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# Selective Inhibition in Children With Attention-Deficit Hyperactivity Disorder off and on Stimulant Medication

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Selective inhibition requires discrimination between auditory signals and is assessed using a modification of the stop-signal task. Selective inhibition was assessed in a group of 59 clinic-referred, *DSM-IV*-diagnosed children with attention-deficit hyperactivity disorder (ADHD) and compared to that of a community sample of 59 children. Methylphenidate (MPH) effects on selective inhibition were assessed in a subset of the ADHD sample that participated in an acute, randomized, placebo-controlled, crossover trial with 3 fixed doses of MPH. Children with ADHD performed more poorly than controls on the majority of selective stop-signal task parameters: they exhibited more anticipatory (invalid) responses, with less accurate and more variable responses on the response execution task, as well as a slower selective inhibition process. MPH improved speed of both inhibition and response execution processes; it also reduced variability of response execution and decreased nonselective inhibition. On the one hand, findings are consistent with purported inhibition deficit in ADHD, but on the other hand, suggest that neither the impairment itself, nor MPH effects, were restricted to inhibition.

**KEY WORDS:** Attention-deficit hyperactivity disorder; selective inhibition; methylphenidate; cognitive impairment; childhood psychopathology.

Attention-deficit hyperactivity disorder (ADHD) is one of the most common developmental psychiatric disorders diagnosed in childhood. According to one current theory, the essential impairment in this disorder is a deficit involving response inhibition (Barkley, 1997). Response inhibition is part of the multidimensional construct of inhibition and is a self-generated, higher-order executive function that refers to the ability to stop (completely and suddenly) a planned course of action (Logan & Cowan, 1984). It is an important cognitive ability required in everyday life (Logan, 1994), and difficulties with response inhibition may be a potential marker for ADHD (Barkley, 1997; Schachar, Tannock, & Logan, 1993).

Deficits in this type of inhibition can be seen most clearly using the stop-signal task (Logan & Cowan, 1984), in which participants are required to intentionally inhibit their responses. Participants are engaged in a reaction time task (e.g., discriminating between visual stimuli), and occasionally, they are presented with an auditory stop signal that requires them to inhibit their response to the current stimulus. This task not only permits direct measurement of how quickly one can execute a response but more importantly provides an estimate of how quickly one can inhibit the prepotent response.

Children with ADHD have generally been found to be slower to inhibit than normal control children (e.g., Nigg, 1999; Purvis & Tannock, 2000; Schachar & Logan, 1990; Schachar, Mota, Tannock, Logan, & Klim, 2000; Schachar, Tannock, Marriott, & Logan, 1995), but there are inconsistencies in the pattern of findings that warrant

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further investigation of this type of inhibition in ADHD children. For example, several studies of inhibition have found that ADHD children are slower in both response execution and inhibition processes, suggesting that the performance decrement may reflect a general speed-of-processing deficit rather than a specific deficit in inhibition (Oosterlaan, Logan, & Sergeant, 1998; Overtoom, et al., 2002; Tannock, 1998). By contrast, others have found no differences in response execution but large differences in response inhibition (e.g., Schachar et al., 2000). Others report no differences in response inhibition but find large differences in response execution and variability in the speed of responding (Kuntsi, Oosterlaan, & Stevenson, 2001; Scheres, Oosterlaan, & Sergeant, 2001). A few studies have even demonstrated larger differences in response inhibition than those in execution (e.g., Oosterlaan et al., 1998). Also, although both Quay (1997) and Barkley (1997) have posited that deficits in motor inhibition processes are associated with the *DSM-IV* (1994) ADHD Combined subtype, differences in inhibition among the ADHD subtypes are inconsistent. For example, one study found that ADHD children of the primarily inattentive subtype were impaired in inhibition relative to control participants whereas the ADHD children of the combined subtype were not once full-scale IQ and reading achievement were controlled for (Chhabildas, Pennington, & Willcutt, 2001) whereas another found that primarily inattentive and combined subtypes did not differ on stops-signal task performance (Nigg, Blaskey, Huang-Pollock, & Rappley, 2002). Finally, it is unclear whether inhibition is specific to ADHD because deficits in inhibition have been linked with other disruptive disorders (e.g., Oosterlaan & Sergeant, 1998) and with reading disorder (Purvis & Tannock, 2000; Willcutt et al., 2001).

Stop-signal studies in ADHD research have thus far focused on nonselective inhibition whereby participants were to inhibit any and all responses whenever a stop signal occurred (e.g., Nigg, 1999; Purvis & Tannock, 2000; Schachar & Logan, 1990; Tannock, Schachar, Carr, Chajczyk, & Logan, 1989). This nonselective inhibition does not afford very sophisticated cognitive control in that all responses are shut down whenever a stop signal is presented (De Jong, Coles, & Logan, 1995). The present study takes a novel approach to the study of inhibition in children with ADHD by using a variant of the stop-signal task to measure selective inhibition. A second tone was added to the basic stop-signal task, and participants were instructed to inhibit response execution whenever presented with the designated or selected auditory tone, and continued to respond to trials when the alternative tone was presented. The second tone increased the perceptual complexity of the stop-signal task by requiring participants to discriminate between selected and nonselected auditory signals with each presentation of an auditory signal, prior to executing an inhibitory response. This selective inhibition has been demonstrated to change dynamically across the life span with a developmental trend that differs from that of response execution (Bedard et al., 2002). No studies to date have examined selective inhibition in children with ADHD.

The stimulant methylphenidate (MPH) is currently the most widely used treatment for children with ADHD, exerting pronounced effects on reducing the core behavioral symptoms (hyperactivity, impulsivity, inattentiveness; see "National Institutes of Health Consensus," 2000; Schachar, Tannock, & Cunningham, 1996). In fact, reported behavioral improvement is estimated in 65–75% of children with ADHD treated (for a review, see Greenhill et al., 2002). Currently, the primary objective of MPH treatment is aimed at management of this overt problem behavior; however, if inhibitory control underlies overt behavior, then we must investigate whether MPH targets the underlying cognitive process and not merely suppresses the overt behavioral symptoms.

Psychostimulant medication such as MPH is believed to activate self-regulatory or control processes, thereby ameliorating the fundamental inhibition deficit in children with ADHD (Barkley, 1997; Douglas, 1999). Reported stimulant trials have demonstrated empirical support for this theory. For example, MPH effects on response inhibition using the basic stop-signal task were investigated and significant speeding of the inhibitory process was found, suggesting an improvement in response inhibition (Tannock et al., 1989). In addition, because improvements in response inhibition were greater at the higher dose (1.0 mg/kg) than at the lower dose (0.3 mg/kg), the beneficial effect of MPH on response inhibition was related to dose.

