

# Attention-Deficit/Hyperactivity Disorder and Behavioral Inhibition: A Meta-Analytic Review of the Stop-signal Paradigm

R. Matt Alderson · Mark D. Rapport · Michael J. Kofler

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**Abstract** Deficient behavioral inhibition (BI) processes are considered a core feature of attention deficit/hyperactivity disorder (ADHD). This meta-analytic review is the first to examine the potential influence of a wide range of subject and task variable moderator effects on BI processes—assessed by the stop-signal paradigm—in children with ADHD relative to typically developing children. Results revealed significantly slower mean reaction time (MRT), greater reaction time variability (SDRT), and slower stop-signal reaction time (SSRT) in children with ADHD relative to controls. The non-significant between-group stop-signal delay (SSD) metric, however, suggests that stop-signal reaction time differences reflect a more generalized deficit in attention/cognitive processing rather than behavioral inhibition. Several subject and task variables served as significant moderators for children's mean reaction time.

**Keywords** Stop-signal · Behavioral inhibition · Attention deficit hyperactivity disorder · ADHD · Meta-analysis

Theories of attention-deficit/hyperactivity disorder (ADHD) evolved from implied brain damage (Strauss and Lehtinen 1947) and dysfunction (Dolphin and Cruickshank 1951; Strauss and Kephart 1955) to single construct theories of sustained attention (Douglas 1972), arousal/activation regulation (Sergeant et al. 1999), working memory (Rapport et al. 2001), delay aversion (Sonuga-Barke 2002), and behavioral inhibition (Barkley 1997). Castellanos and Tannock (2002) provide a comprehensive review of these

models and their underlying psychological/neurobiological constructs and etiological factors.

Behavioral inhibition (BI) has garnered particular interest in recent years as a psychological construct used to describe a cognitive process that (a) sub-serves behavioral regulation and executive function (Barkley 1997), and (b) underlies the ability to withhold or stop an on-going response (Schachar et al. 2000). This latter process, its assumptions and underlying metrics, and moderators of BI function in children with ADHD relative to normal controls, serve as the focus for the current meta-analytic review.

Current models of behavioral inhibition are derived largely from Gray's (1982) theory of brain-behavior processes wherein an underactive behavioral inhibition system fails to provide sufficient anxiety and fearfulness, resulting in the initiation or continuation of unwanted behavior (Quay 1997). This inability to withhold or stop an on-going response is central to current theoretical models of ADHD, and may represent the primary component underlying executive functions such as working memory, self-regulation, internalization of speech, and reconstitution (for a review, see Barkley 1997). Performance measures used to index the BI construct traditionally involve a dual-task paradigm wherein participants respond to a primary stimulus and withhold a response when presented with a secondary stimulus. Examples of common BI measures include the (a) Go-No-Go task (Iaboni et al. 1995), (b) Change Task (Schachar et al. 1995), (c) Stroop Color-Word Interference Test (Barkley 1997), and (d) Stop-Signal Task (Logan et al. 1984). The stop-signal task (Logan et al. 1984) is the premier paradigm used to study children's ability to suppress prepotent and ongoing responses (i.e., inhibitory motor control).

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R. M. Alderson · M. D. Rapport (✉) · M. J. Kofler  
Department of Psychology, University of Central Florida,  
P. O. Box 161390, Orlando, FL 32816-1390, USA  
e-mail: mrappor@pegasus.cc.ucf.edu

## The Stop-signal Task

Investigations using the stop-signal task reveal that children with ADHD tend to have longer stop-signal reaction times relative to normal controls (Oosterlaan et al. 1998) — a finding consistent with current theoretical models of ADHD that emphasize the importance of an individual's ability to stop an ongoing response and inhibit responding to pre-potent stimuli (Barkley 1997). Its widespread adoption as a measure of behavioral inhibition is due to its unique ability to capture theoretically important cognitive processes by means of the stop-signal reaction time (SSRT) metric.

In a prototypical stop-signal paradigm, children are pre-trained to respond differentially to two stimuli (e.g., the letters X and O) using left and right response buttons. The average of these responses reflects the time required to receive the visual input, encode it, and emit a pre-trained motor response, referred to as mean reaction time (MRT). After practice training, children are instructed to withhold their response to the go-signal whenever it is followed by a stop-signal, typically an auditory tone presented within milliseconds following the go-signal. The ability to withhold or stop an activated motor response is reflected by the stop-signal delay (SSD) metric—the measured time interval between the presentations of the go- and stop-signals. For example, if two groups of children emit similar mean reaction times in response to visual stimuli, then differences in behavioral inhibition (SSRT: stop-signal reaction time) are assumed to be due to between-group differences in SSD based on the recommended formula ( $SSRT = MRT - SSD$ ). That is, one of the two groups required a shorter time interval (SSD) between the go- and stop-signals to inhibit their activated motor response when signaled to do so.

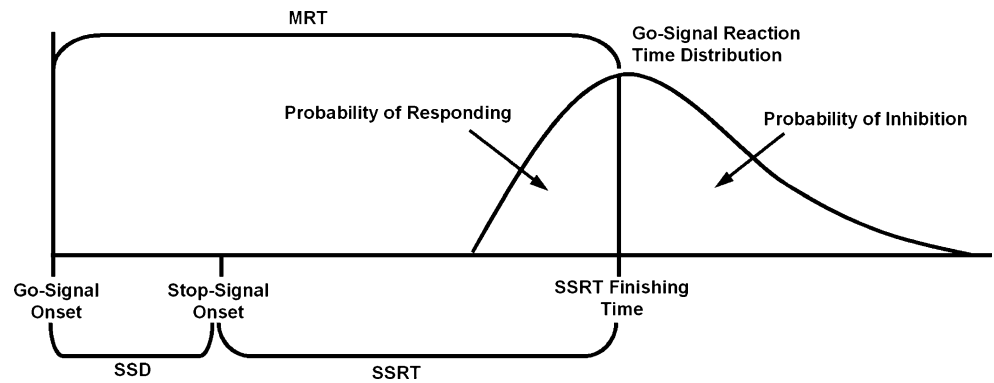
The theoretical underpinnings of the stop-signal paradigm are grounded in Logan's (1981) pioneering work in the field. Go- and stop-processes are hypothesized to operate independently of one another to enable and prevent the occurrence of controlled motor responses, respectively. When both processes are activated in close temporal sequence (i.e., go-signal activation followed by stop-signal activation), response execution depends on whether the stop process can overtake the go-process. Stop-signal reaction time (SSRT)—the primary measure of behavioral inhibition—thus reflects the relative speed of the stop process relative to the go-process, and is estimated by subtracting the time interval difference between the presentations of the go- and stop-stimuli (SSD) from the time required to process and emit a controlled motor response (MRT). This point becomes central to behavioral inhibition deficiencies ascribed to ADHD in the literature; between-group differences in BI functioning must be present after accounting for initial differences in simple reaction time.

