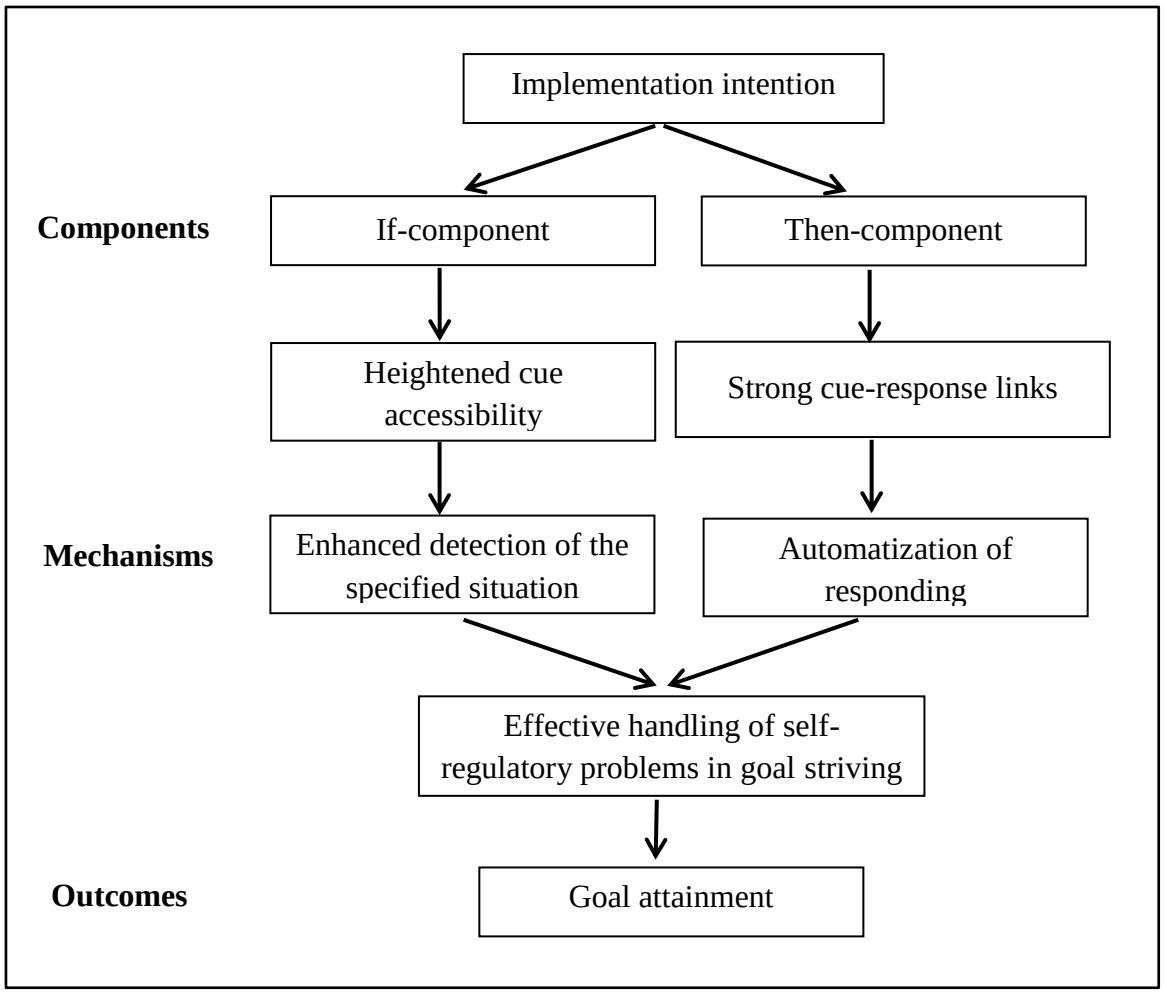

Figure 4.1 Components, mechanisms, and outcomes of implementation intention formation. Figure adapted from Sheeran et al. (2006).

Paul et al. (2007) collected electrophysiological data when children with and without ADHD were performing the Go/NoGo task under two different task instruction conditions: the baseline condition without a self-regulation strategy, and the planning condition with a self-regulation strategy. The children with ADHD went through both conditions, which was the only difference from the previous study (Gawrilow & Gollwitzer, 2008), in which children with ADHD were randomly assigned to either condition. The Go/NoGo task consisted of 300 stimuli (50% colored drawings of transportation vehicles and 50% colored drawings of animals). Children were asked to respond to animals and vehicles by pressing

one of two colored buttons, respectively. The correspondence of stimulus type and response-button was reversed after the first half of the experiment to prevent the task from becoming too easy. In 33% of the trials, a stop sign—a white outspread hand on a circular purple background—was presented before the stimulus. This was actually the only difference to the task used by Gawrilow and Gollwitzer (2008), in which a tone was used as the stop signal. The stop sign indicated that no response was to be given on the following trial.

The trials following stop signs were treated as NoGo trials; all other trials were Go trials. In the baseline condition, children received a neutral instruction containing information regarding how to perform the task (e.g., pressing different buttons corresponding to animals or vehicles and not pressing any buttons whenever the hand was shown). In the planning condition, children were given an instruction furnished with an if-then plan (i.e., if I see a hand, then I will not press a button). This study (Paul et al., 2007) showed that more inhibition errors were found in children with ADHD than controls in the baseline condition. No group difference was significantly revealed in the planning condition. Response errors to Go stimuli did not differ between ADHD and control children in the baseline and planning conditions. Slower reaction times were found to be significant in the ADHD group relative to the control group across both conditions. Moreover, children with ADHD exhibited greater NoGo P3 and NoGo-Go (NoGo minus Go) P3 with the assistance of the self-regulation strategy of if-then plans in comparison to their electrophysiological performance in the baseline condition. This indicates that a better response control was achieved, supported by the high correlation between the successful inhibition and the NoGo-Go P3 amplitude difference (Paul et al., 2007). As the P3 is an ERP component related to selective attention (Picton, 1992) and the NoGo P3 is usually reduced in children with ADHD (e.g., Fallgatter et al., 2004), the self-regulation strategy of if-then plans is

apparently effective for enhancing inhibitory control in children with ADHD (Paul-Jordanov et al., 2010).

More recently, the application of if-then plans has been extended to various settings such as school settings, with promising findings. Guderjahn, Gold, Stadler, and Gawrilow (2013) found that children with ADHD from grades five to ten in a secondary school exhibited improved self-regulatory competencies with the assistance of if-then plans (i.e., the use of goal intention furnished with if-then plans) compared with their performances in the baseline (i.e., the use of goal intention) in everyday school life.

4.3 Brief Summary

To sum up, MPH is effective for improving ADHD symptoms. Careful pharmacological treatment has been demonstrated to be superior to behavioral treatment in reducing ADHD core symptoms. However, combined (i.e., behavioral intervention and stimulant medication) and behavioral treatments relative to medication alone are beneficial in supporting family functioning (The MTA Cooperative Group, 1999a). Research on behavioral intervention, reviewed in this section, provides clinicians another perspective concerning the management of ADHD by demonstrating that forming if-then plans is effective for the behavioral and electrophysiological correlates of response inhibition measured by the Go/NoGo task. Thus, the self-regulation strategy could be an effective behavioral strategy without adverse effects in the support of inhibition in ADHD (Paul-Jordanov et al., 2010).

5. The Current Research

One motive that guides the current research is that developmental patterns are strongly influenced by cultural setting (Cheie, Veraksa, Zinchenko, Gorovaya, & Visu-Petra, 2015; Wanless et al., 2011). Culture, in which individuals are deeply embedded, is an influential factor in shaping how parents educate their children (Keller, 2007) and mediating relationships of parenting with children's developmental functioning (Lansford, et al., 2005). For example, across six countries, Lansford, et al. (2005) demonstrated that mothers' use of physical punishment was associated with children's behavioral problems, but the link was stronger in countries where physical punishment is not viewed as a common rearing strategy. Investigating associations between child's inhibitory control, harsh parental discipline and externalizing problems in preschoolers in the U.S., China, and Japan, Olson et al. (2011) found that a child's externalization problems were pronouncedly associated with harsh maternal discipline and low levels of inhibitory control in all three countries. Moreover, harsh maternal discipline was negatively correlated with a child's inhibitory control, but this was observed only in China and Japan, not in the U.S. (Olson et al., 2011).

Parenting practices may vary across cultures. Generally, in Chinese culture, socially-restrained behaviors are endorsed, while disruptive behaviors are prohibited (Chen, Cen, Li, & He, 2005). Chinese parents have less tolerance of aggressive and disruptive behaviors than Western parents, e.g., Americans, (Entwisle, Alexander, & Olson, 2007), because children's behavioral regulation is highly valued in Chinese society. Also, strong behavioral regulation could contribute to success in children's early academic achievement (Wanless et al., 2011). Similarly, in Taiwanese culture, children are educated to regulate themselves and act in a proper manner, especially when elder family members are around (Hsieh, 2004). Prior research on the socialization of self-regulation found that more aggressive and noncompliant behaviors occurred in German preschoolers than Japanese preschoolers

(Trommsdorff and Kornadt, 2003). Additionally, Asian parents tend to prepare in advance for what their children need before their children tell them, whereas Western parents (i.e., Germans and Americans) prefer to allow their children more independence to cope with a variety of situations (Trommsdorff, Cole, & Heikamp, 2012). Furthermore, parental rearing practices may influence the brain's organization of cognition, resulting in different behavioral performance on neuropsychological measures (Meyer, 2005). Hence, the previous finding of deficient inhibition in ADHD may be constrained by the generalizability to other ethnic groups, because the task to date has been only used in one cultural context (i.e., Germany) (Gawrilow & Gollwitzer, 2008; Paul-Jordanov et al., 2010; Paul et al., 2007).

Thus, the current research aims at assessing behavioral inhibition with the identical assessment and procedure in different cultural settings and further exploring the effect of MPH on inhibition in children with ADHD. To fulfill the aims, we incorporate data and results from prior research (the German sample) with permission (Paul-Jordanov et al., 2010) and add new findings from a sample (the Taiwanese sample) in the present research. Two studies are conducted in the sections that follow. Study 1 compares response inhibition between children with ADHD and healthy controls in Germany and Taiwan. Study 2 further investigates the effect of MPH on response inhibition in children with ADHD at both sites.

6. Study 1: Response Inhibition among Children with and without ADHD in Germany and Taiwan

6.1 Overview of the Study

In Study 1, incorporating the data from the previous study (Paul-Jordanov et al., 2010), we compared the behavioral performance of children with and without ADHD as measured by the Go/NoGo task (Paul-Jordanov et al., 2010) in Germany and Taiwan. We hypothesized that children with ADHD at both sites were more likely than their healthy counterparts to exhibit poor performance on NoGo trials (i.e., they had more difficulties inhibiting their responses on NoGo trials). Additionally, based on the previous research (Broyd et al., 2005; Fallgatter et al., 2004), we predicted that for both sites, children with ADHD would respond to Go trials less accurately and more slowly than would non-ADHD children.