By contrast, a nonlinear dose relationship was reported in Tannock, Schachar, and Logan (1995). This latter study used a more complicated version of the basic stop-signal task (change task) that required children to inhibit their response to a primary task and immediately execute a response to a secondary task when given a signal to do so. A separate interesting finding in both of these aforementioned studies is the evidence of concomitant improvement in aspects of performance (i.e., response execution speed) other than that of response inhibition with MPH. This suggests that perhaps effects not specific to inhibition were occurring or that stimulants enhanced an underlying mechanism common to both response inhibition and execution.

In this study, the primary objectives were to determine whether children with ADHD exhibited deficient

selective inhibition and whether MPH enhanced selective inhibition in children with ADHD. A pronounced deficit in selective inhibition in children with ADHD in comparison to community controls was expected because the stop-signal task was made more perceptually complex than it has been in previous research. Specifically, an effect size ($d$) of at least 0.6 reported in a meta-analysis of stop-signal-task studies in ADHD populations (Oosterlaan & Sergeant, 1998) was predicted. Similarly, because of the added complexity of the selective inhibition process, it was predicted that performance on the selective stop-signal task would discriminate between ADHD subtypes (i.e., the Combined subtype would evidence greater deficits in inhibition than the primarily Inattentive subtype). Also, because the results of previous studies examining stimulant effects on nonselective inhibition have demonstrated global improvements in response inhibition and execution with MPH, similar results are predicted with stimulant-influenced performance on the selective inhibition task.

## METHOD

### Participants

The ADHD sample consisted of 65 children who were referred for the assessment of problems related to attention, behavior, and learning to an outpatient neuropsychiatry clinic in an urban, pediatric hospital. Exclusionary criteria included a full-scale intelligence quotient (FSIQ) score of fewer than 80, any evidence of neurological dysfunction, poor physical health, uncorrected sensory impairments, or a history of psychosis. Of these individuals, 6 (9%) were excluded from analyses because of extreme scores (3 or more standard deviations from the mean) on the two primary outcome variables (4 for stop-signal reaction time [SSRT] and 2 for go-signal reaction time [GoRT]). The remaining sample consisted of 59 *DSM-IV*-diagnosed children with ADHD (50 boys, 9 girls) ranging in age from 6.4 to 12.9 years ($M = 8.7$, $SD = 1.4$); 15 (25%) of these children were subtyped as Predominantly Inattentive, 8 (14%) as Predominantly Hyperactive/Impulsive, and 36 (61%) as Combined. Seventeen (29%) participants were classified as having a concurrent reading disorder, 9 (15%) were diagnosed with a comorbid conduct disorder, and 25 (44%) were identified as having a comorbid oppositional defiant disorder. The clinical characteristics of the different ADHD subtypes are presented in Table I.

Data from community control comparison children were derived from a large sample in an earlier study of selective inhibition across the life span (Bedard et al., 2002). In that study, 317 participants, aged 6–82 years, were tested individually at an urban science museum over a 2-week period. Participants who volunteered to participate in this study were recruited through flyers distributed at the science museum and received a certificate for their participation. From the 102 children aged 6–12 years tested throughout that period, 59 children were selected to match the clinical ADHD sample case by case on the basis of age (and gender where possible). In situations in which a clinical participant could be matched with more than one child in the community sample, the matched pair was constructed by random selection among the potential matches. This community sample consisted of 37 boys and 22 girls ranging in age from 6.4 to 12.1 years ($M = 8.9$, $SD = 1.5$). Both the community-based and the ADHD samples were predominantly Caucasian (i.e., 90% of both samples) with the remaining sample comprising of Black, Hispanic, and Asian participants. Data collection of the community control and clinical samples took place concurrently.

Stimulant effects on selective inhibition were examined in a subsample ($N = 28$) of the children with ADHD described earlier (26 boys, 2 girls) ranging in age from 6.4 to 12.0 years ($M = 8.9$, $SD = 1.4$); 10 (36%) of these children were subtyped as Predominantly Inattentive, 4 (14%) as Predominantly Hyperactive/Impulsive, and 14 (50%) as Combined. Five (18%) participants were classified as having a concurrent reading disorder, 3 (12%)

**Table I.** Description of ADHD Sample by *DSM-IV* ADHD Subtype

| Sample characteristic | Inattentive ($n = 15$) | Hyperactive/ impulsive ($n = 8$) | Combined ($n = 36$) |
|---|---|---|---|
| Age, mean (*SD*) | 8.0 (0.9) | 7.5 (1.4) | 8.4 (1.6) |
| % Females | 20 | 25 | 14 |
| Full Scale IQ, mean (SD)[a] | 103 (12.4) | 98.4 (6.6) | 107 (12.2) |
| Teacher-based:[b] | | | |
| # Inattentive symptoms | 6.4 (1.3) | 3.5 (1.9) | 5.9 (1.6) |
| # Hyperactivity/impulsive symptoms | 2.9 (1.2) | 4.1 (3.1) | 5.0 (2.4) |
| Parent-based:[b] | | | |
| # Inattentive symptoms | 5.6 (1.8) | 4.1 (1.0) | 6.2 (1.6) |
| # Hyperactivity/impulsive symptoms | 4.4 (2.1) | 6.5 (1.9) | 6.4 (2.0) |
| Comorbid diagnoses (% participants) | | | |
| Reading disability | 27 | 38 | 28 |
| Conduct disorder[b] | 14 | 0 | 20 |
| Oppositional defiant disorder[b] | 36 | 50 | 46 |

*Note.* Some comorbidity data were unavailable.

[a] Data from 1 inattentive child, 1 hyperactive/impulsive child, and 1 combined child missing.

[b] Data from 1 inattentive child and 1 combined child missing.

were diagnosed with a comorbid conduct disorder, and 13 (50%) were identified as having a comorbid oppositional defiant disorder. Conduct disorder and oppositional defiant disorder diagnoses were unavailable for two of the children that participated in the medication trial. These children were either specifically referred for evaluation of their responses to stimulant treatment or had stimulant medication been recommended by the clinical diagnostic team (i.e., all children participating in this MPH trial would have received MPH independent of this study).

