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A Psychophysical Measure of Attention Deficit in Children With Attention-Deficit/Hyperactivity Disorder

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The authors explored the temporal mechanism of attention deficit in children with attention-deficit/hyperactivity disorder (ADHD). In rapid serial visual presentation tasks in which two targets (T_1 and T_2) were presented in close temporal proximity among distractors, participants tried to identify T_1 and detect T_2 in one (dual-task) experiment and only to detect T_2 in a second control (single-task) experiment. The sensitivity of T_2 detection was analyzed using signal detection theory. The attentional blink—the impairment in T_2 detection following the identification of T_1—was increased in magnitude and protracted in the patients. Moreover, some ADHD children appeared to have a blink largely normal in magnitude but temporally displaced toward a later time. The authors hypothesize that a slower closing of the attention gate may mediate this specific attention impairment in ADHD children.

Children with attention-deficit/hyperactivity disorder (ADHD) have difficulty giving attention to details and carrying on sustained attention during certain activities. Many studies have tried to elucidate the mechanism underlying the attention impairment in these children. However, the results obtained in the literature are far from converging (Blondis, Snow, & Accardo, 1999), and some have even questioned that attention impairment is a core deficit in ADHD (Van der Meere, 1996). Impairment of other neuropsychological functions, particularly those implicating the frontostriatal circuitry, has also been demonstrated in ADHD (Barkley, Grodzinsky, & DuPaul, 1992; Castellanos et al., 2000; Grodzinsky & Diamond, 1992; Klorman et al., 1999; Øie & Rund, 1999; Ross, Hommer, Breiger, Varley, & Radant, 1994; Shue & Douglas, 1992). These and anatomical studies have largely arrived at consistent conclusions regarding the underlying neuropathology of this disorder (Hynd et al., 1993; Castellanos et al., 1996; Castellanos et al., 2001; Filipek et al., 1997; Semrud-Clikeman et al., 2000; see also Giedd, Blumenthal, Molloy, & Castellanos, 2001, for a review). Of particular interest to the current work is the involvement of the cerebellum in ADHD. The cerebellum has long been implicated in the processing of timing information either in sensorimotor control or in cognitive operations (Ivry, 1997; Salman, 2002). More recently, the role of the cerebellum in attentional functions has also been documented (Allen, Buxton, Wong, & Courchesne, 1997; Courchesne et al., 1994; Le, Pardo, & Hu, 1998; Ravizza & Ivry, 2001). Given the anatomical connections between the cerebellum and the association cortices (Middleton & Strick, 2000, 2001; Schmahmann & Pandya, 1997), it is not unlikely that a network of brain areas involving the cerebellum and the prefrontal-striatal circuitry mediates the temporal aspects of attention functions. A behavioral experiment exploring this particular functional dimension perhaps would provide further clues to understanding the underpinning of the cognitive deficits in this disorder.

The current study represents a step toward this direction. We explored this issue by examining how the allocation of processing resources at one point in time might affect the processing of a subsequent event in children with ADHD differently from that in children without ADHD. We used rapid serial visual presentation (RSVP) paradigms, which have been used extensively in the literature to explore the temporal characteristics of information processing (Broadbent & Broadbent, 1987; Chun & Potter, 1995; Raymond, Shapiro, & Arnell, 1992; Reeves & Sperling, 1986; Shapiro, Raymond, & Arnell, 1994; Ward & Duncan, 1996; Weichselgartner & Sperling, 1987; see also Shapiro, Arnell, & Raymond, 1997, for a review) and, more recently, to examine attention deficits in people with neurological and psychiatric disturbances (Hollingsworth, McAuliffe, & Knowlton, 2001; Husain, Shapiro, Martin, & Kennard, 1997; Li et al., 2002; Rizzo, Akutsu, & Dawson, 2001; Rokke, Arnell, Koch, & Andrews, 2002). Figure 1 illustrates a typical example of this behavioral paradigm. In this behavioral task, a series of stimuli are presented in rapid succession, and the participants are required to identify either one (control experiment) or two targets. When the participants are required to identify two targets, the first one is usually distinguished from the distractors by some physical characteristic, such as luminance or color. In this example task, the participants have to discriminate the first target and detect the presence of a specified character, *X*, under the dual-task condition. In the control single-task condition,