Early versions of the stop-signal paradigm examined the probability of inhibiting using a range of fixed stop-signal delays—children completed blocks of trials with each block having a different SSD. Two limitations of the paradigm were subsequently recognized. The primary metric for estimating behavior inhibition (SSRT) required a complex, multi-step process. Calculating SSRT initially involved estimating the probability of inhibiting a motor response following a stop signal (a response rate value between 0 and 1), rank-ordering the distribution of MRTs, and determining the  $n$ th MRT (i.e., MRT percentile rank corresponding to response rate). SSD was subsequently subtracted from  $MRT_{nth}$  (i.e.,  $MRT_{nth} - SSD = SSRT$ ), and the calculation was repeated for each fixed SSD to obtain an overall mean value. Investigators also realized that children frequently adopted an overly cautious response bias by intentionally delaying their go-stimulus response (slowed MRT) in anticipation of a stop-signal (Logan et al. 1997). A dynamic *tracking* version of the stop-signal paradigm was developed to address these concerns, wherein the SSD was programmed to change following each trial based on a child's performance. Specifically, successful and unsuccessful inhibition of a motor response following the stop-signal causes the ensuing preprogrammed go/stop-signal interval to be shortened or lengthened by 50 ms, respectively. This modification has the desired effect of engendering a successful inhibition response rate of approximately 50% in all children, such that between-group differences in SSRT reflect differences in SSD rather than differential success rates, after MRT differences are factored out of the equation (Logan et al. 1997). Stated differently, any variability in SSRT is derived from three sources: (a) variability in SSD if MRT is held constant; (b) variability in MRT if SSD is held constant; or (c) variability in both MRT and SSD based on the formula of Logan et al. (1997). Specific implications for interpreting meta-analytic review findings are that a slow SSRT, coupled with a slow MRT in ADHD, indicates an inhibitory deficit in children with ADHD only if their SSD is also shorter relative to the control group SSD. An equivalent or longer SSD would suggest that children with ADHD exhibit equal or greater success at inhibiting their responses, relative to control children. The relationships among the go-stimulus, SSD, and SSRT are depicted graphically in Fig. 1.

## Meta-analysis

The original meta-analytic review (Oosterlaan et al. 1998) of stop-signal performance in children with ADHD was based on eight studies published between 1990 and 1997. Participants were males between 6 and 12 years of age, and included normal controls and children with single (i.e.,

**Fig. 1** Relationship of mean reaction time (*MRT*), stop-signal delay (*SSD*), and stop-signal reaction time (*SSRT*)



ADHD, Conduct Disorder) and comorbid disorders (i.e., ADHD+Conduct Disorder, ADHD+Oppositional Defiant Disorder). Children with ADHD and those with CD exhibited slower go and stop processes, and reduced ability to successfully inhibit relative to normal controls when measured with the stop-signal task, go-no-go task, and change task. The potential role of moderator variables on children’s performance was not quantified in the review.

A second meta-analytic review (Lijffijt et al. 2005) examined mean reaction time (MRT), stop-signal reaction time (SSRT), mean reaction time variability (SDRT), and five potential moderators of these indices (child-adult status, stop signal method, IQ, comorbidity with ODD/CD, and ADHD subtype) in 29 studies (17 child, 1 adolescent, 6 adult, and 5 mixed child–adolescent) published since the Oosterlaan et al. (1998) meta-analytic review. Child–adult status was the only significant moderator of between-group effect size differences in mean reaction time (0.29), mean reaction time variability (0.65), and stop signal reaction time (0.58). The authors concluded that the longer response times (MRT) and more lapses of attention (SDRT) in children with ADHD, coupled with a non-significant SSRT–MRT difference score, were consistent with a general inattention rather than behavioral inhibition model of ADHD.

The conclusions reached by Lijffijt et al. (2005) may be premature for several reasons. Including fixed and dynamically changing stop-signal delay studies to examine between-group differences in SSD poses a serious threat to the metric’s validity. Fixed stop signals have no associated within- or between-subject variability, and their inclusion with dynamically changing stop signal studies is likely to artificially deflate between-group differences in SSD effect size estimates. Age alone emerged as a significant moderator for between-group differences for all three BI matrices; however, this finding, based on a child–adult dichotomy rather than distinct child age groupings, may suppress between-group SSRT effect size estimates given the slower and more variable reaction times observed in younger children (Barkley 2005; Rapport et al. 2001). Their MRT–SSRT difference score—based on pooling pooled standard deviation scores—inaccurately reflects the magnitude of

between-group BI differences. Finally, the high within-group variability for study effects reported by the authors indicates that a considerable proportion of unexplained error may be due to uncontrolled sources not considered in either of the earlier reviews. Examination of additional potential moderating variables is warranted to address this issue.

### Goals of the Present Meta-analysis

The present meta-analytic review examines behavioral inhibition in children using the traditional stop-signal paradigm (i.e., two-choice primary task and discrete stop-signal). The unique contribution of the current review is its systematic examination of sample (age, diagnostic selection procedures) and task variable (type of go- and stop-stimuli, task trials, target frequency) moderator effects on children’s stop-signal BI performance either not quantified in previous reviews, or analyzed based on a limited number of studies. Moderating variables warrant scrutiny because of their potential to change the nature of dependent–independent variable relationships, with implications for theory development, refinement, and refutation (Holmbeck 1997). A total of 24 studies were included to accomplish this goal, including four studies published since the original meta-analysis but omitted from the Lijffijt et al. (2005) review (Konrad et al. 2000b; McInerney and Kerns 2003; Schachar et al. 2004; Walcott and Landau 2004), and eight studies included in the original meta-analytic paper (Oosterlaan et al. 1998) but omitted from the review of Lijffijt et al. (2005).<sup>1</sup> The present review also provides a more rigorous analysis of between-group stop-signal delay differences in children. This metric could not be examined and statistically analyzed until 1999—following the development of the dynamic tracking stop-signal para-

<sup>1</sup> Updated meta-analytic reviews frequently exclude studies that were recently reviewed based on a confirmatory approach (i.e., to determine whether ES differences of similar magnitude emerge based on the more recent and different series of studies). The current review includes all published studies to enable a broader moderator analysis and to confirm the SSD effect reported by Lijffijt et al. (2005) after controlling for methodological limitations.

digm—but provides a critical index for assessing between-group differences in stop-signal behavioral inhibition. Failure of the stop-signal delay (SSD) index to account for significant between-group variability in SSRT indicates that between-group study differences are more likely due to pre-existing differences in MRT that reflect inefficient cognitive processing and/or inattention rather than inhibitory control differences (Castellanos and Tannock 2002; Overtoom et al. 2002; Rapport et al. 2001). Larger mean reaction time variability (SDRT) in children with ADHD, which reflects more lapses of attention, may be explained by a general attention deficit consistent with an emerging endophenotypic model (Castellanos and Tannock 2002), a deficit of interference control (Nigg 2001), or a ubiquitous characteristic of ADHD. Inhibitory deficits, however, should be reflected by a disproportionately longer SSRT relative to MRT.