**Diagnostic Assessment**

Clinical diagnosis of ADHD, using *DSM-IV* criteria, was based upon information from semi-structured interviews conducted with parents (Parent Interview for Child Symptoms—IV [PICS]; Ickowicz et al., 2002) and the child's classroom teacher (Teacher Telephone Interview-IV [TTI-IV]; Tannock, Hum, Masellis, Humphries, & Schachar, 2002). In addition, parents, teachers and children completed various standardized rating scales (e.g., Conners' Parent [CPRS-R] and Teacher [CTRS-R] Rating Scales—Revised; Conners, 1997) to provide supportive information.

Each case was reviewed by the clinical team to arrive at a consensus diagnosis. Interviews were conducted independently by separate trained clinicians who were blind to other aspects of the child's assessment.Both interviews require the clinician rather than the informant to rate the presence and severity of each symptom, based upon descriptives elicited from the informant of the child's behavior in prescribed contexts, using prespecified scoring criteria. Individual symptoms were rated on a 4-point (i.e, 0–3 range) rating scale. Clinician ratings for individual symptoms had to exceed a threshold value of "2" to be regarded as an impairing symptom. Reliability and validity for the *DSM-III-R* version of both interviews is high (Schachar et al., 1995); evaluation of the psychometric properties of the *DSM-IV* versions is under way. Preliminary analysis on the *DSM-IV* version of PICS indicates the kappa statistic for the ADHD diagnosis on PICS was 0.84 for 32 cases, 0.80 for ODD (oppositional defiant disorder) diagnosis, and 0.73 for CD (conduct disorder) diagnosis. Kappas for individual PICS symptoms ranged from a low of 0.51 for "avoids work" to a high of 1.00 for "waits turn," "quiet play," and "intrudes." The intraclass correlation coefficients for total number of inattentive symptoms on PICS were 0.93 and 0.97 for total number of hyperactive/impulsive symptoms. Preliminary analyses on the *DSM-IV* version of the TTI-IV based on 10 interviews resulted in interrater reliability on a symptom level ranging from 75 to 100% for the ADHD symptoms.

Among other measures, the Wechsler Intelligence Scale for Children—Third Edition (WISC-III; Wechsler, 1991), the reading subtest of the Wide Range Achievement Test—Third Edition (WRAT3; Wilkinson, 1993), and the Word Attack and Word Identification subtests of the Woodcock Reading Mastery Test—Revised (WRMT-R; Woodcock, 1987) were administered during the initial assessment session. In the event that a psychologist had administered these tests within the past year, those results were obtained with consent from parents.

The *DSM-IV* does not specify an algorithm for combining information across informants. Accordingly, in this study the following "6/4" algorithm was used to classify ADHD subtype. The Inattentive subtype required at least six symptoms of inattentiveness on PICS or TTI-IV, with fewer than six symptoms of hyperactivity–impulsivity on both PICS and TTI-IV plus evidence of pervasiveness of symptomatology. Pervasiveness is defined operationally in this study as at least four symptoms of either inattentiveness or hyperactivity–impulsivity endorsed on each interview (i.e., a child could not receive a diagnosis of ADHD based on symptomatology restricted to home or school settings only). The Hyperactive–Impulsive subtype required at least six symptoms of hyperactivity–impulsivity on the PICS and/or TTI-IV, with fewer than six symptoms of inattentiveness on PICS or TTI-IV. The Combined subtype required at least six symptoms of inattentiveness plus six symptoms of hyperactivity–impulsivity on the PICS and/or TTI-IV, plus evidence of pervasiveness of symptomatology. Each child's diagnostic profile as defined by the preceding research criteria was confirmed by a child psychiatrist, on the basis of clinical review of all of the information gathered during the assessment.

Children were categorized into those with and without reading disorder (RD). We used an IQ-nondiscrepant definition of decoding problems, because extensive research has shown that both IQ-discrepant and IQ-nondiscrepant definitions validly identify children as reading disabled, with little evidence that these definitions differ in chronicity of problems (Fletcher, Francis, Shaywitz, & Lyon, 1998; Shaywitz, Fletcher, Holahan, & Shaywitz, 1992). RD was assessed using a definition of low achievement in standardized tests of single word and nonword reading (WRMT-R Word Attack, Word Identification, WRAT-3 Reading; Fletcher et al., 1998). RD was defined by scores of at least 1.5(*SD*) below the mean for age on at least one of the three tests or if scores were at least 1.0(*SD*) below the mean for age on at least two of the three tests. Diagnoses of CD and ODD were based on information from PICS and TTI-IV interviews with *DSM-IV* criteria.

## The Selective Stop-Signal Task

### *Apparatus and Stimuli*

A stand-alone, desktop computer was used to present the stimuli. Attached to the computer was a pair of adjustable padded headphones through which two distinct auditory signals could be presented without hindrance from potential background noise. In addition, the computer was connected to a handheld response box (14 cm × 8.5 cm × 3.5 cm) that contained three single-pole double-throw buttons. These buttons were arranged on the top of the box in a line formation with the two outermost buttons individually labeled with the visual stimuli for the go task.

The visual stimuli for the go task were the uppercase letters "X" and "O", presented in the center of the screen for 1000 ms. Each go-task stimulus was preceded by a 500-ms fixation point, also presented in the center of the screen. Two 500-ms auditory tones (1000, 250 Hz) were generated by the computer, each presented randomly on approximately 20% of trials and delivered through headphones at a comfortable volume for listening. One of these two tones was designated as the selected auditory signal; the nonselected auditory signal was to be ignored. The stop-signal delay (i.e., the interval between the presentation of the go signal and the selected auditory, i.e., stop, signal) was changed dynamically in 50-ms intervals after each selected stop-signal trial based on the performance of the participant (Logan, Schachar, & Tannock, 1997). Stop-signal delay was initially set at 250 ms and adjusted in the following manner: The stop-signal delay increased by 50 ms if the participant inhibited successfully to the selected auditory signal (making it harder to inhibit on the next selected stop-signal trial) and decreased by 50 ms if the participant failed to inhibit (making it easier to inhibit on the next selected stop-signal trial). This online tracking system of success in inhibition was designed to force a "tie" finish between response execution and response inhibition. Thus, the goal of the tracking algorithm was to allow participants to successfully inhibit responding to the go task on approximately 50% of the selected stop-signal trials. This was necessary for the estimation of SSRT (see Appendix of Williams, Ponesse, Schachar, Logan, & Tannock, 1999). The nonselected auditory signal was fixed (i.e., constantly presented at the same rate of 250 ms) in contrast to the dynamic nature of the selected auditory signal. Mean response-execution speed (i.e., GoRT) was calculated on the basis of the response speeds during those trials in which no auditory tone (both selected and nonselected) was presented, following standard practice (e.g., Bedard et al., 2002; Logan et al. 1997; Logan & Burkell, 1986; Logan & Cowan, 1984; Osman, Kornblum, & Meyer, 1990; Schachar et al., 2000).