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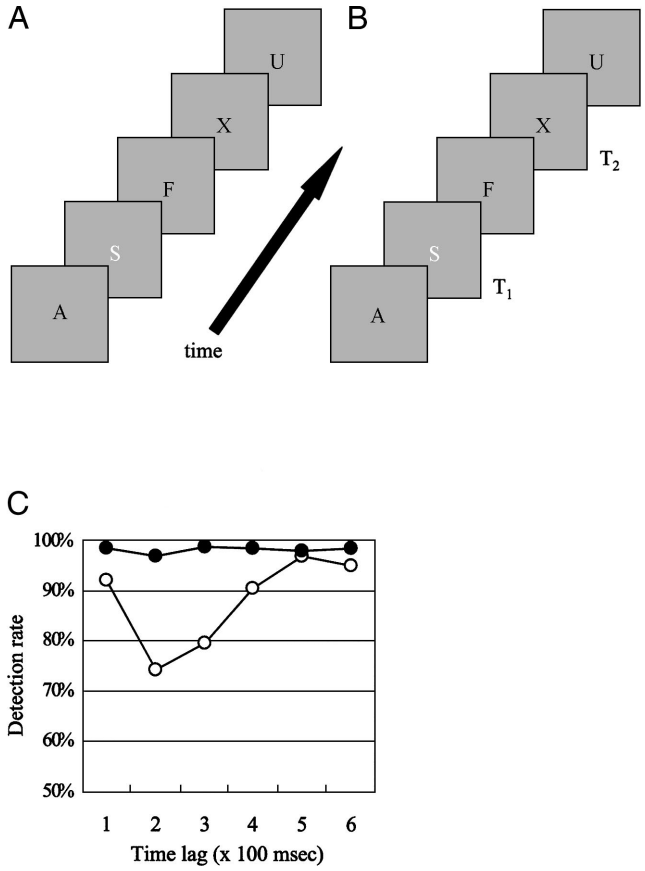

Figure 1. A typical rapid serial visual presentation paradigm and attentional blink. A: Single-target task. A stream of characters appears rapidly one after another. The participant's task is to identify whether there is an *X* in the stream. B: Dual-target task. The visual display is exactly the same as in the single-target task except that one of the characters (first target, or T_1) is brighter than the others. The participant has to identify T_1 (an *S* in this case) and then detect whether there is an *X* in the characters that follow T_1. C: Attentional blink. The detection rate of *X* in the single-target (filled circles) and dual-target (open circles) task is plotted with respect to the time lag between T_1 and *X*. Characteristically, the detection rate of *X* in the dual-target task drops for a period of several hundred msec after the identification of T_1, compared with that in the single-target task. Note that if *X* immediately follows T_1, it can escape the blink—an effect termed *Lag 1 sparing.*

the participant is required only to detect the character *X*. The visual stimuli and test settings are otherwise the same in the two task conditions. Overall, because the stimuli are presented for only a very short duration, focused attention is demanded to correctly identify the target(s) in this behavioral task. From the perspective of visual information processing, a later item in the stream eliminates an earlier stimulus from sensory storage, thus creating a stringent time constraint on the perceptual and cognitive processing of each individual stimulus. One can vary the task difficulty by adjusting the duration of presentation of the visual stimulus and by manipulating the physical similarity between the target and the distractors. With these parameters appropriately set, one can examine how attention and processing resources are temporally allocated to effectively deal with the rapidly changing inputs by comparing the respective performances of the participants for each of the two task conditions.