6.2 Method

6.2.1 Participants

Germany. The sample consisted of 11 (all boys) children who were diagnosed with ADHD combined type (American Psychiatric Association, 2000) as their primary disorder, and 16 (including three girls) age-matched healthy children (mean age ± SD: ADHD: 12.4 ± 0.4 years vs. Control: 12.5 ± 0.3 years, respectively). Children participated after giving written informed consent. All of the children with ADHD were taking prescribed medication, while none of the controls had any clinically relevant diagnoses or were taking any medication, according to their parents. The study was approved by the research ethics committee of the University of Konstanz, and is compliant with the World Medical Association Declaration of Helsinki. Children with ADHD were recruited through a collaborating child psychiatric outpatient center in Konstanz. Control children were

contacted through the participant record system of the University of Konstanz (source from Paul-Jordanov et al., 2010).

Taiwan. The sample included 19 children with ADHD (including two girls) and 16 healthy controls (including one girl), aged 8 to 12 (mean age ± SD: ADHD: 9.72 ± 1.14 years vs. Control: 10.12 ± 0.92 years). All participants were required to have a full IQ score ≥ 80. Likewise, children with ADHD were recruited from the National Taiwan University Hospital and verified by the head child psychiatrist to have ADHD combined type (American Psychiatric Association, 2000) based on the DSM-IV criteria, while controls were recruited from the local schools and had no clinically relevant diagnoses. The study was approved by the research ethics committee of the National Taiwan University Hospital, and is compliant with the World Medical Association Declaration of Helsinki.

6.2.2 Procedure

All children at both sites underwent one Go/NoGo task session. If children with ADHD were taking medication for ADHD during participation, they were required to be free of medication 48 hours before the test, with a physician's approval. Mothers were asked to complete the CBCL/SNAP-IV while their children were performing the task.

6.2.3 Maternal Interview - Chinese Version of the Kiddie Epidemiologic version of the Schedule for Affective Disorders and Schizophrenia (K–SADS-E)

In Taiwan, mothers of children with or without ADHD were interviewed after their child was referred by psychiatrists or recruited from local schools, in order to confirm the child's diagnoses. Previous studies have proved the CK–SADS-E to be a reliable and valid instrument to assess child psychiatric disorders in Taiwan. The CK-SADS-E has been widely used in a variety of studies on the mental illnesses of children and adolescents in Taiwan (Gau et al., 2009; Gau et al., 2005; Gau et al., 2010).

6.2.4 Child Behavior Checklist – Parent Form (CBCL; Achenbach, 1991)

This scale is designed to assess competencies and behavioral/emotional problems in children ages 4 to 18. The problem items are scored into eight specific scales: aggression, anxiety/depression, inattention, delinquency, social problems, somatic complaints, thought problems, and withdrawal. The Chinese (Yang, Chen, & Soong, 2001; Yang, Soong, Chiang, & Chen, 2000) and German (Arbeitsgruppe Deutsche Child Behavior Checklist, 1998) versions of the CBCL were used for the Taiwanese and German samples, respectively. The German and Chinese versions of the CBCL have been widely used in previous ADHD studies (e.g., Gau, Lin, Shang, Liu, & Chiu, 2010; Paul-Jordanov et al., 2010).

6.2.5 Swanson, Nolan, and Pelham IV Scale (SNAP-IV) – Parent Form

In Taiwan, the SNAP-IV was used in addition to the CBCL to assess ADHD and ODD symptoms of children. The SNAP-IV consists of 26 items for ADHD and ODD symptoms of the DSM-IV (Swanson et al., 2001). Items included in the scale address the criteria of ADHD, which are inattention (items 1 – 9) and hyperactivity/impulsivity (items 10 – 18), and the criteria of ODD (items 19 – 26). The SNAP-IV is based on a 4-point rating scale (Not at all = 0, Just a little = 1, Quite a bit = 2, and Very much = 3) to describe the degree to which the behavior is abnormally frequent and severe as compared to normal childhood behavior. The Chinese version of the SNAP-IV (Liu, et al., 2006) was reported to have satisfactory test-retest reliability, internal consistency, concurrent validity, and discriminant validity of the subscales (i.e., inattention, hyperactivity/impulsivity, and ODD) and is used often in ADHD research (e.g., Gau et al., 2007)

6.2.6 Neuropsychological Task

Germany. The classification task combined with a Go/NoGo task consisted of 300 stimuli (50% colored drawings of transportation vehicles and 50% colored drawings of animals) (see Figure 6.1). The stimuli lasted 1000 ms and were presented with an ISI of

1500 ms. A fixation cross was shown 500 ms before each stimulus, in the middle of the screen. Children were asked to respond to animals and vehicles by pressing one of two colored buttons, respectively. The correspondence of stimulus type and response-button was reversed after the first half of the experiment, to prevent the task from becoming too easy. A practice session of 30 trials was introduced in each half of the experiment to ensure that children understood the task. In 33% of the trials, a stop sign—a white spread-out hand on a circular purple background—was presented 150 ms before the stimulus. The stop sign indicated that no response was to be given on the following trial. The trials using stop signs were treated as NoGo trials; all other trials were Go trials (source from Paul-Jordanov et al., 2010).

Taiwan. The Chinese version of the Go/NoGo task was adapted from the task used in Germany. However, the Chinese version is slightly different from the German version of the Go/NoGo task in which a total number of 360 stimuli was included in the Go/NoGo task, and the practice session in each half of the task consisted of 16 trials. The rest of the task was the same as the German version of the Go/NoGo task.

6.2.7 Data Analysis

Behaviorally, three dependent variables—the rate of correct responses on Go trials, the rate of correctly inhibiting responses on NoGo trials, and reaction times in correct Go trials—were analyzed. Student's independent *t*-tests and Mann-Whitney *U*-tests were computed for between-group comparisons when data were normally distributed and not normally distributed, respectively. Accordingly, for the German sample, Mann-Whitney *U*-tests were used for response data of Go and NoGo trials and Student's independent *t*-tests were computed for reaction times in Go trials (source from Paul-Jordanov et al., 2010). For the Taiwanese sample, the Mann-Whitney *U*-test was used only for response data of NoGo

trials; Student's independent *t*-tests were computed for correct response rates and reaction times on Go trials. The significance level of all statistical analyses was 5%.

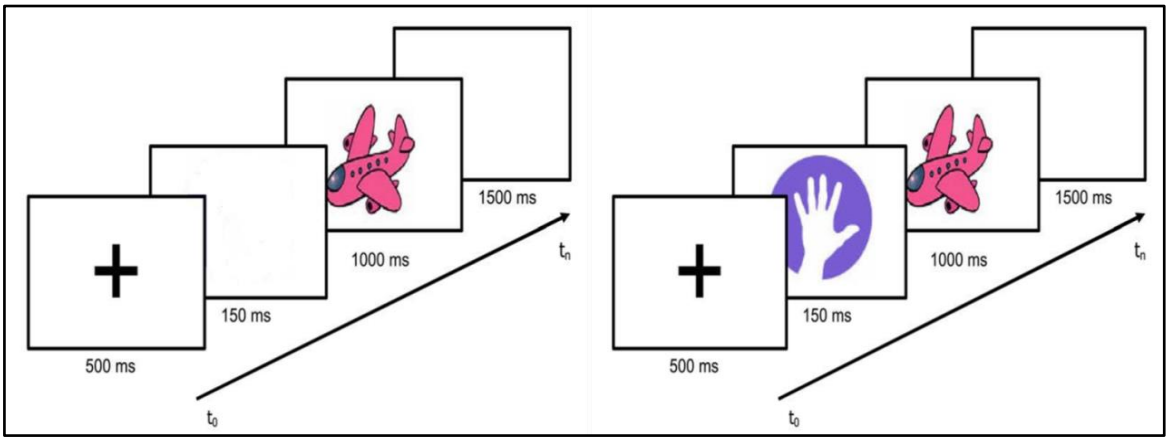

Figure 6.1 Examples of Go and NoGo trials. Source from Gawrilow and Gollwitzer (2008).

6.3 Results

Germany. The ADHD group scored higher than the norm overall on internalization and externalization of the CBCL (all Ts > 63), while the control group scored in the normal range on the three scales (all Ts < 56.91). Children with ADHD had lower correct rates than non-ADHD children on Go and NoGo trials (Z = -2.94, p = .002 and Z = -2.13, p = .03, respectively, see Table 6.1). Additionally, as shown in Table 6.2, children with ADHD exhibited pronouncedly slower reaction times than did controls (p = .004) (source from Paul-Jordanov et al., 2010).

Taiwan. A shown in Table 6.3, children with ADHD scored significantly higher than non-ADHD controls on most of the scales of the CBCL (Aggressive behavior: ADHD: 66.0 ± 13.58 vs. Control: 46.20 ± 8.37, p < .001; Anxious/Depressed: ADHD: 60.86 ± 14.86 vs. Control: 48.59 ± 7.80, p = .007 ; Attention problems: ADHD: 63.11 ± 9.10 vs. Control: 41.14 ± 4.28, p < .001; Delinquent behavior: ADHD: 57.74 ± 10.31 vs. Control: 45.65 ± 3.29, p < .001; Social problems: ADHD: 62.72 ± 12.56 vs. Control: 46.49 ± 6.06, p < .001;

Thought problems: ADHD: 70.27 ± 20.92 vs. Control: 45.38 ± 5.87, $p < .001$; Withdrawn: ADHD: 54.10 ± 10.86 vs. Control: 44.79 ± 5.52, $p = .009$; Internalizing problems: ADHD: 57.31 ± 12.65 vs. Control: 46.0 ± 7.08, $p = .005$; Externalizing problems: ADHD: 64.67 ± 12.92 vs. Control: 45.84 ± 6.21, $p < .001$), except on the scale of somatic complaints (ADHD: 50.98 ± 11.12 vs. Control: 45.12 ± 5.68, $p = .072$).

Similarly, group differences were pronouncedly found on ADHD (Inattention: ADHD: 56.0 ± 7.74 vs. Control: 45.15 ± 5.80, $p < .001$; Hyperactivity/Impulsivity: ADHD: 53.90 ± 4.70 vs. Control: 45.04 ± 3.72, $p < .001$) and ODD symptoms (ADHD: 53.53 ± 11.41 vs. Control: 42.20 ± 5.18, $p = .001$) of the SNAP-IV.