### Moderators and Coding of Moderators

**Age** The influence of children's age on BI performance indices was not examined in either the initial (Oosterlaan et al. 1998) or more recent (Lijffijt et al. 2005) meta-analytic review. It merits scrutiny, however, due to the well-documented developmental changes observed in children across a wide array of cognitive and motor tasks (Bedard et al. 2002; Nigg 1999; Williams et al. 1999). A significant age moderator effect would converge with the finding of Lijffijt et al. (2005; i.e., children are slower and more variable relative to adults), and may indicate that between-group differences in BI are underestimated when study samples include older children, or that underlying mechanisms or processes related to BI improve with development.

The mean of the ADHD and normal control samples were averaged to create an overall combined age mean for each study (the mean age difference between the ADHD and normal control samples was approximately 4 months) and subsequently divided into two categories: young (7 years–0 months to 9 years–11 months), and old (age 10 years–0 months to 12 years–11 months). Three studies reported a range of values and were excluded from the age effects analysis. Table 1 provides a summary of studies included in the meta-analysis.

**Diagnostic Procedures** The current meta-analysis is the first to examine whether differences in group assignment criteria moderate effect size estimates for traditional stop-signal dependent measures. Assignment to groups has varied from exclusive reliance on narrow-band rating scales to comprehensive diagnostic evaluations involving extensive history taking, a semi-structured clinical interview, and standardized rating scale scores. Diagnostic assignment based exclusively on rating scale cutoff scores appears to be

the least face valid method of grouping, considering the myriad disorders and conditions featuring attention and behavioral problems as core or secondary features (American Psychiatric Association 2000). Moreover, significant variability in symptom endorsement on structured and semi-structured clinical interviews is not accounted for by rating scale endorsements (McGrath et al. 2004); and none of the current scales or inventories obtain critical diagnostic information concerning symptom onset and course.

Diagnostic assignment based on single sources of information (e.g., rating scales) is likely to increase group membership heterogeneity and suppress BI effect size estimates by including non-ADHD children in the ADHD group. This is particularly salient owing to the high inter- and intra-day variability observed in children with ADHD across settings (Castellanos et al. 2005), and the moderate specificity of most rating scales (Rapport et al. 2006).

Two groupings of diagnostic criteria were formed. The first included studies that employed a comprehensive diagnostic procedure (i.e., a semi-structured or structured clinical interview complemented by teacher/parent questionnaires). The second included studies that relied exclusively on questionnaires or professional opinion (e.g., pediatric evaluation) to determine diagnostic standing.

**Stop-Signal Stimuli Modality** Stop-signal studies traditionally use either phonological/text-based go stimuli (e.g., “X” and “O”) coupled with an auditory tone as the stop-signal, or visual-spatial go and stop-signal stimuli (e.g., Rubia et al. 1998). Past investigations (Logan 1994; Logan et al. 1984) examining BI performance on the stop-signal task found minimal performance differences when go- and stop-signal stimuli were modality specific (i.e., both phonological or both visual-spatial), but neither same nor different stimulus modality influences on stop signal performance were analyzed in previous reviews (Lijffijt et al. 2005; Oosterlaan et al. 1998). Stimulus modality may prove to be a particularly robust moderator of between-group BI differences, owing to the distinctiveness of the phonological and visual-spatial working memory systems (Baddeley 1996; Michas and Henry 1994; Pickering et al. 1998; Smith et al. 1996), and larger deficits in visual-spatial relative to phonological processing observed in ADHD (Martinussen et al. 2005).

Text based (e.g., letters) and non-text based (e.g., pictures) go-stimuli were assigned to a phonological and visual-spatial grouping, respectively. Stop-stimulus modality was not examined in the analysis because nearly all studies (92%) used an auditory tone stop-signal.

**Stop-Signal Delay (SSD)** The change in stop-signal delay (SSD) methodology—initially incorporating pre-determined delay parameters, and later, a tracking algorithm