Characteristically, when participants are required to identify two targets (as compared with one target), an attentional "blink" in the processing of the second target, or $T_{2,}$ is observed for a few hundred milliseconds after the identification of the first target, or T_1 (Raymond et al., 1992; Figure 1C). According to a two-stage model, the attentional blink is the result of a limited-capacity second stage in which targets detected in the first stage are processed and consolidated serially (Chun & Potter, 1995). When the duration of this limited-capacity processing of T_1 exceeds the stimulus onset asynchrony between T_1 and T_2, interference with the processing of T_2 occurs. The resulting processing deficit suggests an intrinsic limitation of our central nervous system to deal with rapidly changing stimuli, highlighting the serial aspect of visual information processing. A recent study used this technique to examine the attention deficit in adults with ADHD and showed that, compared with healthy participants, the patients exhibited a deeper and wider attentional blink; namely, the blink was greater in magnitude and temporally more extended (Hollingsworth et al., 2001). It is interesting that similar findings have been observed in patients with depression (Rokke et al., 2002), with schizophrenia (Li et al., 2002), with bipolar disorders (Li & Lin, unpublished results), and in those with focal brain lesions (Husain et al., 1997; Rizzo et al., 2001).

An intriguing component of the attentional blink concerns the *Lag 1 sparing*, which describes the finding that the stimulus appearing during the first time lag (i.e., immediately following T_1) is oftentimes spared from the blink. In other words, participants are better able to detect T_2 when it appears at the first serial position than when it appears at the second or third position. The second and third positions typically are affected by the processing blink. A number of different models have been proposed to account for Lag 1 sparing (Raymond et al., 1992; Visser, Bischof, & Di Lollo, 1999), although conclusive evidence supporting these models has been relatively lacking. An interesting finding in the above-mentioned study on ADHD adults was that the patients exhibited Lag 1 sparing, similar to control participants (Hollingsworth et al., 2001). That is to say, compared with healthy controls, the adults with ADHD remained largely intact in detecting T_2 when it appeared at the first serial position after T_1. The authors reasoned that because the T_1 was physically highlighted (brighter) from the distractors, automatic attention engaged by this abrupt onset might account for the intact Lag 1 sparing. On the basis of these results, the authors suggested that the extended attentional blink in ADHD patients was not due to a deficit in short-term memory or a global deficiency in processing multiple targets. Instead, the impairment resulted from a failure of the ADHD patients in terms of their controlled attention, whereas their automatic attention remained unaltered.

In this study, we conducted two RSVP experiments on children with and without ADHD, with two goals in mind: to examine whether a similar deficit could be observed in children with ADHD and to explore for an alternative explanation for the attentional blink observed in this disorder. To this end, we used essentially the same design in the first experiment in order to replicate the results of Hollingsworth and colleagues. In our second experiment, we investigated whether the "automatic versus controlled attention" was a feasible account for attention deficit in ADHD, as

proposed by Hollingsworth and colleagues. We used the same design as in the first experiment, other than having T_1 of the same luminance as the distractors, in which case T_1 would not reflexively engage the attention of the participants. If the Lag 1 sparing of attentional blink occurred as a result of intact automatic attention, we would expect to observe a decrease or even disappearance of Lag 1 sparing in children with ADHD, in contrast to their performance in the first experiment. Conversely, if the Lag 1 sparing remained unaltered for children with and without ADHD in the second experiment, an alternative explanation should be in position.