In terms of the behavioral performance on the Go/NoGo task (i.e., rates of correct Go trials, reaction times on Go trials, and correct inhibition rates on NoGo trials), Shapiro-Wilk's tests ($p > .05$) (Shapiro & Wilk, 1965; Razali & Wah, 2011) and visual inspections of corresponding histograms, normal Q-Q plots, and box plots were used for the normality tests of sample distributions. The results showed that the rates of correct Go trials were approximately normally distributed for both ADHD and control groups. There was a skewness of 0.39 (Standard Error [SE] = 0.52) and a kurtosis of -0.51 (SE = 1.01) for the ADHD group and a skewness of -0.90 (SE = 0.56) and a kurtosis of -0.09 (SE = 1.09) for healthy controls. Similarly, the reaction times on Go trials were approximately normally distributed for both groups, with a skewness of -0.73 (SE = 0.52) and a kurtosis of 0.31 (SE = 1.01) for children with ADHD and a skewness of -0.07 (SE = 0.56) and a kurtosis of -0.02 (SE = 1.09) for controls. The tests of normality for correct NoGo trials (i.e., responses are successfully inhibited) were at significance levels for both groups (ADHD: $p < .001$ vs. Control: $p < .05$). Additionally, Levene's tests were used to verify the equality of variances on the rates of correct Go trials and reaction times on Go trials in the samples (homogeneity of variance) ($p > .05$) (Martin & Bridgmon, 2012). A non-parametric Levene's test verified

the equality of variances in the samples (homogeneity of variance) ($p > .05$) (Nordstokke & Zumbo, 2010; Nordstokke, Zumbo, Cairns, & Saklofske, 2011).

Compared with healthy children, children with ADHD responded to Go trials less accurately ($t(33) = -5.45$, $p < .001$) and more slowly ($t(33) = 3.55$, $p = .001$). No significant group differences were found with respect to the rates of successfully inhibiting responses on NoGo trials ($Z = -0.83$, $p = .406$, see Table 6.4). Additionally, analyses of covariance were used to assess whether the group differences still significantly exist in terms of the rates of correct Go trials and reaction times on Go trials after controlling for the ages of the children. Results showed that the covariate, child's age, was not significantly related to the rates of correct Go trials, $F(1, 32) = 2.90$, $p = .098$. A main effect of Group on the rates of correct Go trials was still pronounced after controlling for age, $F(1, 32) = 26.70$, $p < .001$. However, the covariate, child's age, was significantly related to the reaction times on Go trials, $F(1, 32) = 12.65$, $p = .001$, indicating that older children responded to Go trials more quickly than younger children, $F(1, 32) = 12.65$, $p = .001$. A significant main effect of Group on the reaction times on Go trials remained after controlling for age, $F(1, 32) = 11.28$, $p = .002$.

Table 6.1

Behavioral Results for the ADHD and Control Groups in Germany

Variable	ADHD (N = 11)	Controls (N = 16)	Statistic	*p* value
Correct Go trials (%)				
Median	77.1	85.4	*Z* = -2.94	.002
Minimum	55.8	74.2		
Maximum	86.3	90.8		
Correct NoGo trials (%)				
Median	88.3	95.4	*Z* = -2.13	.030
Minimum	45.8	80.8		
Maximum	99.2	99.2		

Note. RT = reaction time. Table adapted with permission from Paul-Jordanov et al. (2010).

Table 6.2

Reaction Time Results (Go Trials) for the ADHD and Control Groups in Germany

Variable	ADHD (N = 11)	Controls (N = 16)
Mean RT (ms)	656.26*	542.96
95% CI Lower boundary	598.68	497.43
95% CI Upper boundary	713.84	588.48

Note. RT = reaction time; CI = confidence interval. *Significant differences between children with ADHD and control children. Table adapted with permission from Paul-Jordanov et al. (2010).

Table 6.3

Sample Characteristics in Taiwan

Variable	ADHD Mean ± SD	Controls Mean ± SD	Statistics *t* value	Statistics *p* value
	(N = 19)	(N = 16)		
Age (years)	9.72 ± 1.14	10.12 ± 0.92	-1.14	.264
Male, n (%)	17 (89.5)	15 (93.8)		
Right handedness, n (%)	19 (100.0)	15 (93.8)		
CBCL – mother report (T score)	(N = 17)	(N = 13)		
Aggressive behavior	66.0 ± 13.58	46.20 ± 8.37	4.92	< .001
Anxious/Depressed	60.86 ± 14.86	48.59 ± 7.80	2.92	.007
Attention problems	63.11 ± 9.10	41.14 ± 4.28	8.77	< .001
Delinquent behavior	57.74 ± 10.31	45.65 ± 3.29	4.54	< .001
Social problems	62.72 ± 12.56	46.49 ± 6.06	4.66	< .001
Somatic complaints	50.98 ± 11.12	45.12 ± 5.68	1.88	.072
Thought problems	70.27 ± 20.92	45.38 ± 5.87	4.67	< .001
Withdrawn	54.10 ± 10.86	44.79 ± 5.52	2.82	.009
Internalizing problems	57.31 ± 12.65	46.0 ± 7.08	3.11	.005
Externalizing problems	64.67 ± 12.92	45.84 ± 6.21	5.27	< .001
SNAP-IV – mother report (T score)	(N = 17)	(N = 14)		
Total score	55.63 ± 6.43	44.19 ± 4.71	5.53	< .001
Inattention	56.0 ± 7.74	45.15 ± 5.80	4.33	< .001
Hyperactivity/ Impulsivity	53.90 ± 4.70	45.04 ± 3.72	5.73	< .001
Oppositional defiant disorder	53.53 ± 11.41	42.20 ± 5.18	3.66	.001

Note. SD = standard deviation; CBCL = Child Behavior Checklist.

Table 6.4

Behavioral Results for the ADHD and Control Groups in Taiwan

Variable	ADHD (N = 19) Mean ± SD	Controls (N = 16) Mean ± SD	Statistic	*p* value
Go RT (ms)	697.09 ± 55.88	626.62 ± 61.58	*t* = 3.55	.001
Correct Go trials (%)	61.72 ± 12.22	81.79 ± 8.96	*t* = -5.45	<.001
Correct NoGo trials (% median)	96.67	97.49	*Z* = -0.83	.406

Note. SD = standard deviation; RT = reaction time.

6.4 Discussion

In accordance with the hypotheses, the findings in the current study showed that lower correct rates and longer reaction times on Go trials existed among children with ADHD relative to local non-ADHD children both in Germany and Taiwan. However, the finding of decreased correct rates on NoGo trials in the ADHD group in Germany was not observed in the Taiwanese sample; the ADHD and control groups in Taiwan displayed equal rates on correct NoGo trials. We speculate that one possible reason for the discrepancy could be that the task was less demanding or challenging for participants in Taiwan; thus they could perform well on the task where inhibition is required. However, this should be interpreted with caution, as the sample sizes at both sites are small (also see Chapter 8 for more detailed discussion).

7. Study 2: Behavioral Investigation of the Effect of Methylphenidate among Children with ADHD in Germany and Taiwan

7.1 Overview of the Study

The primary aim of Study 2 was to investigate the effect of medication on behavioral performance as measured by the Go/NoGo task among children with ADHD, with the repeated testing of both ADHD and control groups in Germany and Taiwan. Therefore, we invited the children with and without ADHD in Study 1 to take part in Study 2. For the ADHD group, only those who were on medication or planning to take medication were recruited in Study 2. The Go/NoGo task was administered to children with ADHD at both sites when they were off and on medication.

7.2 Method

7.2.1 Participants

Germany. As in Study 1, the sample consisted of 11 (all boys) children who were diagnosed with ADHD combined type (American Psychiatric Association, 2000) as their primary disorder and 16 (including three girls) age-matched non-ADHD children (mean age ± SD: ADHD: 12.4 ± 0.4 years vs. Control: 12.5 ± 0.3 years). Children participated after giving written informed consent. All of the children with ADHD were taking prescribed medication, while none of the controls had any clinically relevant diagnoses or were taking any medication, according to their parents. The study was approved by the research ethics committee of the University of Konstanz and is compliant with the World Medical Association Declaration of Helsinki. Children with ADHD were recruited through a collaborating child psychiatric outpatient center in Konstanz. Control children were contacted through the participant record system of the University of Konstanz (source from Paul-Jordanov et al., 2010 and see Paul-Jordanov et al., 2010 for more details).

Taiwan. The sample included 10 children with ADHD (including two girls) and 14 healthy controls (one girl), aged 8 to 12 (mean age ± SD: ADHD: 9.86 ± 1.05 years, Control: 10.25 ± 0.88 years). All participants are required to have a full IQ score ≥ 80. Likewise, children with ADHD were recruited from the National Taiwan University Hospital and verified by the head child psychiatrist to have ADHD combined type (American Psychiatric Association, 2000) based on the DSM-IV criteria, while controls were recruited from the local schools and had no clinically relevant diagnoses. The study was approved by the research ethics committee of the National Taiwan University Hospital and is compliant with the World Medical Association Declaration of Helsinki.

7.2.2 Procedure

All participants at both sites underwent two Go/NoGo task sessions, approximately one month apart (Germany: ADHD: 42.5 ± 6.9 days, Control: 34.6 ± 5.7 days; Taiwan: ADHD: 36.6 ± 10.17 days, Control: 33.79 ± 7.32 days). Children on medication for ADHD at both sites were required to be free of medication 48 hours before the first test session with physician approval (Study 1), but they took their usual dosage of medication before the second test session. After completing the second session, participants were compensated with 20 Euro (Germany) (source from Paul-Jordanov et al., 2010) or approximately 13 Euro (Taiwan).

7.2.3 Neuropsychological Task

Germany. The classification task combined with a Go/NoGo task consisted of 300 stimuli (50% colored drawings of transportation vehicles and 50% colored drawings of animals) that were presented on a computer screen approximately 60 cm from the children's eyes. The stimuli lasted 1000 ms and were presented with an ISI of 1500 ms. A fixation cross was shown 500 ms before each stimulus, in the middle of the screen. Children were asked to respond to animals and vehicles by pressing one of two colored buttons,

respectively. The correspondence of stimulus type and response-button was reversed after the first half of the experiment, to prevent the task from becoming too easy. A practice session of 30 trials was introduced in each half of the experiment to ensure that children understood the task. In 33% of the trials, a stop sign—a white spread-out hand on a circular purple background—was presented 150 ms before the stimulus. The stop sign indicated that no response was to be given on the following trial. The trials using stop signs were treated as NoGo trials; all other trials were Go trials (source from Paul-Jordanov et al., 2010).

Taiwan. The Chinese version of the Go/NoGo task was based on the task used in Germany. However, the Chinese version was slightly different from the German version of the Go/NoGo task in that a total number of 360 stimuli was included, and the practice session in each half of the task consisted of 16 trials. The rest of the task was the same as the German version of the Go/NoGo task.

7.2.4 Data Analysis

Behaviorally, the rate of correct responses on Go trials, the rate of correctly inhibiting responses on NoGo trials, and reaction times in correct Go trials were analyzed. At both sites, when data were not normally distributed, Mann-Whitney *U*-tests for between-group comparisons or Wilcoxon tests for within-group comparisons were computed. When data were normally distributed, Student's independent *t*-tests or paired *t*-tests were used for independent (i.e., comparisons between ADHD and control groups) or paired (i.e., comparisons between first/without medication and second/with medication sessions) samples, respectively (also refer to Paul-Jordanov et al., 2010 for data analysis in detail on the German sample). The significance level of all statistical analyses was 5%.