**Table 1** Stop-signal studies of between-group comparisons of ADHD and normal control children

Citation	<i>n</i>	ADHD NC	Mean ages (SD)	Diagnostic criteria	IQ	Total task time	Trials per block	Bkls	Total Trials	Go- Stim.	PH	Stop- Stim.	SSD	TD	BC	Results
Schachar and Logan (1990), Ex. 2	13 10	ADHD NC	9.3 (1.8) 10 (1.2)	Semistructured interview+ rating scales	Yes	35–45	48	9	432	PH	PH	PH	FX	25	No	MRT: ADHD=NC, SDRT: ADHD>NC, SSRT: ADHD>NC, Errors: ADHD>NC
Daugherty et al. (1993)	9 15	ADD NC	11.2 (0.9) 11.2 (1.1)	Rating scales	No	NR	48	9	432	PH	PH	PH	FX	25	No	MRT: ADD>NC, SDRT: ADD>NC, SSRT: ADD<NC, Errors: ADD<NC
Oosterlaan and Sergeant (1996)	15 17	ADHD NC	9.3 (2.1) 8.7 (1.9)	Rating scales	No	NR	64	4	256	VS	PH	PH	FX	25	No	MRT: ADHD>NC*, SDRT: ADHD>NC***, SSRT: ADHD>NC*, Errors: ADHD>NC
Pliszka et al. (1997), Ex 1	14 13	ADHD NC	7.2 (1.2) 7.5 (0.9)	Structured interview+ rating scales	No	NR	48	9	432	VS	VS	PH	FX	25	No	MRT: ADHD>NC**, SDRT: ADHD>NC*, SSRT: ADHD>NC**, Errors: ADHD>NC***
Pliszka et al. (1997), Ex 2	25 31	ADHD NC	6–12 6–12	Standard interview+ rating scales	No	NR	48	9	432	VS	VS	PH	FX	25	No	MRT: NR, SDRT: NR, SSRT: ADHD>NC**, Errors: NR
Aman et al. (1998)	22 22	ADHD NC	12.1 (1.2) 12.1 (1.2)	Structured interview+ rating scales	Yes	10	48	4	192	PH	PH	PH	FX	33	No	MRT: NR, SDRT: NR, SSRT: NR, Errors: NR
Brandeis et al. (1998)	11 11	ADHD NC	10.9 (NR) 11.2 (NR)	Rating scales	Yes	NR	40	8	320	VS	VS	VS	FX	50	No	MRT: ADHD>NC, SDRT: NR, SSRT: NR, Errors: NR
Rubia et al. (1998)	11 11	ADHD NC	9.0 9.4	Rating scales	Yes	15	60	4	240	VS	VS	VS	FX	33	No	MRT: ADHD>NC, SDRT: ADHD>NC**, SSRT: ADHD>NC*, Errors: ADHD>NC**
Nigg (1999)	25 25	ADHD NC	9.6 (1.8) 10.1 (1.3)	Structured interview+ rating scales	Yes	20	64	4	256	PH	PH	PH	TK	25	No	MRT: ADHD>NC***, SDRT: ADHD>NC**, SSRT: ADHD>NC***, Errors: ADHD>NC**
Konrad et al. (2000a)	10 10	ADHD NC	10.7 (1.3) 10.2 (1.1)	Semistructured interview+ rating scales	Yes	NR	40	8	320	VS	VS	PH	TK	25	No	MRT: ADHD>NC, SDRT: NR, SSRT: ADHD>NC***, Errors: NR
Konrad et al. (2000b)	31 26	ADHD NC	10.5 (1.6) 10.2 (1.2)	Semistructured interview+ rating scales	Yes	NR	40	8	320	VS	VS	PH	TK	25	No	MRT: ADHD>NC, SDRT: ADHD<NC, SSRT: ADHD>NC***, Errors: NR
Manassis et al. (2000)	15 16	ADHD NC	8–12 8–12	Structured interview+ rating scales	No	NR	NR	NR	NR	PH	PH	PH	TK	25	No	MRT: ADHD>NC, SDRT: ADHD>NC*, SSRT: ADHD>NC, Errors: NR
Pliszka et al. (2000)	10 10	ADHD NC	11.0 (1.2) 11.3 (0.9)	Structured interview+ rating scales	No	30	192	10	1920	PH	PH	PH	FX	25	No	MRT: ADHD<NC, SDRT: ADHD>NC**, SSRT: ADHD>NC, Errors: ADHD>NC**
Purvis and Tannock (2000)	17 17	ADHD NC	9.1 (1.1) 9.5 (1.3)	Semistructured Interview+ rating scales	Yes	NR	NR	NR	256	PH	PH	PH	TK	25	No	MRT: ADHD>NC*, SDRT: ADHD>NC***, SSRT: ADHD>NC, Errors: ADHD>NC

Table 1 (continued)

Citation	<i>n</i>	Mean ages (SD)	Diagnostic criteria	IQ	Total task time	Trials per block	Blks	Total Trials	Go-Stim.	Stop-Stim.	SSD	TD	BC	Results
Schachar et al. (2000)	72 ADHD 33 NC	9.0 (1.4) 9.3 (1.5)	Semistructured interview+ rating scales	Yes	NR	32	8	256	PH	PH	TK	25	No	MRT: ADHD>NC*, SDRT: ADHD>NC, SSRT: ADHD>NC**, Errors: NR
Kuntsi et al. (2001)	49 Hyper. 118 NC	8.8 (1.2) 9.0 (1.4)	Rating scales	Yes	NR	64	4	256	VS	PH	FX	25	No	MRT: Hyp>NC**, SDRT: Hyp>NC*** SSRT: Hyp>NC, Errors: Hyp>NC*
Scheres et al. (2001)	24 ADHD 41 NC	10.1 (1.5) 10.2 (1.6)	Rating scales	Yes	NR	32	6	192	VS	PH	TK	25	No	MRT: ADHD>NC, SDRT: ADHD>NC, SSRT: ADHD>NC, Errors: ADHD>NC
Solanto et al. (2001)	77 ADHD 29 NC	8.5 (0.9) 8.7 (0.9)	Structured interview+ rating scales	Yes	30	48	4	192	PH	PH	FX	33	No	MRT: ADHD<NC, SDRT: NR, SSRT: ADHD>NC***, Errors: ADHD>NC
Nigg et al. (2002)	46 ADHD 41 NC	9.6 (1.5) 10.1 (1.0)	Structured interview+ rating scales	Yes	20	64	4	256	PH	PH	TK	25	No	MRT: ADHD>NC, SDRT: NR, SSRT: ADHD>NC, Errors: NR
Overtom et al. (2002)	16 ADHD 16 NC	10.4 (1.4) 10.3 (1.5)	Structured interview+ rating scales	Yes	48	117	8	936	VS	PH	FX	40	Yes	MRT: ADHD>NC*, SDRT: NR, SSRT: (Overall) ADHD>NC**, (SOA 125) ADHD>NC*, (SOA 200) ADHD>NC*, Errors: ADHD>NC**
Stevens et al. (2002)	76 ADHD 76 NC	10.0 (1.6) 9.9 (1.6)	Rating scales	No	7	40	4	160	PH	PH	TK	25	No	MRT: ADHD<NC, SDRT: ADHD>NC**, SSRT: ADHD>NC*, Errors: NR
Dimoska et al. (2003)	13 ADHD 13 NC	9.8 (1.5) 9.8 (1.1)	Rating scales	Yes	NR	120	2	240	PH	PH	TK	33	No	MRT: ADHD>NC*, SDRT: ADHD>NC**, SSRT: ADHD>NC*, Errors: ADHD>NC**
McInerney and Kerns (2003)	30 ADHD 30 NC	10	Structured interview+ rating scales	Yes	NR	NR	NR	128	PH	PH	TK	25	No	MRT: ADHD=NC, SDRT: ADHD>NC***, SSRT: ADHD>NC**, Errors: ADHD>NC**
Walcott and Landau (2004)	26 ADHD 23 NC	6–11 6–11	Rating scales	No	NR	64	4	256	PH	PH	TK	NR	No	MRT: NR, SDRT: NR, SSRT: ADHD>NC***, Errors: NR
Schachar et al. (2004)	151 ADHD 41 NC	8.7 (1.7) 9.0 (1.8)	Semistructured interview+ rating scales	Yes	NR	24	4	96	PH	PH	TK	25	No	MRT: ADHD>NC*, SDRT: NR, SSRT: ADHD>NC**, Errors: ADHD<NC