The experimental task comprised 192 trials divided into six 32-trial blocks. There were an equal number of "X"s and "O"s presented in each block. The auditory tone stimuli (1000, 250 Hz tone) were presented on 12 (i.e., 38%) of the response execution trials (distributed randomly in each block of 32 trials): 6 (19%) were 1000-Hz and 6 (19%) were 250-Hz tones. Each of the auditory signals was presented half of the time with an "X" and half of the time with an "O." The order in which the trials were presented was randomized separately for each participant. Once started, the program ran continuously presenting one trial every 3.5 s. Measures of SSRT and GoRT were the primary outcome measures for this task.

### *Administration Procedure*

The study was approved by the institutional ethics review board. Parents of all participants gave written informed consent for their children to participate in the study and all participating children gave verbal informed assent. Children were tested individually. The experimenter remained in the testing room with the participant, read a uniform set of instructions, operated the computer, and monitored the participant's progress from start to completion of the computer task (approximately 20 min in length). Each participant completed one practice block before commencing the six test blocks. Participants were told that they would see a fixation point followed by one of two letters ("X" or "O") and that their task was to respond to the letter (by pressing the appropriate response button) as quickly as possible without making mistakes. Also, they were told that although they were to respond to the presented letters as quickly as possible, when the selected auditory signal was presented they were to attempt to stop their response during that given trial. They were instructed not to wait for the auditory signals as they occurred unpredictably. GoRT was displayed at the end of the practice block. The selection of the designated auditory signal was counterbalanced so that approximately an equal number of participants in both the clinical and the normal control groups inhibited selectively to the high tone and to the low tone. The examiners testing the children with ADHD were blind to child diagnosis and study hypotheses.

### *Drug Protocol*

A total of 28 children participated in a 5-day randomized double-blind placebo controlled crossover trial of MPH conducted in a pediatric hospital laboratory. Testing

occurred over a period of five consecutive days, Monday through Friday, for approximately 3 hr per session. In each session, participants completed the selective stop-signal task and a variety of other cognitive and academic measures (not reported here).

After baseline measures were obtained on the 1st day ("practice day"), each child received each of three fixed doses of MPH (5, 10, and 15 mg for children who weighed equal to or less than 25 kg; 10, 15, and 20 mg for those who weighed over 25 kg) and a placebo dose. Fixed doses of MPH were used because there is no clear evidence that response to medication is dependent on body weight (Rapport & Denney, 1997). This translated to the following mean milligram per kilogram for each of the MPH dose levels: low ($X = 0.29$, $SD = 0.08$); medium ($X = 0.45$, $SD = 0.10$); high ($X = 0.61, SD = 0.14$). The doses were administered in a counterbalanced order so that approximately equal numbers of children received each of the possible drug condition orders. The two exceptions to this rule were that no directly ascending (i.e. P L M H) or descending (H M L P) medication orders were permitted because they would have made it difficult to interpret drug effects for the individual child. The examiner, psychiatrist, child, and child's family were unaware of the medication condition for each trial day until trial completion. Placebo and active medication was prepared by the hospital pharmacist, powdered, and packaged in an opaque gelatin capsule to prevent identification of contents by color, taste, or volume. Each child's medication was placed in an individually named and dated envelope and administered by the research staff to ensure accurate administration. The selective stop-signal task was administered 2 hr after ingesting the capsule containing MPH or placebo. The letters (i.e., response execution visual stimuli) presented on the screen varied for each day of the medication trial (Day 1: F D, Day 2: K R, Day 3: E P, Day 4: S Z, Day 5: C H) to minimize any potential practice effects on the response execution task. Also, the selected auditory signal was altered from participant to participant so that an equal number of participants were instructed to selectively inhibit to the high (1000 Hz) and low (250 Hz) tones, respectively (15 inhibited to the high tone and 13 to the low tone). The designated auditory signal for each individual was kept constant across the 5 days of the medication trial.

### *Statistical Analyses*

Data from the first block of the selective stop-signal task was excluded, leaving five test blocks in the analyses because of the number of trials required by the selective stop-signal task to adjust the stop-signal delay to the point where the participant is successfully inhibiting on approximately 50% of selected stop-signal trials. The total number of trials in which an early anticipatory (invalid) response (i.e., a response within 200 ms of the onset of each response trial) was computed and then excluded from further analyses. These anticipatory responses could occur on either response execution or response inhibition trials. An examination of the stability of performance in SSRT, GoRT, and within-participant variability in GoRT (SDGoRT) across the five experimental blocks was conducted as a reliability check of the data obtained by the selective stop-signal task.

Multivariate and univariate analyses of variance (MANOVA and ANOVA) were used to examine group and gender differences on variables from the selective stop-signal task. The Wilks $\lambda$ was used as the overall test of significance ($p < .05$). Significant differences in any of the dependent variables were further examined by calculations of effect size (Cohen's $d$). Chi-square analyses were used for group comparison of dichotomous variables for the children with ADHD versus community controls. Performance on the selective stop-signal task between the ADHD *DSM-IV* subtypes was examined using a MANOVA followed by measures of estimated effect size, as calculated by $\eta^2$. Supplementary analyses included a comparison of ADHD subgroups defined by comorbidity (ADHD vs. ADHD + RD; ADHD vs. ADHD + ODD/CD) on performance measures of the selective stop-signal task using independent samples $t$ tests. Also, zero-order correlations (Pearson product–moment correlations) were conducted to examine the relationship between FSIQ and performance on the selective stop-signal task.These supplementary analyses were performed for the ADHD group because relevant data on FSIQ and behavioral ratings were not available for the community sample.

A repeated-measures MANOVA was conducted to examine the effects of MPH on performance on the selective stop-signal task. All dependent variables from the selective stop-signal task were entered, with dose (four levels) as the repeated measure. Trend analyses followed to determine the relationship between performance variables and overall MPH dose and post hoc Sidak pairwise comparisons were conducted to examine significant differences between specific dose levels.