7.3 Results

Germany. Children with ADHD had lower correct rates than non-ADHD children on Go and NoGo trials during the first (unmedicated) session ($Z = -2.94$, $p = .002$ and $Z = -$

2.13, p = .03, respectively, see Table 7.1). During the second (medicated) session, significant group differences existed only on Go trials (Z = -2.1, p = .03) but not on NoGo trials (Z = -0.7, p = .5). No within-group differences between the first and second sessions were revealed for either group (ADHD: Z = 0.3, p = .76 and Z = 1.2, p = .23 for Go and NoGo trials, respectively; Control: Z = 0.9, p = .36 and Z = 0.1, p = .91 for Go and NoGo trials, respectively) (source from Paul-Jordanov et al., 2010).

Additionally, as shown in Table 7.2, a significant interaction was revealed for reaction time on correct Go trials (F (1, 24) = 7.24, p = .013). Children with ADHD exhibited pronouncedly longer reaction times than did controls in the first (unmedicated) session (p = .004), while the groups did not differ in the second (medicated) session (p = .13). That is, the children with ADHD responded to Go trials significantly quicker in the medicated session as compared to the unmedicated session (p = .001). No differences between the sessions were observed for the control group (p = .80) (source from Paul-Jordanov et al., 2010).

Taiwan. As shown in Table 7.3, children with ADHD scored significantly higher than controls on most of the scales of the CBCL (Aggressive behavior: ADHD: 65.27 ± 16.50 vs. Control: 46.73 ± 8.50, p = .01; Anxious/Depressed: ADHD: 65.89 ± 18.12 vs. Control: 48.94 ± 8.04, p < .05; Attention problems: ADHD: 64.61 ± 10.55 vs. Control: 41.60 ± 4.14, p < .001; Delinquent behavior: ADHD: 57.78 ± 12.19 vs. Control: 45.95 ± 3.25, p < .05; Social problems: ADHD: 62.33 ± 12.91 vs. Control: 46.96 ± 6.08, p < .01; Thought problems: ADHD: 72.32 ± 22.01 vs. Control: 45.68 ± 6.03, p < .01; Internalizing problems: ADHD: 59.83 ± 16.07 vs. Control: 46.27 ± 7.32, p < .05; Externalizing problems: ADHD: 64.09 ± 15.42 vs. Control: 46.34 ± 6.21, p < .01), except on the scales of somatic complaints (ADHD: 51.30 ± 12.39 vs. Control: 45.34 ± 5.87, p = .158) and withdrawn (ADHD: 53.72 ± 14.42 vs. Control: 44.90 ± 5.75, p = .068).

Similarly, group differences were pronouncedly found on ADHD (Inattention: ADHD: 57.07 ± 8.86 vs. Control: 45.90 ± 5.28, $p = .001$; Hyperactivity/Impulsivity: ADHD: 55.46 ± 5.18 vs. Control: 45.28 ± 3.76, $p < .001$) and ODD symptoms (ADHD: 54.29 ± 14.90 vs. Control: 42.65 ± 5.10, $p = .05$) of the SNAP-IV.

With regard to the behavioral performance (i.e., the rates of correct Go and NoGo trials and reaction times on Go trials) on the Go/NoGo task, we used Student's independent *t*-tests or Mann-Whitney *U*-tests to assess whether ADHD and non-ADHD children who participated in Study 2 are different from ADHD and non-ADHD children who did not participate in Study 2, respectively, before we compared children with ADHD and controls in Study 2. Results showed that significant differences were not observed in the rates of correct Go trials (ADHD: $t(17) = -0.34$, *ns* vs. Control: $t(14) = 0.02$, *ns*), reaction times of Go trials (ADHD: $t(17) = 0.18$, *ns* vs. Control: $t(14) = -0.66$, *ns*), and rates of correct NoGo trials (ADHD: $Z = -0.45$, *ns* vs. Control: $Z = -0.88$, *ns*).

Consistent with those findings in the German sample, unmedicated children with ADHD in Taiwan were more likely than healthy controls to display lower rates of correct Go responses (ADHD: $M = 62.66\%$, $SD = 14.18\%$ vs. Control: $M = 81.77\%$, $SD = 8.83\%$, $t(22) = 2.35$, $p < .05$) and longer reaction times (ADHD: $M = 694.79$ ms, $SD = 68.25$ ms vs. Control: $M = 630.53$ ms, $SD = 64.30$ ms, $t(22) = -4.08$, $p = .001$) (see Figures 7.1 and 7.2), while the two groups differed pronouncedly on the rate of correct Go trials (ADHD: $M = 68.63\%$, $SD = 12.73\%$ vs. Control: $M = 82.11\%$, $SD = 11.25\%$, $t(22) = -2.74$, $p < .05$) but not on reaction times to Go trials (ADHD: $M = 671.26$ ms, $SD = 90.87$ ms vs. Control: $M = 624.84$ ms, $SD = 72.33$ ms, $t(22) = 1.39$, $p = .177$) during the second (medicated) session (see Figures 7.1 and 7.2). For the ADHD group, a significant difference between sessions was observed on the rate of correct Go trials ($t(9) = -3.18$, $p < .05$) with a large effect size ($d = 1.01$) but not on reaction times to Go trials ($t(9) = 1.63$, $p = .138$) with a medium effect

size (d = 0.51) (see Table 7.4). For the control group, no differences between the two sessions were pronouncedly observed on the rate of correct Go trials ($t(13) = -0.41$, $p = .893$) and reaction times to Go trials ($t(13) = 0.39$, $p = .706$) (see Table 7.4).

Similar to the results in Study 1, significant differences on the rate of correct NoGo trials between the ADHD and control groups were not observed in either of the two sessions (1^{st} session: Z = -0.824, $p = .410$; 2^{nd} session: Z = -0.479, $p = .632$). No within-group differences between the sessions existed for the children with ADHD (Z = -1.020, $p = .308$) and non-ADHD controls (Z = -1.735, $p = .083$) (see Table 7.5).

Table 7.1

Behavioral Results (% Correct Go Responses; % Correct Inhibitions after NoGo Sign) for the ADHD and Control Groups in Germany

Group	Trial	Session	Median % correct	Minimum % correct	Maximum % correct
ADHD	Go	Unmedicated/1	77.1*	55.8	86.3
		Medicated/2	80.4*	53.3	87.1
	NoGo	Unmedicated/1	88.3*	45.8	99.2
		Medicated/2	91.7 n.s.	42.5	99.2
Control	Go	1	85.4	74.2	90.8
		2	84.2	74.2	90.8
	NoGo	1	95.4	80.8	99.2
		2	96.3	87.5	98.3

Note. *Significant differences between children with ADHD and control children in corresponding conditions; n.s., no significant differences between the two groups in corresponding conditions. Table adapted with permission from Paul-Jordanov et al. (2010).

Table 7.2

Reaction Time Results (Go Trials) for the ADHD and Control Groups in Germany

Group	Session	Mean RT (ms)	95% CI Lower boundary	95% CI Upper boundary
ADHD	Unmedicated/1	656.26*	598.68	713.84
	Medicated/2	584.96 n.s.	537.35	632.56
Control	1	542.96	497.43	588.48
	2	538.93	501.30	576.57

Note. RT = reaction time; CI = confidence interval. *Significant differences between children with ADHD and control children in corresponding conditions; n.s., no significant differences between the two groups in corresponding conditions. Table adapted with permission from Paul-Jordanov et al. (2010).

Table 7.3

Sample Characteristics in Taiwan in Study 2

Variable	ADHD Mean ± SD	Controls Mean ± SD	Statistics	
			t value	*p* value
	(N = 10)	(N = 14)		
Age (years)	9.86 ± 1.05	10.25 ± 0.88	-0.99	.333
Male, n (%)	8 (80.0)	13 (92.9)		
Medication naïve, n (%)	2(20.0)	--		
Right handedness, n (%)	10 (100.0)	13 (92.9)		
CBCL – mother report (T score)	(N = 9)	(N = 12)		
Aggressive behavior	65.27 ± 16.50	46.73 ± 8.50	3.08	.010
Anxious/Depressed	65.89 ± 18.12	48.94 ± 8.04	2.62	.025
Attention problems	64.61 ± 10.55	41.60 ± 4.14	6.20	< .001
Delinquent behavior	57.78 ± 12.19	45.95 ± 3.25	2.84	.020
Social problems	62.33 ± 12.91	46.96 ± 6.08	3.31	.007
Somatic complaints	51.30 ± 12.39	45.34 ± 5.87	1.47	.158
Thought problems	72.32 ± 22.01	45.68 ± 6.03	3.53	.006
Withdrawn	53.72 ± 14.42	44.90 ± 5.75	1.94	.068
Internalizing problems	59.83 ± 16.07	46.27 ± 7.32	2.36	.039
Externalizing problems	64.09 ± 15.42	46.34 ± 6.21	3.26	.009
SNAP-IV – mother report (T score)	(N = 9)	(N = 13)		
Total score	57.23 ± 7.11	44.73 ± 4.45	5.09	< .001
Inattention	57.07 ± 8.86	45.90 ± 5.28	3.71	.001
Hyperactivity/ Impulsivity	55.46 ± 5.18	45.28 ± 3.76	5.36	< .001
Oppositional defiant disorder	54.29 ± 14.90	42.65 ± 5.10	2.26	.050

Note. SD = standard deviation; CBCL = Child Behavior Checklist.

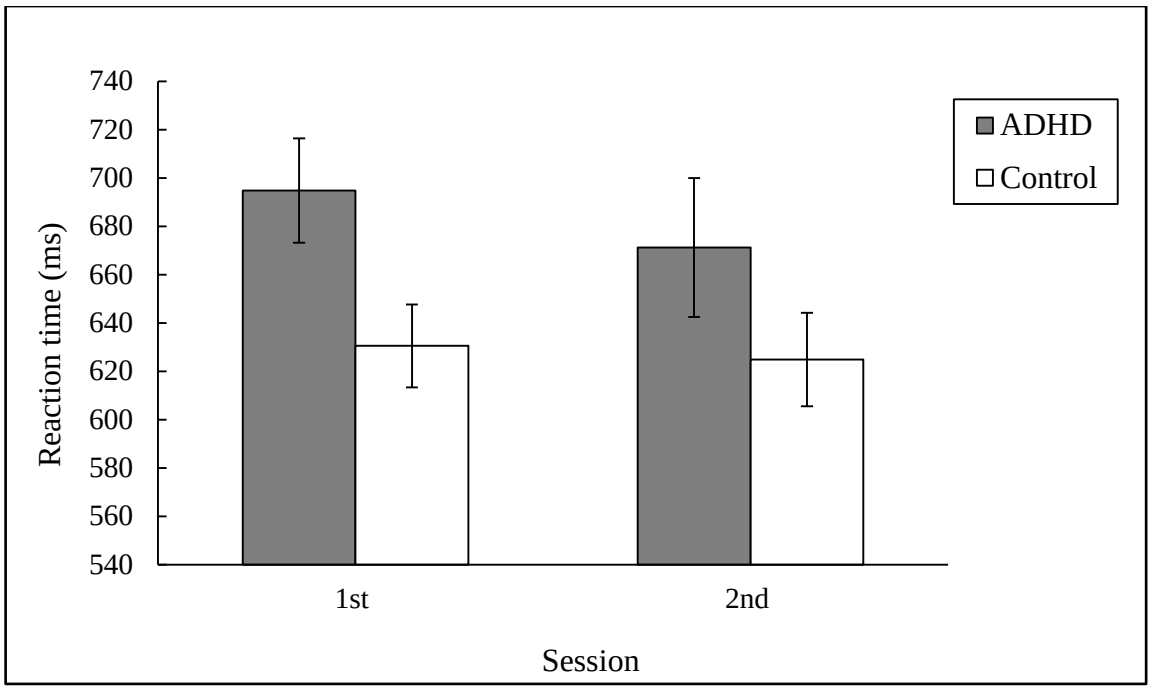

Figure 7.1 Differences on reaction times to Go trials between the ADHD and control groups in Taiwan.