Method

Participants

Forty-three children with ADHD (34 boys, 9 girls; mean age = 10.6 years, *SD* = 1.6) of the combined type (*Diagnostic and Statistical Manual of Mental Disorders* [4th ed.; *DSM–IV*; American Psychiatric Association, 1994]) and 40 age- and education-matched healthy children (28 boys, 12 girls; mean age = 10.9 years, *SD* = 1.4) participated in the study, with all of them having normal or corrected-to-normal vision. The participants with ADHD were recruited from the outpatient clinic of the Chang Gung Memorial Hospital. All control participants were children of hospital employees. Prior to the experiment, both control and ADHD children received a clinical interview by a child psychiatrist (Hsueh-ling Chang) based on a Chinese version of K–SADS (Kiddie—Schedule for Affective Disorders and Schizophrenia; Chambers & Puig-Antich, 1985). The interview confirmed the diagnosis of children with ADHD and ensured that the control participants were free of any psychological issues that required clinical attention. Specifically, we examined for any psychotic or nonpsychotic mental conditions that might warrant a clinical diagnosis. History of the use of medications or illicit substance was routinely obtained. Participants were also given a parental rating on a modified Chinese version of the Child Behavior Checklist (Achenbach, 1991). Children with ADHD demonstrated mixed symptoms of hyperactivity/impulsivity and attentional disturbance and had a higher average total score than the control participants (ADHD $M = 70.1$ [$SD = 10.2$] vs. control $M = 46.4$ [$SD = 7.2$]), $t(81) = 13.52$, $p < .00$, two-tailed. Nine ADHD children had a comorbid diagnosis of learning disorder (8 in written expression, 1 in reading), one had oppositional defiant disorder, and one had phonological disorder (*DSM–IV*). None of them had psychotic or major depressive disorders or any history of neurological insult or of use of substance. Thirty-two control and 23 ADHD participants took a Chinese version of the WISC—III (Wechsler, 1991). The mean full-scale IQ of participants with ADHD was lower than that of the healthy controls (ADHD $M = 96.1$ [$SD = 8.2$] vs. control $M = 110.7$ [$SD = 10.5$]), $t(53) = 5.79$, $p = .00$. The remaining participants declined taking the IQ test because of time concerns or other undisclosed personal reasons. Children with ADHD were prescribed methylphenidate 10 mg once or twice a day at the time and were medication-free for at least 24 hours before the experiment. None of the participants with ADHD were taking the slow-release form of methylphenidate. After receiving a complete description of the study, the participants and their parents gave written informed consent, according to institute guidelines.

Test Setting, Behavioral Task, and Experimental Procedures

The testing took place in an interview room in the outpatient clinic. Although the room was not soundproof, the setting was quiet and free of interruptions. Only the participant and the person who administered the test were allowed in the room. The experiments were in most cases conducted on a weekend or a holiday at any time during the day. This was particularly the case with ADHD participants, as these children were generally on methylphenidate on weekdays when they had to attend school. Approximately half of the healthy controls were tested after school on a weekday.

In the RSVP task, the visual stimuli, consisting of 34 Chinese phonological characters and 6 Arabic numerals (2 to 7), were presented in rapid succession at the location of visual fixation (Figure 2). All of our participants had learned these characters in school. Each trial consisted of 14 to 22 stimuli, one of which was a number. Each stimulus was presented for 33 msec with an interstimulus interval of 67 msec. The number was preceded by anywhere from 7 to 15 characters and followed by 6 more in a trial. In one experiment, the participants had to (a) identify the number, (b) detect whether there was an *X*-like character (XLC) following the number, and (c) rate their subjective confidence in the accuracy of their response on a 5-point scale (dual-task condition) ranging from 1 (*absolutely sure that there was no XLC*) to 5 (*absolutely sure that there was an XLC*.) The XLC appeared in half of the trials pseudorandomly at one of the six serial positions ("lags") following the number, with each position repeated 20 times in an experiment. In a second, control experiment, participants were instructed to ignore the number and only to detect the XLC (single-task condition). Participants pressed the space bar to initiate a trial at their own pace. The experiments lasted approximately 80 min for each participant.

The detection sensitivity of the XLC was computed for each time lag using receiver operating characteristic (ROC) analysis based on signal detection theory (Macmillan & Creelman, 1991). A deterioration of the detection sensitivity of XLC in the dual-task compared with the single-task condition has been termed an *attentional blink* (Raymond et al., 1992). The attentional blink can be quantified on two aspects: overall magnitude and temporal pattern. The former was derived by summing up the decrease in detection sensitivity at each individual time lag, and the latter reflected the temporal dynamics in attention processing over a time period of 600 msec examined in the current study. The response bias in the detection of the XLC was also computed for each participant in units of root-mean-squared standard deviation (Macmillan & Creelman, 1991).