## RESULTS

### Preliminary Checks on Data and the Race Model

The novel application of the selective stop-signal task was successful. For the sample as a whole (59 children

with ADHD, 59 community controls), the percent inhibition given the selected auditory signal was 46 and the percent inhibition given the nonselected auditory signal was 7. This indicates that the sample as a whole was able to successfully discriminate between auditory signals, and successfully inhibit to the selected auditory signal. Also, overall mean accuracy in response execution was 90.5%, demonstrating that the children were able to match their response to the stimuli presented. Reliability over three blocks was consistently high, with $\alpha = .93$ for SSRT, $\alpha = .95$ for GoRT, and $\alpha = .80$ for SDGoRT. Lastly, response-execution speeds for the no-signal and nonselected auditory signals were examined for adherence to the race model of stop-signal task inhibition (please see Appendix).

## Selective Inhibition in Children With ADHD Versus Community Controls

Performance on the primary outcome measures of the selective stop-signal task by the children with ADHD versus the community control group was significantly different (Wilks' $\lambda$: $F = 4.19$, $p < .001$) and is summarized as Table II. In comparison to the community control group, children with ADHD had a significantly higher percentage of invalid anticipatory responses (% EARLY) (~6% of total presented trials) than did matched community controls (<1% of total presented trials). Also, the children with ADHD had significantly poorer selective inhibition, as demonstrated by a mean SSRT 120 ms slower than that of the community controls. Mean GoRT, however, did not differ significantly between the groups. Other aspects of performance were significantly worse in the ADHD group than in the community controls, including impaired go task accuracy (% CGR), a greater variability in response execution speed (SDGoRT), and a poorer ability to inhibit to the selected auditory signal [P(I/S)]. Although the mean percent inhibition to the selected auditory signal [P(I/S)] differed significantly from 0.5 in both groups of participants, very few participants in any group produced values of P(I/S) that were significantly different from 0.5 when tested individually with a binomial test. We computed the 95% confidence interval for P(I/S) = 0.5 and reanalyzed data excluding participants whose P(I/S) values fell outside of the 95% confidence interval. The pattern of results was the same as that in the full sample. Lastly, the mean difference in response inhibition and execution speeds (calculated by subtracting mean SSRT from mean GoRT) was much larger for the community controls (SSRT 180 ms faster than GoRT) than for the ADHD group (SSRT only ~40 ms faster than GoRT).

To further examine the specificity of a selective inhibition deficit in ADHD children, we used a categorical approach to determine inhibition deficits in ADHD. Impairment in selective inhibition was defined as a mean SSRT greater than one standard deviation above that for the comparison community sample. One third (36%,

**Table II.** Mean Scores (+*SD*) for Performance on the Selective Stop-Signal Task for the ADHD Sample and Matched Community Controls

| Variable | Control group ($N = 59$) | | ADHD group ($N = 59$) | | Group difference ($p$) | Effect size ($d$) |
|---|---|---|---|---|---|---|
| | *M* | *SD* | *M* | *SD* | | |
| % EARLY[a] | 0.9 | 1.6 | 5.5 | 8.8 | .001 | 0.73 |
| Response inhibition | | | | | | |
| SSRT (ms) | 402.7 | 186.7 | 524.0 | 235.3 | .002 | 0.57 |
| P(I/S) | 48.8 | 9.6 | 43.0 | 10.7 | .002 | 0.57 |
| P(I/N) | 5.2 | 11.3 | 9.0 | 14.4 | .11 | 0.30 |
| Response execution | | | | | | |
| GoRT (ms) | 586.8 | 222.6 | 566.8 | 157.6 | .58 | 0.10 |
| SDGoRT (ms) | 170.6 | 69.5 | 223.1 | 92.8 | .001 | 0.65 |
| % CGR | 92.3 | 6.3 | 86.3 | 9.3 | <.001 | 0.76 |

*Note.* % EARLY = percentage of early (invalid) responses (calculated out of the total 192 trials); SSRT = stop-signal reaction time (ms); GoRT = go-signal reaction time (ms); SDGoRT = standard deviation of go-signal reaction time (ms); P(I/S) = percent inhibition given the selected auditory signal; P(I/N) = percent inhibition given the nonselected auditory signal; % CGR = accuracy of go task responding as percentage of correct go-signal responses; NB – SDGoRT is a within-participant measure (trial-to-trial variability); by contrast, the *SD* of GoRT refers to between-participant differences in mean GoRT.

[a] Early responses may occur on any trial (i.e., those with and without a stop signal) and are excluded from all analyses and interpretation of GoRT, SSRT, SDGoRT, etc.

**Table III.** Mean Scores (+*SD*) for Performance on the Selective Stop-Signal Task for the ADHD Sample by Comorbid Diagnoses

| | Presence/absence of RD | | | Presence/absence of ODD or CD[a] | | |
|---|---|---|---|---|---|---|
| | ADHD + RD ($N = 17$) | ADHD − RD ($N = 42$) | Group difference, $F(1, 58)$ | ADHD + CD/ODD ($N = 34$) | ADHD − CD/ODD ($N = 23$) | Group difference, $F(1, 56)$ |
| % Early | 6.6 (10.5) | 5.0 (8.1) | 0.43 | 6.5 (9.4) | 3.5 (7.8) | 1.46 |
| Response inhibition | | | | | | |
| SSRT (ms) | 517.6 (230.2) | 526.5 (240.0) | 0.02 | 483.2 (200.5) | 567.8 (278.5) | 1.78 |
| P(I/S) | 46.7 (8.1) | 41.4 (11.3) | 3.00 | 43.4 (9.9) | 43.4 (11.7) | 0.00 |
| P(I/N) | 10.8 (15.8) | 8.3 (13.9) | 0.36 | 9.2 (13.5) | 9.3 (16.5) | 0.001 |
| Response Execution | | | | | | |
| GoRT (ms) | 613.4 (126.6) | 548.0 (166.2) | 2.12 | 562.4 (181.4) | 579.8 (119.4) | 0.16 |
| SDGoRT (ms) | 240.1 (104.9) | 216.2 (87.8) | 0.80 | 225.9 (99.9) | 219.7 (84.9) | 0.06 |
| % CGR | 86.9 (12.5) | 86.0 (7.7) | 0.12 | 85.7 (10.3) | 87.2 (7.9) | 0.38 |

*Note.* % EARLY = percentage of early (invalid) responses (calculated out of the total 192 trials); SSRT = stop-signal reaction time (ms); GoRT = go-signal reaction time (ms); SDGoRT = standard deviation of go-signal reaction time (ms); P(I/S) = percent inhibition given the selected auditory signal; P(I/N) = percent inhibition given the nonselected auditory signal; % CGR = accuracy of go task responding as percentage of correct go-signal responses. Values represent mean (standard deviation).
[a]Comorbidity diagnoses from two children are unavailable.