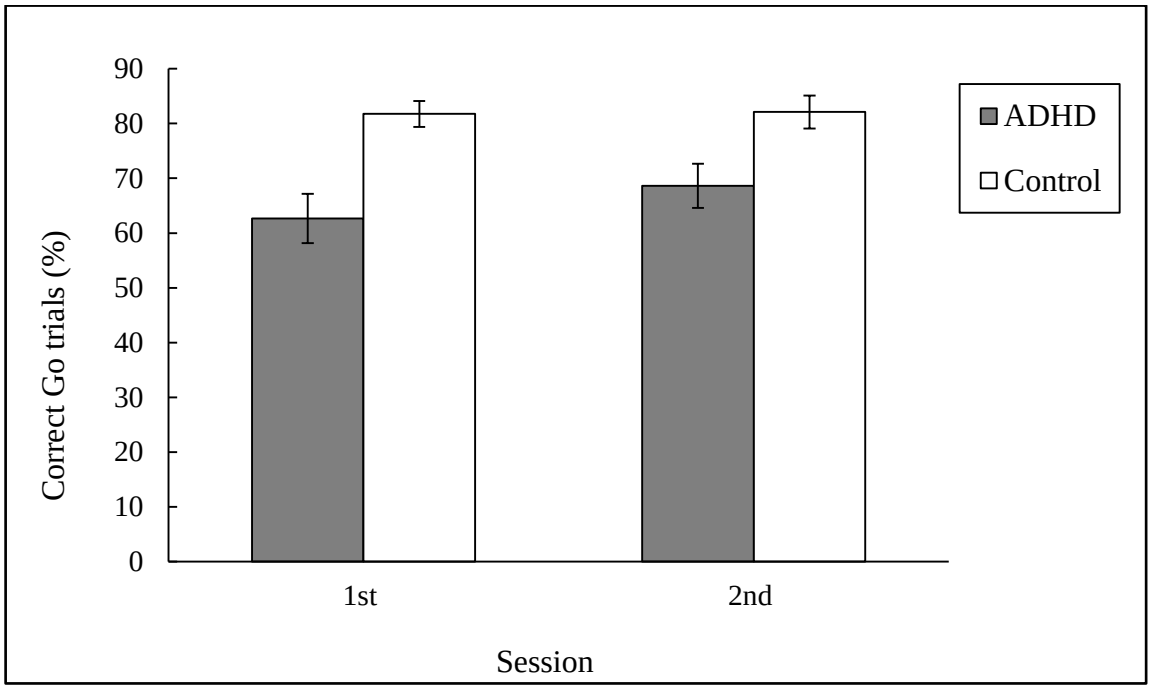

Figure 7.2 Differences on the rates of correct Go trials between the ADHD and control groups in Taiwan.

Table 7.4

Behavioral Results (Reaction Times on Go Trials; % Correct Go Responses) for the ADHD and Control Groups in Taiwan

Variable (Mean ± SD)	1st session	2nd session	Statistics		
			t value	*p* value	Cohen's *d*
ADHD					
Go RT (ms)	694.79 ± 68.25	671.26 ± 90.87	1.63	.138	0.51
Correct Go trials (%)	62.66 ± 14.18	68.63 ± 12.73	-3.18	.011	1.01
Controls					
Go RT (ms)	630.53 ± 64.30	624.84 ± 72.33	0.39	.706	0.10
Correct Go trials (%)	81.77 ± 8.83	82.11 ± 11.25	-0.14	.893	0.04

Note. SD = standard deviation; RT = reaction time.

Table 7.5

Results of Rates of Correct Inhibitions after NoGo Sign for the ADHD and Control Groups in Taiwan

Group	Variable	1st session	2nd session	Statistic	*p* value
ADHD	Median (%)	95.56	98.75	*Z* = -1.02	.308
	Minimum (%)	85.56	85.83		
	Maximum (%)	100.00	100.00		
Controls	Median (%)	97.07	99.17	*Z* = -1.735	.083
	Minimum (%)	90.83	90.83		
	Maximum (%)	100.00	100.00		

7.4 Discussion

In Germany, medication has proven effective in enhancing the rates of correct NoGo trials and decreasing reaction times to Go trials in children with ADHD, while significant differences on the rates of correct Go trials were still observed between the medicated children with ADHD and non-ADHD children (source from Paul-Jordanov et al., 2010 and see Paul-Jordanov et al., 2010 for more detailed discussion).

Similarly, in Taiwan, the ADHD and control groups were compatible on reaction times to Go trials, but not on the rates of correct Go trials in the second (medicated) session. A significant difference was observed between medicated children with ADHD and healthy children on the rate of correct Go trials. However, we found that children with ADHD displayed higher rates of correct Go trials in Session 2 (with medication) than in Session 1 (without medication), with a large effect size. As the efficacy of MPH persists over one year (The MTA Cooperative Group, 1999a), these results may indicate that MPH is effective to some extent in improving children's responses on Go trials—and that children with ADHD need a period of time in order to reach the same behavioral performance as normally developing children (Konrad, Neufang, Fink, & Herpertz-Dahlmann, 2007). This could explain that, after an interval of approximately 30 days, a significant difference was still observed between medicated children with ADHD and non-ADHD children in terms of the rate of correct Go trials.

Additionally, children with ADHD did not differ from controls regarding reaction times to Go trials in the second (medicated) session. Children with ADHD tended to respond to Go trials more quickly when on medication than when off medication, but the difference between sessions was non-significant. The effect size is moderate, which may indicate that MPH is effective in enhancing alertness (e.g., shorter reaction times) in ADHD to some extent (also see Chapter 8 for more detailed discussion).

With respect to the rates of correct NoGo trials, no significant differences between the ADHD and control groups in the Taiwanese sample were observed in the first (unmedicated) and second (medicated) sessions. No significant within-group differences were found for both ADHD and control groups (see Chapter 8 for more detailed discussion).

8. General Discussion and Implications for Future Research

Incorporating the data from the previous study (Paul-Jordanov et al., 2010), the current research is an initial attempt to investigate response inhibition among children with ADHD and normally developing children in Germany and Taiwan, using the same study design and task. Moreover, it explores the effect of MPH on response inhibition among children with ADHD at both sites. In this chapter, discussion is focused more on the results from the Taiwanese sample and the inconsistent findings with those observed in the German sample (please refer to Paul-Jordanov et al., 2010 regarding the discussion on the German sample).

8.1 Response Inhibition in ADHD

Consistent with the previous studies (Fallgatter et al., 2004; Gawrilow & Gollwitzer, 2008), unmedicated children with ADHD at both sites responded to Go trials less accurately (i.e., lower rates on correct Go trials) and more slowly (i.e., longer reaction times to Go trials) than did their non-ADHD counterparts (both Studies 1 and 2) (see Paul-Jordanov et al., 2010 for detailed discussion regarding the results from the German sample).

Findings at both sites may reflect that children with ADHD are impaired in selective attention and alertness (Fallgatter et al., 2004). The finding of decreased rates on correct NoGo trials (i.e., a failure to inhibit responses) in the unmedicated ADHD group in Germany (Paul-Jordanov et al., 2010) could not be replicated in the Taiwanese sample. This is because we found that, prior to the treatment of medication, Taiwanese children with ADHD exhibited equal rates on correct NoGo trials as healthy children (both Studies 1 and 2). This observation in the Taiwanese sample, however, is in line with the previous research that reported no significant group differences in child (Fallgatter et al., 2004) and adult (Dresler et al., 2010) populations with respect to inhibitory control. The mixed findings may indicate that deficits in response control, rather than a specific deficit in inhibition *per se*,

exist in ADHD (Fallgatter et al., 2004). These findings also support the earlier research, which demonstrated that ADHD is heterogeneous in etiology as a proportion of children with ADHD are impaired in other neuropsychological functions (e.g., delay aversion or sustained attention) rather than in behavioral inhibition (de Zeeuw, Weusten, Dijk, van Belle, & Durston, 2012).

The discrepancy observed between the German and Taiwanese samples with respect to the rate of correct NoGo trials could be accounted for by the following possibilities. First, the two studies, to our knowledge, are the first studies to apply the Go/NoGo task used by Paul-Jordanov et al. (2010) and Paul et al. (2007) in an ethnic group quite different from the German sample. Therefore, we speculate that according to Meyer (2005), cultural factors may account in part for the discrepancy in inhibition observed between the two ethnic groups because:

> ….cultural differences do affect the performance on neuropsychological measures. The reason may be that cultural factors are important for determinants of child rearing practices which may affect the brain's organization of cognition. There is therefore a need for assessment methods that are culturally valid for different ethnic groups. (p. 105)

For example, previous studies have suggested that parenting makes critical contributions to the development of executive functioning and the expression of ADHD behaviors (Blair, Raver, & Berry, 2014; Blair et al., 2011; Hammond, Müller, Carpendale, Bibok, & Liebermann-Finestone, 2012; Nikolas, Klump, & Burt, 2015), and thus it would be valuable to include relevant information regarding parenting to investigate whether parenting style plays a potential role in moderating children's behavioral performances on the Go/NoGo task (e.g., making children more inhibited or less inhibited).

Second, as suggested by Klorman (1991), deficient behavioral performance and abnormal electrophysiological components manifested by children with ADHD are more evident while they are performing difficult and demanding tasks. One study (Huizenga, van Bers, Plat, van den Wildenberg, & van der Molen, 2009) also demonstrated that inhibitory dysfunction is more pronounced in children and adolescents with ADHD for relatively complex tasks. Accordingly, the Go/NoGo task used in these present studies could be less demanding for participants in Taiwan, accounting for why there was no significant difference between children with ADHD and healthy controls on the correct rates of NoGo trials. Another possibility could be that a ceiling effect existed for groups. However, these speculations should be treated with caution due to the limited sample size. Thus, future research with a larger sample size and with various task difficulties is required to investigate these speculations more fully.