We used two versions of the RSVP task in the current study. The two tasks were identical except for the luminance of the first target, the number.

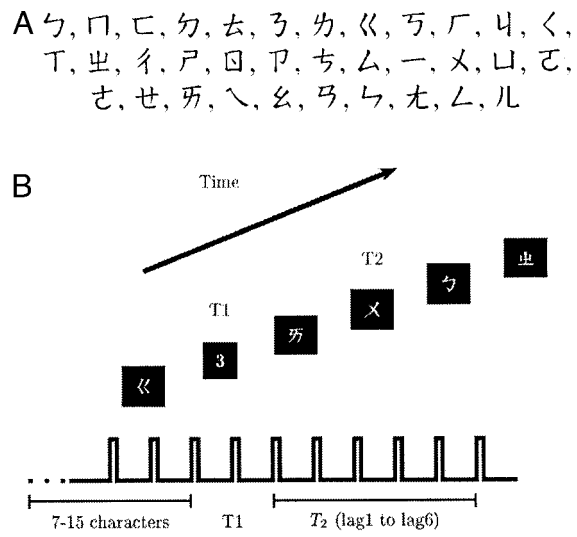

Figure 2. The stimulus set (A) and rapid serial visual presentation (RSVP) task (B) in Experiment 1. Chinese phonological characters and Arabic numbers were presented on the basis of one stimulus per 0.1 s. The Arabic number was brightened. The participants identified the number first and detected an *X*-like character (XLC), which could appear anywhere from Lag 1 to Lag 6. In a control experiment, the participants ignored the number and tried to detect only the XLC. The RSVP task in relation to Experiment 2 was identical except that the number was not brightened.

In the first, number-highlighted (or NH) task (Experiment 1), the number had a luminance of 18.67 cd/m^2, compared with 4.85 cd/m^2 for the characters and 0.2 cd/m^2 for the dark background, against which all visual stimuli were presented. In the second, number-nonhighlighted (or NnH) task (Experiment 2), all stimuli had the same luminance, namely, 4.85 cd/m^2.

Results

General Performance

Twenty control and 22 children with ADHD participated in the NH task (Experiment 1), and 20 control and 21 children with ADHD participated in the NnH task (Experiment 2). In both experiments, the control participants had a higher correct number (T_1) discrimination rate than the ADHD participants. In Experiment 1, control participants correctly identified the number in an average of 89.8% (*SD* = 12.1%) of the trials, whereas ADHD participants did so in an average of 82.0% (*SD* = 13.2%) of the trials, $t(40) = 1.99$, $p = .03$, two-tailed. In Experiment 2, control participants correctly identified the number in 82.5% (*SD* = 10.3%) of the trials, whereas ADHD participants did so in 67.5% (*SD* = 13.9%) of the trials, $t(39) = 3.92$, $p = .00$, two-tailed. On the other hand, healthy controls and participants with ADHD did not differ from each other in terms of their response bias (in units of root-mean-squared standard deviation) either in Experiment 1 ($M = -0.30$ [*SD* = 0.43] for controls; $M = -0.08$ [*SD* = 0.52] for ADHD participants), $t(40) = 1.45$, $p = .15$, two-tailed, or Experiment 2 ($M = -0.20$ [*SD* = 0.43] for controls; $M = -0.31$ [*SD* = 0.47] for ADHD participants), $t(39) = 0.77$, $p = .44$, two-tailed.