$N = 21$) of the children with ADHD exhibited an SSRT that was at least one standard deviation above the mean for the age matched normal group; none exhibited an SSRT greater than 1.5 standard deviation above the mean for age.

There were no differences between the ADHD group with and without comorbid RD or between the ADHD group with and without comorbid ODD or CD on any of the dependent variables of the selective stop-signal task (Table III). Also, FSIQ did not correlate with any measures of the selective stop-signal task for the children with ADHD.

### Selective Inhibition Across the ADHD Subtypes

The clinical characteristics of the children within each of the three ADHD subtypes are reported in Table I. Mean scores, significance values, and effect sizes of the selective stop-signal task outcome variables among the three ADHD subtypes are presented in Table IV. As this table indicates, no statistically significant differences among subtypes were found on any of the outcome measures.

### MPH Effects on Selective Inhibition

The means and standard deviations for the dependent variables of the selective stop-signal task obtained for each of the three active treatment conditions and placebo are presented in Table V. In addition, mean scores on the selective stop-signal task during baseline ("practice") day are also presented for comparison purposes in Table V (baseline values were not included in subsequent analyses).

**Table IV.** Mean Scores (+SD) for Performance on the Selective Stop-Signal Task for the ADHD Sample by *DSM-IV* ADHD Subtype

| | Inattentive ($N = 15$) | | Hyperactive/impulsive ($N = 8$) | | Combined ($N = 36$) | | Group difference, | | Effect |
|---|---|---|---|---|---|---|---|---|---|
| Variable | *M* | *SD* | *M* | *SD* | *M* | *SD* | $F(2, 56)$ | *p* | size ($\eta^2$) |
| % EARLY | 4.7 | 5.7 | 8.9 | 12.7 | 5.1 | 9.0 | 0.66 | .52 | 0.02 |
| Response inhibition | | | | | | | | | |
| SSRT (ms) | 480 | 277 | 403 | 186 | 569 | 219 | 2.04 | .14 | 0.07 |
| P(I/S) | 43.0 | 11.5 | 48.2 | 5.0 | 41.8 | 11.1 | 1.19 | .31 | 0.04 |
| P(I/N) | 6.0 | 7.7 | 18.3 | 25.5 | 8.2 | 12.9 | 2.13 | .13 | 0.07 |
| Response execution | | | | | | | | | |
| GoRT (ms) | 545 | 144 | 604 | 106 | 568 | 174 | 0.35 | .70 | 0.01 |
| SDGoRT (ms) | 211 | 90 | 232 | 95 | 226 | 96 | 0.17 | .85 | 0.01 |
| % CGR | 85.9 | 8.7 | 88.0 | 6.3 | 86.3 | 9.3 | 0.15 | .86 | 0.01 |

*Note.* Nonparametric Kruskal–Wallis Test analyses showed nonsignificant differences between the ADHD subtypes across all performance variables. % EARLY = percentage of early (invalid) responses (calculated out of the total 192 trials); SSRT = stop-signal reaction time (ms); GoRT = go-signal reaction time (ms); SDGoRT = standard deviation of go-signal reaction time (ms); P(I/S) = percent inhibition given the selected auditory signal; P(I/N) = percent inhibition given the nonselected auditory signal; % CGR = accuracy of go task responding as percentage of correct go-signal responses.

**Table V.** Means, Standard Deviations, and Repeated-Measures ANOVA Results for the Primary Dependent Variables Measured on the Four Drug Days

| | Drug dose | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Dependent variable | Baseline (Day 1) | Placebo | Low | Medium | High | ANOVA ($F$) | Result ($p$) | Effect size ($\eta^2$) |
| % EARLY | 20.6 (21.2) | 12.1 (12.3) | 11.3 (18.1) | 8.0 (10.8) | 8.4 (11.4) | 1.58 | .215 | 0.055 |
| Response inhibition | | | | | | | | |
| SSRT (ms) | 533 (229) | 578 (314) | 426 (234) | 483 (221) | 466 (222) | 5.22 | .006 | 0.162 |
| P(I/S) | 43.4 (10.1) | 42.0 (10.5) | 41.2 (12) | 43.1 (7.9) | 44.7 (10.0) | 1.05 | .364 | 0.038 |
| P(I/N) | 13.3 (16.0) | 15.4 (18.7) | 7.9 (14.9) | 7.8 (12.7) | 6.0 (10.6) | 3.83 | .028 | 0.128 |
| Response execution | | | | | | | | |
| GoRT (ms) | 509 (107) | 548 (140) | 469 (163) | 480 (115) | 476 (143) | 5.87 | .003 | 0.179 |
| SDGoRT (ms) | 226 (103) | 275 (150) | 189 (143) | 174 (83) | 156 (69) | 12.10 | .001 | 0.309 |
| % CGR | 82.8 (8.0) | 77.3 (12.2) | 80.3 (13.9) | 81.5 (12.4) | 82.3 (13.8) | 2.75 | .064 | 0.092 |

*Note.* NB – data from baseline (Day 1) provided for comparison purposes only and is not included in MPH analyses.

Trend analyses results and post hoc dose level comparisons between placebo and the three active treatment conditions are presented in Table VI. MPH had no effect in reducing the percentage of early (invalid) responses (% EARLY), which remained high (ranging from ∼8% to ∼12%) across all trial days.

MPH had an overall effect of accelerating the inhibitory process ($F = 5.22$, $p < .01$). Trend analysis revealed significant quadratic and cubic dose–response trends (Table VI). At low dose, the inhibitory process was approximately 150 ms faster than at placebo and 50 ms faster than the mean response inhibition latency of medium and high doses combined. Under the effect of medium and high doses of MPH, mean SSRT remained approximately 100 ms faster than that of placebo demonstrating marked improvements in response inhibition latency across all of the drug doses when compared to placebo. Post hoc dose level comparisons revealed significant differences between placebo and low dose, and between placebo and high dose (Table VI).