Third, some earlier research (Groom et al., 2008; A. Smith et al., 2006) has demonstrated that aberrant performance manifested by clinical patients may be observed at the electrophysiological level linked to brain activation, but not at the behavioral level (i.e., the behavioral performance of clinical patients is equivalent to or not significantly different from that of healthy individuals). Accordingly, despite the existence of no significant group difference in response inhibition (i.e., the rates of correct NoGo trials) in the Taiwanese sample at the behavioral level, it cannot be fully ascertained that no deficient inhibitory control exists among children with ADHD in Taiwan. As “brain activation measures may be more sensitive to abnormalities than performance” (A. Smith et al., 2006, p. 1049), more investigations from electrophysiological or neuroimaging studies with the same Go/NoGo task (Paul-Jordanov et al., 2010) would be helpful to clarify this finding.

8.2 The Effect of MPH on Response Inhibition in ADHD

After the treatment of medication, children with ADHD in Germany continued to exhibit lower rates of correct Go trials than their healthy counterparts, but no significant group differences were observed on reaction times during Go trials. In addition, the rates of correct NoGo trials did not differ markedly between medicated children with ADHD and non-ADHD controls (i.e., both groups were equally successful at inhibiting responses on NoGo trials) (source from Paul-Jordanov et al., 2010). Similarly, for the Taiwanese sample, significant group differences in the second (medicated) session were found on the rates of correct Go trials, but not on the reaction times on Go trials. Also, medicated ADHD children did not significantly differ from controls in terms of response inhibition, as the rates of correct NoGo trials were equal between the two groups.

In the German sample, within-subject comparisons between sessions showed that children with ADHD did not display significant differences on the rates of correct Go trials and of correct NoGo trials, but showed a significant difference on reaction times during Go trials (source from Paul-Jordanov et al., 2010). In the Taiwanese sample, within-subject comparisons between sessions showed that children with ADHD displayed a significant difference on the rate of correct Go trials, but showed no significant differences on reaction times during Go trials and the rate of correct NoGo trials. These differences could be explained by the difference in treatment duration or response to medication. Additionally, medication may have differential effects on different ethnic groups or populations, which could result from the difference in genetic background between the German and Taiwanese samples (McGough et al., 2006). Regarding this speculation, future cross-cultural studies will be needed to extend our understanding by exploring shared and group-specific effects of MPH on ADHD.

Specifically, in the Taiwanese sample, within-subject comparisons showed that children with ADHD appeared to benefit from MPH in terms of the rate of correct Go trials, even though the enhancement did not allow children with ADHD to reach the same performance as non-ADHD children did (e.g., no significant group difference between medicated ADHD children and non-ADHD children). As highlighted by the MTA cooperative groups (1999a), "Statistical significance, of course, cannot be interpreted as necessarily indicative of clinical or practical significance, and lack of significance is ever proof of the equivalency of treatments" (p.1083). Accordingly, the finding of the large effect size may indicate that MPH is effective in improving ADHD children's responses regarding the rate of correct Go trials, but children with ADHD may need a certain period of time in order to reach the same level as controls (Konrad et al., 2007). With respect to the efficacy of MPH on ADHD over time, future research with extended study periods will help to explore the long-term efficacy of MPH on response inhibition measured by this Go/NoGo task in ADHD.

Regarding reaction times in Go trials, children with ADHD did not display significant differences between sessions (i.e., without medication and with medication), but a medium-sized effect existed between sessions. Along with the finding that children with ADHD did not differ pronouncedly from controls in the second (medicated) session, it may indicate that MPH is effective to some extent in reaction times during Go trials. Likewise, studies with larger sample sizes are needed to clarify this observation.

With respect to the inhibition-related index (i.e., the rate of correct NoGo trials), children with ADHD did not significantly differ from non-ADHD children in the second (medicated) session, as observed in the first (unmedicated) session. Within-subject comparisons showed no pronounced differences between sessions for both groups. The finding of no significant MPH effect on inhibition in ADHD in the Taiwanese sample could

be explained by the hypothesis of baseline-dependent effects of psychostimulants (Eagle et al., 2008)—that is, patients with worse baseline performances improve more regarding commission errors after the treatment with psychostimulants. Also, as pointed out by Eagle et al. (2008, p.448), "In up to 30% of ADHD cases, methylphenidate fails to improve or even worsens symptoms such as deficient action inhibition (Cantwell, 1996; Krause et al., 2005), perhaps because MPH only improves deficient action inhibition in the cases that have the most pronounced action-inhibition deficits." As, prior to stimulant treatment, no significant group difference in inhibition was observed between children with ADHD and non-ADHD controls in the Taiwanese sample (see Studies 1 and 2), it is speculated that most children in the ADHD group may have less symptom severity. Instead of taking the sample as a whole, future research investigating the effect of MPH on inhibition in subgroups exhibiting different levels of symptom severity will be helpful in clarifying the speculation and observation.

8.3 Limitations of the Current Research

Although the current research advances the literature on the effect of medication on response inhibition in children with ADHD, several limitations remain. First, we did not control for comorbid disorders, intelligence, and the severity of ADHD at both sites. These factors could lead to different interpretations of the current results, as they moderate the association between ADHD and executive functioning, and thus affect children's behavioral performance during inhibition tasks (The MTA Cooperation Group, 1999b; Willcutt et al., 2005). Second, the small age ranges of the samples at both sites—between approximately 8 and 12 years—and the sample composition (i.e., mostly male participants at both sites) may hamper the generalization of the current findings to other age or gender groups (The MTA Cooperative Group, 1999b). Third, and most importantly, the small sample sizes both in Germany and Taiwan may preclude a definitive statement for the present research.

9. Implications for Clinical Practice and Conclusion

9.1 Implications for Clinical Practice

Currently, clinicians and practitioners follow the criteria of the DSM or ICD system regarding the diagnosis of ADHD. They may partly rely on parental reports regarding children's behaviors and refer to children's performance on neuropsychological tasks (e.g., CPT) during the diagnosis period. The current research brings our attention to the potential role of culture on children's performance during neuropsychological tasks, and even on the patterns of parent-rated reports. As discussed earlier, children with ADHD may manifest differential deficient behavioral patterns during neuropsychological tasks due to the heterogeneity of ADHD and/or the influence of parenting practices on children's behaviors.

Parental reports on children's behaviors may be influenced by personal characteristics (Olson et al., 2011) or biased by culture (Thorell, Veleiro, Siu, & Mohammadi, 2013). For instance, a cross-cultural study conducted by Thorell et al. (2013) found that, relative to other samples (i.e., Sweden, Spain, and Iran), Chinese children aged 6-11 years were rated as having more executive functioning deficits, which could result from cultural bias. Moreover, prior research has demonstrated that professionals across countries display variations in rating hyperactive-disruptive behaviors, even though identical criteria are applied (Mann et al., 1992). Based on these observations, it may be valuable to explore the culture-specific behavioral patterns in ADHD in addition to the commonly-observed impaired symptoms, particularly during a specific neuropsychological task, across cultures. Also, as parental perceptions of children's behaviors (e.g., the presence and extent of hyperactive behaviors) vary across cultures, understanding parental response patterns or characteristics when rating ADHD-related behaviors would be helpful for clinical practices during the diagnosis of ADHD.

With respect to the management of ADHD, even though medication is shown to be superior in improving the core symptoms of ADHD, multimodal and behavioral treatments may be effective for some domains, e.g., academic performance, social skills (The MTA Cooperative Group, 1999a, 1999b). In addition, several factors (e.g., gender, previous medication treatment, comorbid disorders, and treatment acceptance) are reported to be related to the treatment effect across outcome domains. As highlighted by Hinshaw and Arnold (2015), it is important to consider relevant moderators or mediators involved in differential patterns of outcomes during the treatment period. Moreover, differential response patterns after the treatment of medication, and the extent to which medication exerts its effect on ADHD, may vary across cultures or countries. Consequently, understanding the potential factors related to the treatment effect and response patterns, and characteristics across cultures, would be of great help for clinicians and practitioners to effectively manage not only ADHD symptoms but also associated dysfunctional behaviors.

ADHD is presently viewed as a chronic disorder. The long-term effectiveness of the treatments remains unclear, although the short-term effectiveness of medication and behavioral treatments has been clearly demonstrated (Hinshaw & Arnold, 2015). Therefore, it would be compelling to explore the long-term effectiveness of the treatments (e.g., pharmacological treatment, behavioral intervention, and combined treatment) on ADHD and how different types of treatments interact with psychosocial factors (e.g., maternal characteristics, family functioning, parent-child relationship) on the development of ADHD and other behaviors (e.g., aggressive and disruptive behaviors).

In addition to the treatment of medication, clinicians and practitioners may apply behavioral strategies on the management of ADHD. As indicated by Oettingen, Sevincer, and Gollwitzer (2008), the context of socio-culture may have a potential impact on the effect of forming behavioral strategies. Hence, it would be worthwhile to consider the extent

to which a specific behavioral strategy is applied in one cultural setting and the differential effects it has on ADHD symptoms and associated functioning. Also, clinicians and practitioners need to help children with ADHD and their parents learn and consolidate their skills and apply behavioral strategies in daily life in order to obtain long-lasting benefits (The MTA Cooperative Group, 1999a).

Finally, the "one size fits all" approach (The MTA Cooperative Group, 1999a, p.1083) may not be appropriate for the treatment of ADHD across cultures, or even within one cultural setting. The current research and previous findings (e.g., The MTA Cooperative Group, 1999b) highlight the impacts of culture, moderators, and mediators on the treatment of ADHD. Considering these potential influential factors on the diagnosis and management of ADHD would lead clinicians and psychiatrists to offer more effective and client-based treatments for ADHD.

9.2 Conclusion

To summarize, this is the first research that used the identical inhibition task paradigm to explore the effect of MPH on behavioral performance in children with ADHD in Germany and Taiwan. The current findings, in accordance with previous studies (Broyd et al., 2005), may indicate that MPH is effective in ameliorating deficient response inhibition (shown in the German sample) and other behavioral performances (shown both in the German and Taiwanese samples). However, future studies investigating psychosocial factors on the development and prognosis of ADHD would be helpful for the management of this disorder and other associated functioning. Finally, we expect that future studies with larger sample sizes or more evidence from studies using electrophysiological (e.g., EEG) or imaging techniques (e.g., fMRI) will confirm the present observations to better reveal the underlying mechanism in ADHD and the effects of medication on this disorder.