T_2 Detection Sensitivity and Attentional Blink

One control and 4 ADHD participants were excluded from further analyses of the results in Experiment 1 either because their correct number identification rate was at chance level (2 ADHD participants) or because they reported pressing the wrong key in the confidence judgment regarding the accuracy of their response (2 ADHD and 1 control participant). For the same reasons, 1 control and 4 ADHD participants were excluded in Experiment 2. This left us with 19 controls and 18 ADHD participants in Experiment 1 and 19 controls and 17 ADHD participants in Experiment 2. These exclusion criteria were imposed because we were primarily interested in investigating how allocation of attention to the first target would impair the processing of the second. A less-than-ideal performance in the identification of the number (T_1) in the dual-task condition suggested that the participant was not adequately engaged in the task, making it difficult to interpret the results. The faulty keypress about confidence level judgment occurred when, for instance, the participant selected the *5* key when in fact the *1* key was intended, or vice versa. Our ROC analysis showed that these response errors greatly decreased the detection sensitivity and altered the temporal profile of the attentional blink. We thus opted to treat these patients as outliers.

Figure 3 plots the attentional blink obtained in the two experiments for both groups of participants. The results showed that, compared with control children, children with ADHD had a blink that was overall larger in magnitude, $F(1, 35) = 9.567$, $p = .00$ (group main effect, repeated measures analysis of variance [ANOVA]), and different in terms of the temporal profile, $F(5, 31) = 2.431$, $p = .04$ (Group × Lag interaction) in Experiment 1. Similar results were obtained in Experiment 2: $F(1, 34) = 18.412$, $p = .00$, for group main effect; $F(5, 30) = 2.352$, $p = .04$, for Group × Lag interaction. Children with ADHD on average had a 136% and 179% increase in blink magnitude over the healthy controls in Experiments 1 and 2, respectively. Moreover, the attentional blink appeared temporally more extended in children with ADHD. Including in these analyses those participants who were excluded because of their faulty keypress in confidence judgment rendered the Group × Lag interaction statistically less significant, $F(5, 34) = 2.253$, $p = .07$, for Experiment 1; $F(5, 33) = 2.197$, $p = .06$, for Experiment 2, but did not alter the group main effect: $F(1, 38) = 10.986$, $p = .00$, for Experiment 1; $F(1, 37) = 20.905$, $p = .00$, for Experiment 2. The processing deficit of the children with ADHD was thus deeper and wider in both experiments—that is, regardless of whether the number was highlighted or not. The main finding of Hollingsworth et al. (2001) was replicated here in children with ADHD.

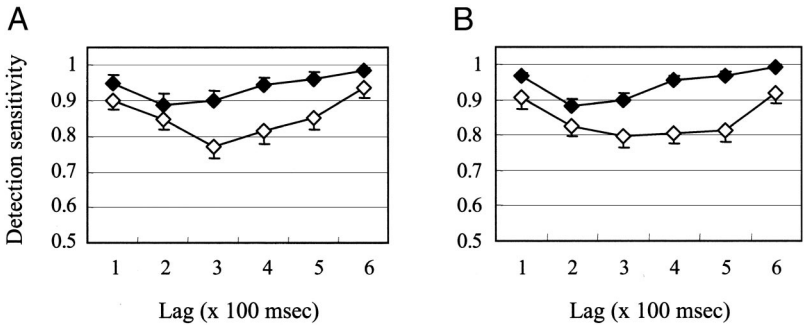

Figure 3. Attention blink in control (filled diamonds) and attention-deficit/hyperactivity disorder (ADHD; open diamonds) participants. Each data point represents an average across all participants of the difference in detection sensitivity between single-target and two-target conditions. The error bars represent standard errors. A: In Experiment 1, in which the first target (T_1) is highlighted, control and ADHD participants demonstrate an attentional blink that peaks at Lag 2 and Lag 3, respectively. However, the magnitude of the blink is greater in the patient participants. B: In Experiment 2, T_1 is not highlighted. As compared with control participants, ADHD participants demonstrate a blink larger in magnitude and with a peak that is more delayed.