The percent inhibition given the selected auditory signal [P(I/S)], did not significantly improve with medication, remaining stable across drug doses (between 41 and 44%). However, MPH was shown to improve an additional aspect of selective inhibition performance: the ability to continue to respond to the go stimuli despite the presentation of the nonselected (i.e., distracter) auditory signal [P(I/N)]. P(I/N) was relatively high at placebo (15%) and significantly decreased with MPH (to levels of 8% at both low and medium and 6% at high), best fitting a linear dose–response trend ($F = 6.11$, $p = .02$). Post hoc comparisons revealed significant differences between placebo and all three drug doses (Table VI).

Beneficial effects of MPH on response execution measures were also observed (Table V). Of primary focus, MPH was found to significantly increase speed of response

**Table VI.** Analysis of Trend for MPH Effects on Primary Dependent Variables

| | $F$ value | | | Effect size ($\eta^2$) | | | |
|---|---|---|---|---|---|---|---|
| Dependent variable | Linear | Quadratic | Cubic | Linear | Quadratic | Cubic | Post hoc dose comparison |
| Early responses (%) | 6.57* | 0.19 | 0.40 | 0.196 | 0.007 | 0.015 | P > M* |
| Response inhibition | | | | | | | |
| SSRT (ms) | 3.92 | 6.61* | 5.68* | 0.127 | 0.197 | 0.174 | P > L**, H* |
| P(I/S) | 2.28 | 0.05 | 0.04 | 0.078 | 0.018 | 0.013 | |
| P(I/N) | 6.11* | 2.59 | 1.72 | 0.185 | 0.087 | 0.060 | P > L*, M*, H** |
| Response execution | | | | | | | |
| GoRT (ms) | 6.39* | 11.68** | 2.38 | 0.191 | 0.302 | 0.080 | P > L***, M***, H** |
| SDGoRT (ms) | 19.01*** | 8.36** | 1.52 | 0.413 | 0.237 | 0.054 | P > L***, M***, H*** |
| % CGR | 7.23* | 1.06 | 0.049 | 0.211 | 0.038 | 0.002 | P > L*, H** |

*Note.* P = placebo, L = low, M = medium, H = high. % EARLY = percentage of early (invalid) responses (calculated out of the total 192 trials); SSRT = stop-signal reaction time (ms); GoRT = go-signal reaction time (ms); SDGoRT = standard deviation of go-signal reaction time (ms); P(I/S) = percent inhibition given the selected auditory signal; P(I/N) = percent inhibition given the nonselected auditory signal; % CGR = accuracy of go-task responding as percentage of correct go-signal responses.
* $p < .05$. ** $p < .01$. *** $p < .001$.

execution (GoRT) and reduce variability of response execution speed (SDGoRT). At placebo dose, mean GoRT was 548 ms and it improved with MPH by a range of 68 ms (medium) to 80 ms (low). The improvements in GoRT with MPH best fit linear and quadratic functions (Table VI) and post hoc analyses revealed significant differences between placebo and all three drug doses (Table VI). Similarly, mean SDGoRT was 275 ms at placebo and improved (i.e. decreased) by a range of 86 ms (low) to 119 ms (high), best fitting linear and quadratic dose respond trends as well (Table VI). Also, significant differences in SDGoRT were found between placebo and all three drug doses (Table VI).

The mean difference between stopping (SSRT) and going (GoRT) latencies did not appear to increase with drug, remaining similar across the drug days (ranged from 3 to 43 ms).

## DISCUSSION

This is the first study to examine selective inhibition in children with ADHD using a novel experimental manipulation of the stop-signal task. The primary findings from the study are threefold: (1) children with ADHD demonstrated impairments in selective inhibitory control compared to matched community controls, (2) there was no clear evidence that selective inhibition differed among the *DSM-IV* ADHD subtypes, and (3) MPH improved selective inhibition in children with ADHD.

On average, children with ADHD were 120 ms slower to selectively inhibit than community controls. The effect size of this difference in inhibition speed ($d = 0.57$) is consistent with that found in previous studies comparing nonselective inhibition speeds in children with ADHD versus normal controls (Nigg, 1999; Oosterlaan et al., 1998; Schachar et al., 2000). This indicates that the present study's manipulation of the stop-signal task produced differences in inhibition consistent with those previously reported in the literature: clearly, children with ADHD experienced greater difficulty in inhibiting to the selected auditory signal than did community controls.

The experimental manipulation of the stop-signal task used to measure selective inhibition was evidently successful. In the selective stop-signal task, the response inhibition task was made more complex by requiring the initial perceptual discrimination between different auditory signals while the response execution task remained unchanged relative to the basic, nonselective stop-signal task (Logan, 1985). Results indicate that this version of the stop-signal task was indeed successful at challenging the participants' inhibition process while having little impact on their response execution. Mean SSRTs were greater for both the children with ADHD and community controls than those previously reported using simpler response inhibition tasks while response execution (GoRT) speeds remained very similar (see Nigg, 1999; Purvis & Tannock, 2000, for nonselective SSRT means). In addition, participants were able to selectively inhibit to the selected auditory signal, as evident by percent inhibition to the selected and nonselected auditory signals, respectively.

Interestingly, despite the increased challenge of the inhibition process, mean SSRT remained faster (180 ms) than mean GoRT for the community controls, as has been previously shown with nonselective inhibition in children both with and without ADHD (Nigg, 1999; Purvis & Tannock, 2000; Schachar et al., 2000). However, this was not the case for the children with ADHD who had SSRTs very similar to their GoRTs in the selective stop-signal task. In addition, MPH did not separate SSRT and GoRT in these children, as will be discussed later. The significance of this unexpected pattern of findings for SSRT and GoRT in children with ADHD is unknown and needs further investigation.

Although the selective stop-signal task was successful in stressing the inhibitory process in children with ADHD, it was no more successful than the nonselective stop-signal task in capturing a greater proportion of children with ADHD with impaired inhibition relative to controls. That is, 36% of the ADHD sample found to have impaired selective SSRT was equivalent to the proportion of the ADHD sample previously found to have impaired nonselective SSRT using the same categorical approach in classifying impairment (Purvis & Tannock, 2000). This finding suggests that deficits in stop-signal inhibition are not characteristic of most children with ADHD, and/or that this task is not sensitive to the type of inhibitory deficit that may exist in ADHD.

In this study, children with ADHD showed poorer performance on a number of parameters in addition to selective inhibition than did community controls. For instance, children with ADHD showed increased variability and poorer accuracy of response execution, as well as a greater total number of invalid anticipatory responses than did controls. This suggests that the cognitive deficit in children with ADHD may not be limited to inhibition, as previously suggested. Perhaps difficulty encountered on the selective stop-signal task by children with ADHD is reflective of a more general deficit in information processing or of other cognitive processes used during the task such as the demands continuously placed on working memory in remembering which auditory signal requires inhibition of the go task response.