All studies were between-groups comparisons of ADHD and normal control children. Target density as percentage. Total task time in minutes. *BC* Baseline control; *FX* fixed stop-signal delays; *Go-Stim.* go-stimulus modality; *IQ* intelligence quotient; *NC* normal control; *NR* not reported; *PH* phonological stimuli; *MRT* mean reaction time to the go-stimulus; *SDRT* mean reaction time variability to the go-stimulus; *SOA* stimulus onset asynchrony; *SSD* stop-signal delay; *SSRT* stop-signal delay; *Stop-Stim.* stop-stimulus modality; *TD* target density; *TK* stop-signal delay tracking method (i.e., ±50 ms); *VS* visual-spatial stimuli

\* $p < 0.05$

\*\* $p < 0.01$

\*\*\* $p < 0.001$

(Schachar and Logan 1990)—warrants scrutiny to examine whether variation among study results are partly accounted for by this uncontrolled source. The newer methodology is expected to reflect more precise and hence larger between-group ES estimates owing to its intra-individual adjustment features and control for instructional demands (Logan et al. 1997). For example, Schachar et al. (2004) found that typically developing children artificially slowed their MRT following unsuccessful stop-trials to a greater extent relative to children with ADHD, which resulted in smaller between-group BI differences. This effect is expected to be larger in studies that fail to control for artificial MRT slowing (i.e., fixed SSD studies). The inclusion of SSD as a moderator also addresses whether results can be generalized across studies using the SSD fixed and dynamic methodologies.

Studies using predetermined stop-signal delays across experimental blocks were assigned to a fixed category. Those in which stop-signal delay changed dynamically based on the child's response were assigned to a tracking category.

*Trials* The number of pre-programmed trials used in stop-signal paradigms is highly inconsistent across studies, ranging from 192 to 432 experimental trials in the Oosterlaan et al. (1998) meta-analytic review, and 96 to 1,920 (i.e., approximately 5.6 to 112 min) in more recent studies. Differences in trials indicate that task duration ranges from a few minutes to nearly 112 min depending on programmed experimental parameters.<sup>2</sup> The breadth of this parameter in published studies obscures interpretations concerning the causal nature of performance differences; specifically whether they reflect deficient BI, an inability to sustain attention (Douglas 1999; Hooks et al. 1994; Lijffijt et al. 2005), or elements of both processes.

The total number of experimental trials was analyzed as an indication of task duration due to the infrequent reporting of time data (only 8 of 24 studies included task duration data in time units). Total number of experimental trials was analyzed as a grouping variable using three categories: (1) low (<200 trials), (2) medium (200–300 trials), (3) and high (>300 trials).

*Stop-Signal Target Density* Target density refers to the proportion of trials within an experimental block that are stop-trials, and is typically reported as a percentage (i.e., percent of stop trials out of total experimental trials). Children's accuracy and reaction time show significant changes due to target density manipulations and the differential demands they place on working memory (Denney et al. 2005; Losier et al. 1996). A significant target density moderator effect would indicate that other

factors, such as increased demand on the central executive system for switching between stimuli or between phonological and visual-spatial working memory subsystems (Baddeley 1996), influence BI effects.

Stop-signal target density was examined as a grouping variable using two categories based on the median split of the target densities reported across reviewed studies (median=25, mean=27.75): low (<25%) and high (>25%).

## Materials and Methods

### Literature Searches

Searches of the stop-signal behavioral inhibition literature were conducted using the databases PSYCINFO, ERIC, MEDLINE, PsychARTICLES, and Social Science Citation Index. The following headings were used within each database: Attention, ADD, ADHD, Hyper\*, behavioral inhibition, stop-signal, stop task, go-no-go, and inhib\*. An asterisk following a root word instructs search engines to look for any derivative of the word that is followed by the asterisk (e.g., inhibit, inhibits, inhibited, inhibition). Articles located by the search engines were scrutinized for additional references relevant to the review using front- and back-search methodology until no additional references relevant to stop-signal behavioral inhibition were located.

### Inclusion Criteria

All studies included in the review compared the performance of children (age 7–12 years) with ADHD to normal controls on the stop-signal task. This age range was selected based on the well-documented developmental differences in cognitive strategies and processes observed in children relative to adolescents and adults (Lijffijt et al. 2005; Williams et al. 1999). Five additional inclusion criteria required that: (a) the primary task be a dichotomous two-choice reaction time task; (b) the inhibition response be initiated by a visual or auditory stop-signal; (c) responses to the stop-signal be measured by means of simple reaction time (i.e., change tasks were excluded); (d) participants be medication-free during the experiment; (e) participants not receive performance feedback—a condition occasionally included to examine between-group motivation differences; and (f) experimental conditions that included clearly defined comorbid disorders (e.g., ADHD and anxiety disorder).<sup>3</sup>

<sup>2</sup> Task duration could not be estimated directly owing to insufficient details reported by the studies.

<sup>3</sup> Relatively few studies included children with comorbid disorders or other ADHD subtypes, and Lijffijt et al. (2005) reported that comorbidity with ODD/CD was not a significant moderator for any of the three BI metrics.

Seventeen studies were excluded from the meta-analysis using these criteria.<sup>4</sup>

Studies that report multiple effect sizes from the same sample risk threats to statistical independence (Lipsey and Wilson 2001). Among the studies reviewed for the current meta-analysis, multiple conditions and/or experiments were reported in five studies, and these additional conditions and separate experimental conditions were omitted from the review.<sup>5</sup>

Three stop-signal studies required special consideration. One reported two experiments that included independent samples (Pliszka et al. 1997). Both experiments were included in the current meta-analysis. SSRT was calculated using the subtraction and integration methods in one study (Scheres et al. 2001), and only the subtraction method was included in the review based on a coin toss. Finally, performance data for two SSDs were reported in one study (Overtoom et al. 2002), and only one set of data was used for the review to avoid inflating effect sizes by over representing a particular sample (Lorber 2004). Collectively, 25 stop-signal studies (59% of all stop-signal studies) were included in the final sample for analyses.