10. References

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慣用手問卷

日期(西元年/月/日): 2013/ ___ ___/ ___ ___ ID:

不同於一般調查問卷的方式，下列各個活動應該用手勢表達出來。施測者依序提出請求，然後在左手/右手欄位打勾。請勾選出慣用手，也就是，哪一隻手握著/使用剪刀，筷子，掃帚(在上端)，火柴，或是轉開瓶蓋? 當受測者不確定哪一隻手是慣用手時，也就是有時是用右手，有時是用左手，那麼請在左手/右手兩個欄位都打勾 (或是再進一步詢問)。對於第八題，很多受測者不是很確定，這時施測者可要求受測者在原地做出打掃的動作，來幫助他們。

請你示範一次, 你如何...		左手	右手
1)	... 寫字		
2)	... 畫圖		
3)	... 丟球		
4)	... 拿剪刀剪東西		
5)	... 刷牙		
6)	... 拿筷子夾東西		
7)	... 拿湯匙吃東西		
8)	... 用掃帚掃地		
9)	... 點火柴		
10)	... 將瓶子上的瓶蓋轉開		

慣用手(註解:腦側化)商值(LQ)的計算是根據於勾選的左手/右手次數總和(正值為右手，負值為左手)， 如下:

LQ = (R-L)/(R+L)x100 [ ]

他們用哪一隻手寫字?

媽媽	(左)	(右)	(?)	姊妹	(左)	(右)	(?)	女兒	(左)	(右)	(?)
爸爸	(左)	(右)	(?)	兄弟	(左)	(右)	(?)	兒子	(左)	(右)	(?)

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姓名:

ID: _____

日期/時間: _____ /

M-Check 電腦測驗(遊戲)問卷

1. 你覺得專心在這個遊戲有多容易?

1 2 3 4 5 6 7

一點也不容易 非常容易

2. 你有多專心在這個遊戲上?

1 2 3 4 5 6 7

一點也不專心 非常專心

3. 你覺得這個遊戲會令人感到辛苦嗎?

1 2 3 4 5 6 7

一點也不辛苦 非常辛苦

4. 你有多辛苦地玩這個遊戲?

1 2 3 4 5 6 7

一點也不辛苦 非常辛苦

5 你有多好地完成總是按下正確按鍵的任務?

1 2 3 4 5 6 7

一點也不好 非常好

6. 在手掌出現時，你有多好地完成總是不去按按鍵的任務?

1 2 3 4 5 6 7

一點也不好 非常好

7. 你有為這個遊戲想到任何的策略嗎?

沒有 有

如果有， 是什麼: _____

8. 你有多好地能將策略轉化為行動?

1 2 3 4 5 6 7

一點也不好 非常好

9. 你有意圖刻意在手掌出現時不去按按鍵嗎?

1 2 3 4 5 6 7

沒有，一點也沒有 有， 非常

10. 你有多好地能將這個意圖轉化為行動?

1 2 3 4 5 6 7

一點也不 非常好

---

11. 在手掌出現時不要去按按鍵， 對你而言達成這個目標有多重要?

1 2 3 4 5 6 7

一點也不重要 非常重要

12. 在手掌出現時不要去按按鍵， 對你而言達成這個目標有多辛苦?

1 2 3 4 5 6 7

一點也不辛苦 非常辛苦

13. 在手掌出現時不要去按按鍵， 對你而言維持這個目標有多簡單?

1 2 3 4 5 6 7

一點也不簡單 非常簡單

14. 在手掌出現時不要去按按鍵，你確信可以達到這個目標嗎?

1 2 3 4 5 6 7

不，一點也不 是的， 非常

15. 你有認真地看待這個遊戲嗎?

1 2 3 4 5 6 7

不，一點也不 是的， 非常

16. 你想要在這個遊戲表現特別地好嗎?

1 2 3 4 5 6 7

一點也不 非常好

17.你對於自己在遊戲中的表現感到自豪嗎?

1	2	3	4	5	6	7
一點也不						非常

18.你參加這個遊戲，只是因為可以得到獎金報酬嗎?

1	2	3	4	5	6	7
不是						是的

19. 你想要比別人好嗎?

1	2	3	4	5	6	7
一點也不						非常

20.你想要向施測者證明，你表現得好嗎?

1	2	3	4	5	6	7
一點也不						非常

21. 你認為你犯了多少錯誤呢?

1	2	3	4	5	6	7
一點也沒有						非常多

22. 你有多確定，你很好地完成這個任務了?

1	2	3	4	5	6	7
一點也不確信						非常確信

23. 你想要在這個遊戲表現好嗎?

1	2	3	4	5	6	7
不，一點也不						是的，非常好

請檢查是否有漏答的問題! 非常謝謝你的回答!

Task Instruction

ID:	_____
日期/時間:	_____ / _____
施測者:	_____
組別:	□ ADHD □ 控制組
施測語:	Control Instruction & Counterbalancing 1
測驗:	□ 1 □ 2

1. 指導語 階段一

我們現在開始這個電腦遊戲。 它總共有四個階段。

重要的是， 你應該試著盡可能安靜地坐著， 同時也不要亂動你的頭。

在螢幕上看到**交通工具** (汽車， 飛機， 火車頭， 船， 大貨車)， 你應該用右手按下**藍色的鍵**，
看到**動物**(老鼠， 豬， 牛， 貓， 雞)， 用左手按下**黃色的鍵**。

如同在每個電腦遊戲中， 要快速且正確，
也就是說，你應該總是盡可能**快速地**按下**正確的鍵**。

現在你就照著這樣做， 直到螢幕上出現**鐘鈴**。 然後， 我會再向你說明接下來要做什麼。

2. 指導語 階段二

現在你再做一次同樣的， 非常簡單地， 看到交通工具， 就用右手按下藍色的鍵，
看到動物， 就用左手按下黃色的鍵。

不同之前的是， 現在有時候在圖片之前會出現一隻手， 看到手的時候， 不論接下來的圖片是什麼， 你都不應該再按鍵。

3. 提問

你可以向我簡短的說明， 現在你應該做什麼嗎?

4. 指導語

很好，現在你的任務是:
看到交通工具按藍鍵， 看到動物按黃鍵， 在手後面出現的圖片， 不要按鍵。

當**鐘鈴**在螢幕上出現時， 我們會有一個短暫的休息。
然後我會再向你說明， 接下來要做什麼。

5. 指導語 階段三

接下來的會較困難， 要交換按鍵。

現在看到**交通工具**時， 用左手按下**黃色的鍵**。 看到**動物**時， 用右手按下**藍色的鍵**。

現在你就照著這樣做， 直到螢幕上出現**鐘鈴**， 然後我會再向你說明， 接下來要做什麼。

6. 指導語 階段四

現在你再做一次同樣的， 非常簡單， 看到交通工具， 用左手按黃鍵， 看到動物， 用右手按藍鍵。

不同的是， 現在手會再次出現，看到手的時候， 不論接下來的圖片是什麼， 你都不應該再按鍵。

7. 問題

你可以向我簡短的說明， 現在你應該做什麼嗎?

8. 指導語

很好，現在你的任務是:

看到交通工具， 按黃鍵， 看到動物， 按藍鍵， 在手後面出現的圖片， 不要按鍵。

當**鐘鈴**在螢幕上出現時， 這個遊戲就結束了。

9. 結束

在第一次的測驗之後，

1. 感謝他/她的參與!

在第二次的測驗之後，

1. 詢問 M-Check

2. 給受試者 500 元

3. 感謝他/她的參與!

Task Instruction

ID:	_____
日期/時間:	_____ / _____
施測者:	_____
組別:	□ ADHD □ 控制組
施測語:	Control Instruction & Counterbalancing 2
測驗:	□ 1 □ 2

1. 指導語 階段一

我們現在開始這個電腦遊戲。 它總共有四個階段。

重要的是， 你應該試著盡可能安靜地坐著， 同時也不要亂動你的頭。

在螢幕上看到**交通工具** (汽車， 飛機， 火車頭， 船， 大貨車)， 你應該用左手按下**黃色的鍵**，
看到**動物**(老鼠， 豬， 牛， 貓， 雞)， 用右手按下**藍色的鍵**。

如同在每個電腦遊戲中， 要快速且正確，
也就是說，你應該總是盡可能**快速地**按下**正確的鍵**。

現在你就照著這樣做， 直到螢幕上出現**鐘鈴**。 然後， 我會再向你說明接下來要做什麼。

2. 指導語 階段二

現在你再做一次同樣的， 非常簡單地， 看到交通工具， 就用左手按下黃色的鍵，
看到動物， 就用右手按下藍色的鍵。

不同之前的是， 現在有時候在圖片之前會出現一隻手， 看到手的時候， 不論接下來的圖片是什麼， 你都不應該再按鍵。

3. 提問

你可以向我簡短的說明， 現在你應該做什麼嗎?

4. 指導語

很好，現在你的任務是:
看到交通工具按黃鍵， 看到動物按藍鍵， 在手後面出現的圖片， 不要按鍵。

當**鐘鈴**在螢幕上出現時， 我們會有一個短暫的休息。
然後我會再向你說明， 接下來要做什麼。

5. 指導語 階段三

接下來的會較困難， 要交換按鍵。

現在看到**交通工具**時， 用右手按下**藍色的鍵**。 看到**動物**時， 用左手按下**黃色的鍵**。

現在你就照著這樣做， 直到螢幕上出現**鐘鈴**， 然後我會再向你說明， 接下來要做什麼。

6. 指導語 階段四

現在你再做一次同樣的， 非常簡單， 看到交通工具， 用右手按藍鍵， 看到動物， 用左手按黃鍵。

不同的是， 現在手會再次出現，看到手的時候， 不論接下來的圖片是什麼， 你都不應該再按鍵。

7. 問題

你可以向我簡短的說明， 現在你應該做什麼嗎?

8. 指導語

很好，現在你的任務是:

看到交通工具， 按藍鍵， 看到動物， 按黃鍵， 在手後面出現的圖片， 不要按鍵。

當**鐘鈴**在螢幕上出現時， 這個遊戲就結束了。

9. 結束

在第一次的測驗之後，

1. 感謝他/她的參與!