Stimulant medication (MPH) improved selective inhibition in children with ADHD. When compared to other stimulant effect studies on inhibition using different manipulations of the stop-signal task, this study's inhibition dose–response more closely resembled the nonlinear dose improvements seen in inhibition using a stop-signal task with a complicated response execution (Go) task (Tannock et al., 1995) than that of linear dose improvements observed using the basic stop-signal task (Tannock et al., 1989).

The significance of the unexpected pattern of overlapping SSRT and GoRT speed in the children with ADHD in this study is unknown. Moreover, although MPH had an overall beneficial effect on performance, it still could not address this processing difficulty in children with ADHD. Perhaps children with ADHD are particularly impaired in dealing with unpredictable stimuli, especially when it requires an attentional and response shift, and MPH does not help this set shifting.

Interestingly, children with ADHD showed improved performance not only in selective inhibition, but also in speed and variability of response execution when given MPH. Thus, MPH may influence global cognitive processes, such as attentional capacity or working memory, that are deficient in children with ADHD and result in improvements in aspects of response inhibition, as well as response execution. Alternatively, MPH may influence a number of distinct executive functions including response inhibition and those involved in the selection, execution, or maintenance of an optimal response strategy (Tannock et al., 1989).

Limitations of this study must be considered in interpreting the findings. The recruitment methodology of our sample of community controls did not permit collection of some types of data such as IQ or behavioral profiles. Thus, we cannot confirm that the community sample was free of psychopathology or was of comparable intellectual ability to the children with ADHD. Also, because of our MPH study sample characteristics, differences in MPH effects on selective inhibition among the three *DSM-IV* ADHD subtypes were not investigated.

A critical question is whether a cognitive task can be used in the diagnosis of ADHD and to quantify degree of impairment. To address this issue, we used a categorical approach to compare the proportion of individuals with deficient inhibition in ADHD and controls. However, the categorical approach used in this study did not provide better discrimination between children with ADHD and community controls on the selective stop-signal task than had been previously observed in nonselective inhibition (Purvis & Tannock, 2000). Future studies with large samples of children with ADHD using receiver–operator curve (ROC) analyses might provide precise impairment cut-off scores of inhibition.

A future study that directly assesses differences between selective and nonselective inhibition in the same group of children with ADHD would provide insight into the relationship between nonselective and selective inhibition. This type of study would help clarify which domains of function or specific measures are affected by the additional manipulation in selective inhibition. Also, it might provide information about the impact of particular cognitive functions, such as working memory, on different types of inhibition.

In addition, studies comparing the performance of children with ADHD and other psychiatric or cognitively impaired groups on the selective stop-signal task are required to ascertain whether deficits in selective inhibition are (a) unique to children with ADHD, (b) characteristic of a disorder which is commonly seen comorbid with ADHD, or (c) evident only in a circumscribed group of children with ADHD.

In summary, this novel study was highly successful in examining selective inhibition in children with ADHD both on and off stimulant medication. Results generated from this study clearly demonstrate impairment in selective inhibition in children with ADHD compared to community controls. This study's findings both complement and build on the existing ADHD inhibition literature and validate the use of the selective stop-signal task for future studies examining response inhibition in childhood psychopathology.

## APPENDIX

Stop-signal reaction time is estimated from a model that assumes a race between the stop processes and the go processes. If the stop process wins, the response to the go task is inhibited. If the go process wins, the response to the go task is executed. Typically, the finishing time of the go process is estimated from go trials in which no-stop signal occurs. Indeed, the SSRTs in the present experiment were estimated that way. However, the present experiment provides a second way to estimate the finishing time of the go process. Participants were presented with two auditory signals, one designated as the selected auditory signal (i.e., stop-signal) and one designated as the nonselected auditory signal. They were required to respond to the go task when the nonselected auditory signal occurred. Their reaction times to the go task on these nonselected auditory signal trials can also be used as an estimate of the finishing time of the go task. Indeed, reaction times on nonselected auditory signal trials may provide a more appropriate

estimate of the finishing time of the go process because an auditory signal was presented, just as on stop trials, and the auditory signal may influence the finishing time of the go process (De Jong, 1991). If the go reaction times on nonselected auditory signal trials were significantly different from go reaction times on auditory signal absent trials, it may be more appropriate to use go reaction times from nonselected auditory signal trials to calculate SSRT.

To assess this possibility, we calculated mean go reaction time on nonselected auditory signal trials. It turned out to be faster than mean go reaction time on auditory signal absent trials for both ADHD participants (mean nonselected auditory signal trial GoRT = 500 ms; mean auditory signal absent trial GoRT = 567 ms) and control participants (mean nonselected auditory signal trial GoRT = 530 ms; mean auditory signal absent trial GoRT = 587 ms). The difference was significant, $F(1, 116) = 47.29$, $p < .01$, but it did not interact with diagnostic group, $F(1,116) = 0.30$, *ns*. Consequently, we recalculated SSRT, using the mean go reaction times from nonselected auditory signal trials to estimate the finishing time of the go process. With this calculation, SSRT was still significantly longer for ADHD participants than for controls, mean SSRTs were 457 ms for ADHD participants and 342 ms for controls, $F(1, 116) = 7.94$, $p < .01$. Thus, ADHD participants inhibit more slowly than controls, no matter how SSRT is calculated.

Finding that the nonselected auditory signal trials sped up the go reaction times may appear to challenge the assumption that go and stop processes are independent. This is an important issue because the independence assumption is essential in justifying the calculation of SSRT (Logan & Cowan, 1984). However, the kind of independence assumed in the race-model calculation (*stochastic independence*) is different from the kind of independence that may be violated by the auditory signal speeding up go reaction times (*functional independence*), so the finding may not challenge our application of the race model to the data. Stochastic independence means that the joint probability of two events is the product of the marginal probabilities of the events, that is $P(A \cap B) = P(A)P(B)$. Functional dependence means that the probability of one event is related to the probability of another event, that is, $P(A)$ is correlated in some way with $P(B)$. It is possible to have a violation of functional independence and maintain stochastic independence. Some manipulation may increase both $P(A)$ and $P(B)$, violating functional independence, but stochastic independence will still be maintained if $P(A \cap B)$ still equals $P(A)P(B)$. Thus, the auditory signal may speed up the go and the stop processes but that need not violate the stochastic independence that the race model assumes.

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