#### Effect Size Estimation

Effect size (ES) estimates were computed using Comprehensive Meta-Analysis software. They reflect the magnitude of difference between children with ADHD and typically developing children. Positive and negative ESs indicate higher and lower scores for the ADHD group relative to the control group (longer MRT and SSRT, larger SDRT), respectively. Hedges' (1982)  $g$  effect sizes were used for MRT, SDRT, and SSRT to correct for the upward bias of studies with small sample size (Lipsey and Wilson 2001). The MRT–SSRT ES was computed using an unstandardized mean gain score. Effect sizes are classified as small ( $ES \leq 0.30$ ), medium ( $0.30 < ES < 0.67$ ), or large ( $ES \geq 0.67$ ), whereas an ES of zero indicates no difference between means (Lipsey and Wilson 2001). Unless otherwise specified, all ESs were computed using means, standard deviations, and sample size.

<sup>4</sup> A listing of excluded studies is available from the author.

<sup>5</sup> One study included a second experiment that examined stop-signal performance in adults (Schachar and Logan 1990). Stop-signal performance for both medicated and unmedicated children were reported in one study (Aman et al. 1998), and only the unmedicated participant results were included in the review. One study included a second condition with unconventional stop-signal delays (Rubia et al. 1998). Another study reported three additional conditions that examined the effects of reinforcement and repetition (Konrad et al. 2000a). Finally, emotional regulation was examined by means of a separate experimental condition in one study (Walcott and Landau 2004).

#### Effect Size Calculation Exceptions and Exclusions

*MRT* One study (McInerney and Kerns 2003) reported a non-significant difference between ADHD and normal controls on MRT, but did not report a specific  $p$  value. This study was assigned an effect size value of zero to avoid inflating effect size estimates and reduce the likelihood of Type I error (Rosenthal 1995). Three additional studies (Aman et al. 1998; Pliszka et al. 1997, Exp. 2; Walcott and Landau 2004) did not report sufficient data to compute effect size estimates of MRT, and were excluded from this analysis.

*MRT Variability (SDRT)* Effect size estimates for three studies (McInerney and Kerns 2003; Nigg 1999; Stevens et al. 2002) were computed using a reported  $p$  value and sample size. Eleven additional studies provided insufficient data to compute MRT variability (SDRT) effect size estimates and were excluded from this analysis.<sup>6</sup>

*SSRT* One study's effect size was estimated based on the reported means, sample size, and  $p$  value (Stevens et al. 2002). Two studies (Aman et al. 1998; Daugherty et al. 1993) provided insufficient data to compute an effect size for SSRT, and were excluded from this analysis.

*Stop-Signal Delay (SSD)* The SSD analysis included only newer tracking stop-signal studies owing to the lack of variation associated with earlier fixed stop-signal studies. A SSD between-group effect size was computed for eight tracking studies as an unstandardized mean gain with corresponding confidence intervals (Lipsey and Wilson 2001). This approach was followed because none of the studies reported SSD means or standard deviations. SSD was algebraically solved using the functional equivalent of Logan et al.'s (1997) formula:  $MRT - SSD = SSRT$ .

#### Data Analysis

*Homogeneity analyses* A  $Q$ -test was performed on each outcome variable (i.e., MRT, SDRT, SSRT, and SSD) to examine the distribution of effect sizes from the included studies. A significant  $Q$  rejects the assumption of homogeneity and supports the examination of potential moderator effects (Lipsey and Wilson 2001).

*Moderator analyses* A fixed effects weighted regression approach using SPSS for Windows 12.0 was adopted to provide a measure of overall fit ( $Q_R$ ), as well as an error/

<sup>6</sup> Excluded studies available from author.



residual term ( $Q_E$ ).<sup>7</sup> A significant  $Q_R$  indicates that the model accounts for significant variability among effect sizes. A significant  $Q_E$  indicates that the residual variance is greater than what is expected from random study-level sampling error. Both statistics are distributed as chi-square. A corrected  $B$ -weight standard error for each moderator was then tested against the  $z$ -distribution (Lipsey and Wilson 2001).

## Results

**Overall Effect Size Summary** Twenty-two studies provided sufficient information to compute effect sizes for mean reaction time (MRT). The mean effect size of MRT between ADHD and typically developing children was 0.45 (95% confidence interval=0.33–0.56), and indicates that children with ADHD have moderately slower MRTs relative to normal controls. The distribution of effect sizes was heterogeneous,  $Q(20)=42.42$ ,  $p<0.01$ , ranging from  $-0.41$  to  $1.24$ . All effect sizes fell within two standard deviations of the mean effect size for MRT, suggesting the heterogeneity was not due to outliers. A *Fail-safe N* analysis (Rosenthal 1995) indicated that an unlikely 339 studies would be needed to reduce the confidence interval of the effect size to include zero (i.e., result in no significant differences in MRT between ADHD and typically developing children).

Twelve studies provided sufficient information to compute effect sizes for MRT variability (SDRT). The mean effect size of SDRT between ADHD and typically developing children was 0.73 (95% confidence interval=0.59–0.87), and indicates that children with ADHD have more variable MRTs relative to normal controls. The distribution of effect sizes was heterogeneous,  $Q(11)=22.22$ ,  $p=0.02$ , ranging from 0.39 to 1.37. All effect sizes fell within two standard deviations of the mean effect size for SDRT, suggesting the heterogeneity was not due to outliers. The *Fail-safe N* analysis indicated that 343 studies would be needed to reduce the confidence interval of the effect size to include zero (i.e., result in no significant between-group differences).

Twenty-two studies provided sufficient information to compute effect sizes for Stop-Signal Reaction Time (SSRT). The mean medium effect size of SSRT between ADHD and typically developing children was 0.63 (95% confidence interval=0.52–0.74), and indicates that children with ADHD are on average 0.63 standard deviations slower reacting to stop signals compared to normal controls. The

distribution of effect sizes was homogeneous,  $Q(21)=32.33$ ,  $p>0.05$  (range=0.23–1.33), and all effect sizes fell within two standard deviations of the mean effect size for SSRT. The *Fail-safe N* analysis indicated that 741 studies would be needed to reduce the confidence interval of the effect size to include zero. The non-significant  $Q$ -statistic indicates that the amount of between-study variance can be attributed to random, study-level error variance, and does not support analysis of potential SSRT moderator effects.

Eight studies provided sufficient information to compute effect sizes for stop-signal delay (SSD). The mean effect size of  $-0.025$  (95% confidence interval= $-0.207$  to  $0.157$ ) indicates that children with ADHD do not differ significantly in SSD relative to typically developing children. A *Fail-safe N* analysis was not performed because the obtained confidence interval includes zero.