在第二次的測驗之後，

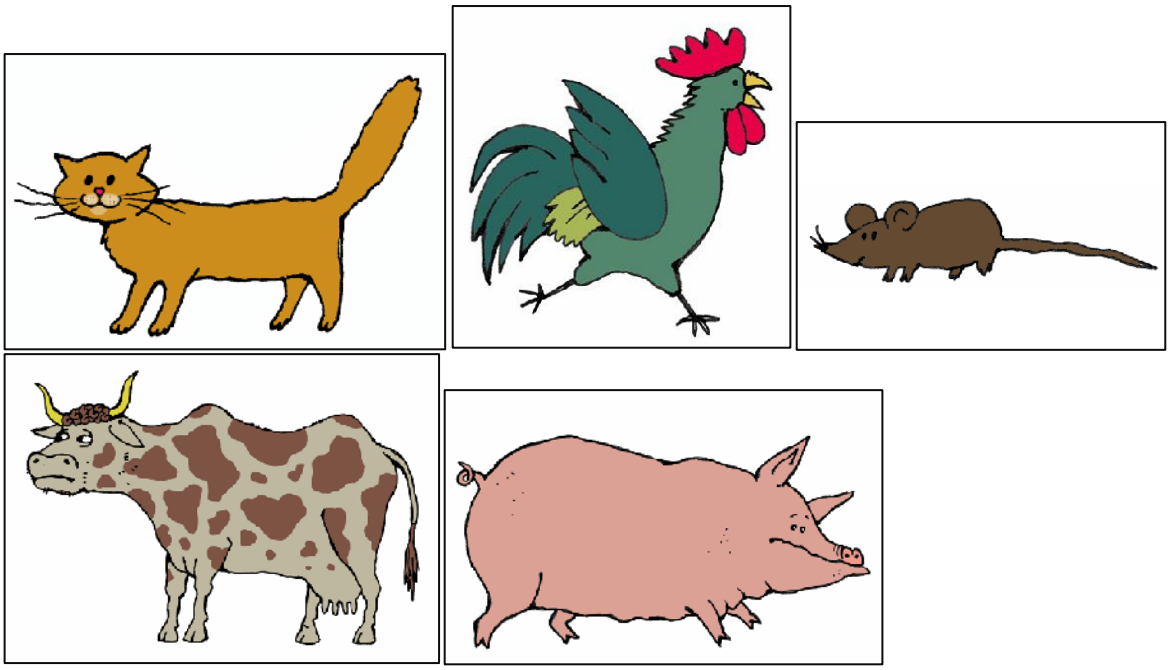
1. 詢問 M-Check

2. 給受試者 500 元

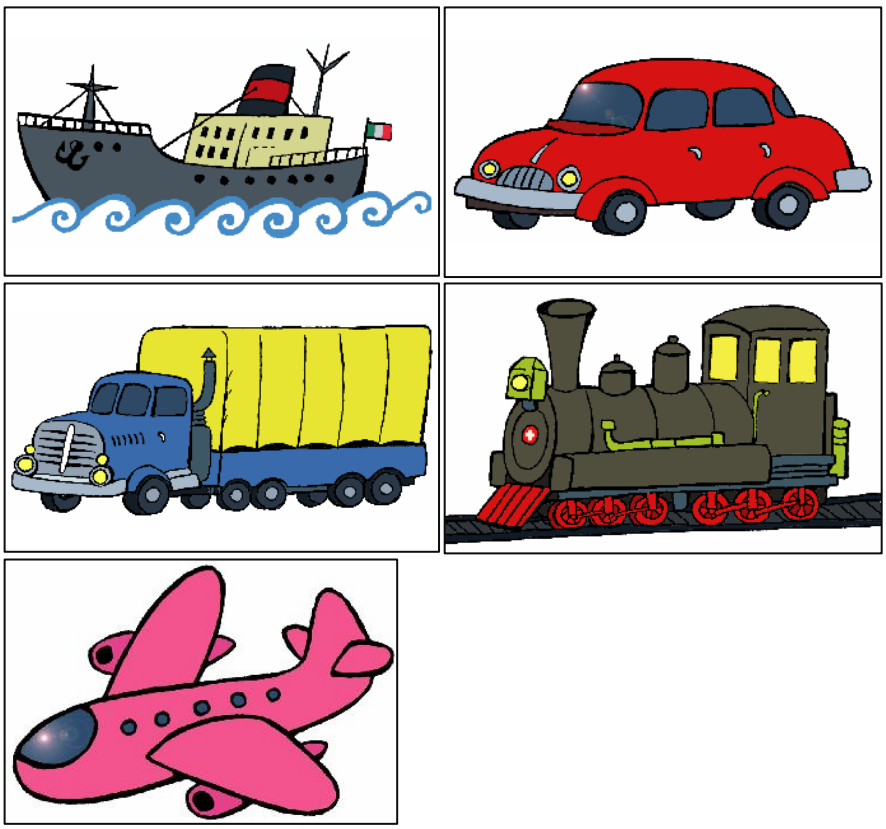
3. 感謝他/她的參與!

Task Stimuli

Animals

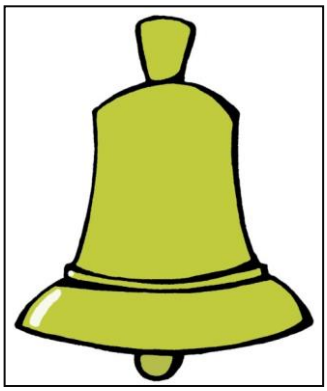


Transportation Vehicles



Stop Sign-Hand

Bell



Child Behavior Checklist – Parent Form

以下為行為的敘述，請以您的孩子**目前或最近六個月**的行為表現，圈選最符合他的答案。

不符合	部分符合	相當符合		
0	1	2	1	表現得比他的實際年齡小
0	1	2	2	好爭辯
0	1	2	3	好吹牛、自誇
0	1	2	4	不能長時間集中注意力
0	1	2	5	無法停止想有些事情；強迫性思考
0	1	2	6	坐不住、靜不下來，或活動量過高
0	1	2	7	黏著大人或太依賴
0	1	2	8	抱怨孤獨、寂寞
0	1	2	9	思想、語言雜亂，或看起來頭腦不清的樣子
0	1	2	10	好哭
0	1	2	11	待人殘忍或卑賤、欺負弱小
0	1	2	12	好做白日夢或沉溺於自己的思想中
0	1	2	13	太要求別人的注意
0	1	2	14	破壞自己的東西
0	1	2	15	破壞家人或同伴的東西
0	1	2	16	在家裡不守規矩
0	1	2	17	在學校不守規矩
0	1	2	18	不能跟其他小朋友和好相處
0	1	2	19	犯錯後好像沒有罪惡感
0	1	2	20	善妒
0	1	2	21	怕自己可能會想或做不對的事
0	1	2	22	覺得自己可能必須完美無缺
0	1	2	23	覺得或抱怨沒有人愛他
0	1	2	24	覺得別人對他有敵意
0	1	2	25	自卑或覺得自己沒有價值
0	1	2	26	常打架
0	1	2	27	常被嘲笑
0	1	2	28	常跟惹禍的同伴在一起
0	1	2	29	聽到不存在的聲音
0	1	2	30	衝動或不經考慮即行動
0	1	2	31	喜歡獨處
0	1	2	32	會說謊或欺騙
0	1	2	33	神經質、容易緊張
0	1	2	34	出現緊張性動作或抽筋
0	1	2	35	不被其他小孩喜歡
0	1	2	36	過度害怕或焦慮
0	1	2	37	覺得頭暈
0	1	2	38	有過度的罪惡感
0	1	2	39	過度疲倦
0	1	2	40	體重過重

不符合	部分符合	相當符合		
0	1	2	41	退縮不和別人相處
0	1	2	42	擔心、煩惱
			43	有以下找不出生理原因的病痛
0	1	2		a. 疼痛
0	1	2		b. 頭痛
0	1	2		c. 想吐，覺得身體不舒服
0	1	2		d. 與眼睛有關的問題
0	1	2		e. 發疹或其他皮膚方面的問題
0	1	2		f. 胃吐或常說肚子痛
0	1	2		g. 嘔吐
0	1	2	44	用身體攻擊他人
0	1	2	45	功課不好
0	1	2	46	動作協調不好或笨拙
0	1	2	47	特別喜歡和年紀較大的孩子玩
0	1	2	48	特別喜歡和年紀較小的孩子玩
0	1	2	49	拒絕說話
0	1	2	50	不斷重複某些動作；強迫性行為
0	1	2	51	離家出走
0	1	2	52	常大聲尖叫
0	1	2	53	喜歡保守秘密，不希望別人知道他的事
0	1	2	54	看到不存在的東西
0	1	2	55	敏感、容易受窘
0	1	2	56	縱火
0	1	2	57	好賣弄或好扮小丑
0	1	2	58	害羞或膽小
0	1	2	59	發呆
0	1	2	60	在家裡偷東西
0	1	2	61	在外面偷東西
0	1	2	62	怪異的行為
0	1	2	63	怪異的念頭
0	1	2	64	倔強固執、悶悶不樂、煩躁易怒
0	1	2	65	情緒突然地轉變
0	1	2	66	常鬧彆扭
0	1	2	67	多疑
0	1	2	68	咒罵或說髒話
0	1	2	69	太多話或愛說話
0	1	2	70	常嘲笑別人
0	1	2	71	脾氣暴躁
0	1	2	72	太常想到性
0	1	2	73	恐嚇他人
0	1	2	74	曠課逃學
0	1	2	75	活動量低、動作緩慢，或無精打采
0	1	2	76	不快樂、悲傷，或沮喪
0	1	2	77	說話異常大聲

不符合	部分符合	相當符合		
0	1	2	78	非醫療用途而使用酒精、興奮劑或麻醉品
0	1	2	79	有破壞物品的行為

SNAP-IV – Parent Form

請選擇一個代碼，最能表達在**過去的一個星期**中，您的孩子的狀況。

完全沒有	有一點點	還算不少	非常的多		
0	1	2	3	1	無法專注於細節的部份，或在做學校作業或其他的活動時，出現粗心的錯誤
0	1	2	3	2	很難持續專注於工作或遊戲活動
0	1	2	3	3	看起來好像沒有在聽別人對他（她）說話的內容
0	1	2	3	4	沒有辦法遵循指示，也無法完成學校作業或家事（並不是由於對立性行為或無法了解指示的內容）
0	1	2	3	5	組織規劃工作及活動有困難
0	1	2	3	6	逃避，或表達不願意，或有困難於需要持續性動腦的工作（例如學校作業或是家庭作業）
0	1	2	3	7	會弄丟工作上或活動所必需的東西（例如 學校作業，鉛筆，書，工具，或玩具）
0	1	2	3	8	很容易受外在刺激影響而分心
0	1	2	3	9	在日常生活中忘東忘西的
0	1	2	3	10	在座位上玩弄手腳或不好好坐著
0	1	2	3	11	在教室或是其他必須持續坐著的場合，會任意離開座位
0	1	2	3	12	在不適當的場合，亂跑或爬高爬低
0	1	2	3	13	很難安靜地玩或參與休閒活動
0	1	2	3	14	總是一直在動或是像被馬達所驅動
0	1	2	3	15	話很多
0	1	2	3	16	在問題還沒問完前就急著回答
0	1	2	3	17	在遊戲中或團體活動中，無法排隊或等待輪流
0	1	2	3	18	打斷或干擾別人（例如 插嘴或打斷別人的遊戲）
0	1	2	3	19	發脾氣
0	1	2	3	20	與大人爭論
0	1	2	3	21	主動地反抗或拒絕大人的要求或規定
0	1	2	3	22	故意地做一些事去干擾別人
0	1	2	3	23	因自己犯的錯或不適當的行為而怪罪別人
0	1	2	3	24	易怒的或很容易被別人激怒
0	1	2	3	25	生氣的及怨恨的
0	1	2	3	26	惡意的或有報復心的