## Moderator Variables

**Mean Reaction Time (MRT)** The results of the weighted regression analysis indicate that the model explains a significant proportion of the variability across the MRT effect sizes,  $Q_R=180.77$ ,  $df=6$ ,  $p<0.001$ , and accounts for 41% of the variability. The moderators age ( $z=-2.78$ ,  $p=0.003$ ), diagnostic evaluation ( $z=-2.40$ ,  $p=0.008$ ), delay schedule ( $z=7.78$ ,  $p<0.001$ ), total experimental trials ( $z=2.88$ ,  $p=0.002$ ), and go-stimulus modality ( $z=4.30$ ,  $p<0.001$ ) were significant predictors of effect size variability across studies.

Younger children, the use of rating scales rather than comprehensive diagnostic procedures, newer stop-signal paradigms that dynamically alter the stop-signal delay interval based on children's ability to inhibit a response, a greater number of experimental trials, and visual-spatial rather than phonological go-stimuli, were associated with large effect sizes. Stop-signal target density was not a significant predictor of MRT. A significant sum-of-squares residual ( $Q_E=117.31$ ,  $df=12$ ,  $p<0.001$ ) was obtained, indicating that there is residual variance in the model beyond study-level sampling error even after including the six moderator variables (see Table 2). This finding indicates that there may be additional moderators other than those considered in this review that affect children's MRT.

**Mean Reaction Time Variability (SDRT)** The regression analysis indicates that the model does not explain significant variability across the SDRT effect sizes,  $Q_R=0.03$ ,  $df=6$ ,  $p>0.05$ . This finding indicates that moderator effects cannot explain the heterogeneous distribution of effect sizes. Table 2 displays a summary of the data for SDRT.

**Stop-Signal Reaction Time (SSRT)** A regression analysis of potential moderator effects on SSRT was not performed due

<sup>7</sup> The  $Q_B$  and  $Q_W$  analog to ANOVA technique reported in many meta-analytic reviews was not used for primary analyses because it inflates Type I error when used with several moderator variables—see Lipsey and Wilson (2001), for details.

**Table 2** Weighted regression model and moderating variables for MRT and SDRT

	MRT				SDRT			
	<i>Q</i>	<i>df</i>	<i>p</i>		<i>Q</i>	<i>df</i>	<i>p</i>	
Regression	180.8	6	<0.001		0.03	6	n.s.	
Residual	117.3	12	<0.001		0.01	4	n.s.	
$R^2$	0.61				0.79			
Adjusted $R^2$	0.41				0.49			
Constant	-0.14				0.53			
Moderator variables	<i>B</i>	SEB	<i>z</i>	<i>p</i>	<i>B</i>	SEB	<i>z</i>	<i>p</i>
Age	-0.14	0.05	-2.78	0.003	0.2	3.14	0.06	n.s.
Diagnostic evaluation	-0.16	0.07	-2.4	0.008	-0.22	6.56	-0.0	n.s.
Go-stimulus modality	0.3	0.07	4.3	<0.001	0.23	11.24	0.02	n.s.
Stop-signal delay	0.46	0.06	7.78	<0.001	0.24	10.54	0.02	n.s.
Target density	-0.01	0.07	-0.21	n.s.	0.19	4.28	0.05	n.s.
Total experimental trials	0.08	0.03	2.88	0.002	0.17	8.4	0.02	n.s.

*B* Regression coefficients; *df* degrees of freedom; *MRT* mean reaction time; *SDRT* mean reaction time variability; *SEB* standard error of the regression coefficients; *Q* chi-square value;  $R^2$  variance accounted for by the model; and *z* z-value

to the non-significant *Q*-statistic, which indicated that between-study variance is attributable to random, study-level error variance. Examination of moderator effects could potentially be justified a priori based on past findings; however, the non-significant *Q*-statistic, coupled with the non-significant overall effect size for SSD ( $ES=-0.025$ ), indicates that any residual variability in SSRT likely reflects systematic variability associated with MRT coupled with measurement and random error ( $SSRT=MRT-SSD$ ).

**Stop-Signal Delay (SSD)** A regression analysis to examine moderator effects on SSD was not conducted due to the non-significant overall effect size for the variable.

## Discussion

The current study updates past (Oosterlaan et al. 1998) and recent (Lijffijt et al. 2005) meta-analytic reviews, and provides a unique examination of task and subject variable moderator effects for traditionally employed stop-signal performance indices. Our results corroborate those reported in previous meta-analytic reviews (Lijffijt et al. 2005; Oosterlaan et al. 1998) in finding that children with ADHD exhibit slower and more variable reaction times to primary task stimuli (i.e., go-stimuli). The effect size estimates for these variables are remarkably consistent across reviews (i.e., MRT  $ES=0.49, 0.52, 0.45$ ; SDRT  $ES=0.73, 0.72, 0.72$  for the Oosterlaan et al. 1998, Lijffijt et al. 2005, and current study, respectively), despite the inclusion of 16 and 12 studies not considered in the past two reviews, respectively. The slower and more variable reaction times in children with ADHD is not unexpected, as these

performance outcomes are commonly observed on a wide array of standardized tests, neurocognitive tasks, and experimental paradigms (for a review, see Barkley 2005; Rapport et al. 2001). The differences have been attributed to slower cognitive processing (Kalff et al. 2005), slower motor speed (van Meel et al. 2005), deficient cognitive energetic resources (Sergeant et al. 1999), and deficient attentional processes (Lijffijt et al. 2005). The increased ADHD-related variability has also been proposed recently as a potential endophenotype of ADHD related to catecholaminergic deficiencies, and consequently tertiary symptoms such as processing/attentional deficits and careless errors (Castellanos et al. 2005).

Other factors may also contribute to the slower and more variable mean reaction times observed in ADHD. All reviewed stop-signal studies calculated children's mean reaction times to the go-stimulus (MRT) by selecting out non-stop trials within the experimental task, rather than obtaining a measure of pure motor speed uninfluenced by intermittent signals to withhold responding. Implicit to this methodology are the underlying assumptions that children's motor speed is uninfluenced by intermittent stop signals, and that children with ADHD and normal controls are similarly affected by intermittent exposure to stop signals. Previous research with adults shows that their primary reaction time is slower following successful and unsuccessful stop-trials relative to control trials (Rieger and Gauggel 1999). Moreover, Schachar et al. (2004) found that children with ADHD differentially slow their MRT following unsuccessful stop-trials relative to typically developing children. Children with ADHD also performed more poorly under intermittent relative to continuous schedules of reinforcement (Douglas and Parry 1983).

Collectively, the possibility that intermittent cues contribute to between-group differences in MRT, and indirectly to SSRT based on conventional formula ( $SSRT = MRT - SSD$ ), becomes an important consideration for future stop-signal investigations. The specific contributions of SSD and MRT to SSRT are central for quantifying the construct, and future studies may need to include uncontaminated experimental sessions for estimating children's motor reaction time independent of intermittent stop-signals.

The moderate effect size for stop-signal reaction time (SSRT) is consistent with extant literature and previous meta-analytic reviews. For example, Oosterlaan et al. (1998) and Lijffijt et al. (2005) reported SSRT effect sizes of 0.64 and 0.58, respectively, compared to an ES of 0.63 in the current review. Oosterlaan et al. (1998) interpreted their finding as evidence of deficient inhibitory control in children with ADHD relative to normal controls, but did not dissect the SSRT metric to determine the extent to which it reflected mean reaction time (MRT) relative to stopping speed differences (SSD) in ADHD. Lijffijt et al. (2005) examined SSRT–MRT between-group differences (i.e., SSD) to determine whether the SSRT effect size metric disproportionately reflected initial reaction time rather than inhibitory differences in ADHD. They reported a non-significant SSD ES (–0.22), coupled with a large MRT variability effect size, and concluded that the results reflected an underlying attention deficit rather than deficient inhibitory control. Several factors, however, may have biased the Lijffijt et al. (2005) SSD estimate. These include pooling pooled standard deviation scores, including studies that reflect motivational (i.e., reinforcement conditions) rather than inhibitory processes, and including fixed SSD with dynamic SSD tracking studies, the former of which has no associated variance and may deflate the estimate. These methodological issues were addressed in the current analyses, but did not alter the outcome. Our findings of a negative and non-significant between-group SSD effect size (–0.025) corroborates the Lijffijt et al. (2005) results, and indicates that the moderate SSRT effect size estimate reflects differences in children's mean reaction time (MRT) to go-stimuli rather than between-group differences in stopping speed.

The impact of this finding transcends stop-signal research and raises important concerns regarding the central role of behavioral inhibition in extant models of ADHD. It is noted, however, that these findings only pertain to executive-motor inhibition, while interference control and cognitive inhibition (Nigg 2001) were not addressed by the current review. Examination of other candidate endophenotypes such as working memory and response variability warrants further scrutiny, and may reveal that performance on the stop-signal task reflects processing that is downstream from other core deficits.

## Moderator Effects

Several variables served as significant moderators for mean reaction time differences between children with ADHD and typically developing children, and these findings were relatively consistent with extant literature. For example, the finding that younger children are associated with larger MRT ES estimates are consistent with lifespan and developmental studies (Bedard et al. 2002; Williams et al. 1999). Delayed motor development is commonly reported in children with ADHD, as is poorer motor coordination (Diamond 2000) and slower motor speed (Barkley 2005). The results do not appear to reflect improvements of inhibitory control given the non-significant SSD ES.

The larger effect size favoring rating scales rather than comprehensive clinical diagnostic evaluation procedures appears incongruous without considering the influence of performance variability on the ES statistical formula. Comprehensive diagnostics typically increase sensitivity and specificity for diagnostic grouping (i.e., higher rate of true positives and fewer false positives). Extant reviews have consistently revealed that children with ADHD are more variable as a group on speeded and neurocognitive tasks (Barkley 2005; Losier et al. 1996). Furthermore, direct comparisons of children with ADHD relative to children selected based on high rating scale scores (i.e., children with clinical disorders other than ADHD) reveal that children with ADHD are significantly more variable (Roberts 1990). Thus, identifying more true positives (i.e., children with ADHD) is likely to lower the effect size estimates for most speeded performance indices because it inflates the ES denominator ( $sd_{ADHD} + sd_{Control} / 2$ ). That is, although within-group diagnostic heterogeneity decreases with comprehensive diagnostic methodologies, within-group performance variability increases, consequently reducing the overall effect size magnitude.

Studies that adjusted SSD following each trial (i.e.,  $\pm 50$  ms based on the previous trial's outcome) were associated with larger MRT effect sizes relative to studies that changed SSD following a specified number of trials. Continuously adjusting the stop signal, such that children's probability of inhibiting approximates 0.50, may function to minimize the tendency of typically developing children (relative to ADHD) to slow their motor response following unsuccessful stop-trials as reported in previous studies (Schachar et al. 2004). The non-significant difference between ADHD and normal control stop-signal delay (SSD) ES estimates highlighted earlier, suggests that this effect probably reflects initial between-group differences in mean reaction time that are detected more accurately by the dynamic task. The finding also suggests that results cannot be generalized across studies using the SSD fixed and dynamic methodologies.

Larger between-group differences for MRT were also associated with greater numbers of experimental trials. This finding may reflect a greater fall-off in performance in children with ADHD over time, however, the potential interaction effect between group and performance over time could not be directly examined. A more likely explanation for the effect is that it represents the greater reliability of results associated with incorporating a larger number of trials—a common finding in the experimental literature (cf. Band et al. 2003).

Go-stimulus modality was the second strongest predictor of MRT effect size variability. This finding reflects the larger between-group differences in mean reaction time required for processing visual–spatial relative to phonological go-stimuli, and is consistent with recent findings of more pronounced deficits in visual–spatial processing in ADHD relative to typically developing children (Martinussen et al. 2005).

### Limitations

Children with clinical disorders other than ADHD and comorbid ADHD samples were intentionally excluded from the current review for three reasons. Only a handful of stop-signal studies included separate samples of carefully diagnosed psychopathological control children ( $n=10$ ), or children with comorbid disorders ( $n=6$ ). Meta-analytic findings based on such small samples may be highly unstable and thus unreliable (Rosenthal 1995). Moreover, Lijffijt et al. (2005) included comorbidity in their meta-analytic moderator analysis—despite the small number of samples available—and reported that it was not a significant moderator of mean reaction time, mean reaction time variability, or stop-signal reaction time. Finally, confirmation of a behavioral inhibition deficit in ADHD would clearly warrant comparison with appropriate psychiatric controls to ascertain whether the deficit is diagnosis-specific rather than a nonspecific effect of psychiatric diagnosis in general. Our results, coupled with the earlier Lijffijt et al. (2005) review, however, suggest a more generalized attentional or cognitive processing deficit in ADHD, and these deficiencies clearly warrant scrutiny in future investigations to determine whether they are pathognomonic of ADHD.

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