To clarify the nature of the observed Group × Lag interactions, we computed the simple main effect of group at each time lag with univariate ANOVAs. The results from Experiment 1 showed that the magnitude of the attentional blink differed between groups for Lag 3 through Lag 5 but not for Lags 1, 2, or 6: Lag 1, $F(1, 35) = 1.600$, $p = .21$; Lag 2, $F(1, 35) = 1.066$, $p = .31$; Lag 3, $F(1, 35) = 11.263$, $p = .00$; Lag 4, $F(1, 35) = 9.419$, $p = .00$; Lag 5, $F(1, 35) = 7.031$, $p = .01$; Lag 6, $F(1, 35) = 3.178$, $p = .08$. Similar results were obtained in Experiment 2. The magnitude of the attentional blink differed between groups for Lag 3 through Lag 6 but not for Lags 1 or 2: Lag 1, $F(1, 25) = 2.769$, $p = .11$; Lag 2, $F(1, 35) = 3.032$, $p = .09$; Lag 3, $F(1, 35) = 10.073$, $p = .00$; Lag 4, $F(1, 35) = 14.233$, $p = .00$; Lag 5, $F(1, 35) = 19.707$, $p = .00$; Lag 6, $F(1, 35) = 6.100$, $p = .02$. The magnitude of attentional blink was indeed greater at the later time lags in children with ADHD as compared with healthy controls.

We separated the children with ADHD into two groups, one with a comorbid diagnosis of learning disorder (LD; $n = 8$) and the other without ($n = 27$). We conducted another repeated measures ANOVA with time lag as the within-participant variable, group and experiment as the between-participant factors, and the magnitude of attentional blink as the dependent measure. The results showed that there was a main effect of group, $F(1, 31) = 6.872$, $p = .01$: Children with ADHD and a comorbid LD on average had a 67% increase in the magnitude of attentional blink over those without LD. None of the second- or third-order interactions were significant. The temporal pattern of attentional blink did not appear to differ between the two groups.

We examined the temporal profile of attentional blink in each individual and noticed that some children with ADHD had a blink relatively "normal" in magnitude (within half a standard deviation of the mean of the control participants) but with their peak distinctively displaced toward a later time lag. Furthermore, some other children with ADHD had a blink that, other than being deeper than that in controls, appeared to weigh toward later time lags. To quantify the extent of these observations, we computed for each individual participant the magnitude of the blink (the difference in XLC detection sensitivity between the dual- and single-task conditions) for an early time period, comprising Lag 1 to Lag 3, and a late time period, comprising Lag 4 to Lag 6. A participant was considered to be an "early blinker" if there was a larger decrease in the detection sensitivity in the early time period and a "late blinker" if the reverse applied. Table 1 shows the number of participants in each category for both experiments. We looked for a difference in this frequency measure between control and ADHD participants in a hierarchical log-linear model with the group (ADHD and control), experiment (1 and 2), and blink period (early and late) as categorical variables. The results showed that there was a significant blink period (partial $\chi^2 = 8.737$, $p = .00$) but not an experiment or group (partial $\chi^2 < 0.2$, $p > .70$, for both) main effect, suggesting that there were overall more early blinkers. More important, there was a significant Blink Period × Group interaction (partial $\chi^2 = 7.673$, $p = .01$) but not a Group × Experiment (partial $\chi^2 = 0.24$, $p = .88$) or Experiment × Blink Period (partial $\chi^2 = 0.016$, $p = .90$) interaction. The frequency distribution of early and late blinkers thus differed between control and ADHD participants, with more early blinkers in the former, but not in the latter, group of participants. There was no significant third-order interaction (partial $\chi^2 = 0.189$, $p = .66$).

Table 1
Number of Early Versus Late Blinkers

	Group	
Experiment	Control	ADHD
1		
Early blinker	16	9
Late blinker	3	9
2		
Early blinker	15	9
Late blinker	4	8

Note. ADHD = attention-deficit/hyperactivity disorder.

Correlation of Performance With IQ

Because the children with ADHD as a group had a lower IQ than the healthy controls, the question arose whether the results obtained so far could simply be attributed to their general intellectual level. Seventeen control and 12 ADHD participants who took the WISC–III took part in Experiment 1, and 15 control and 11 ADHD participants participated in Experiment 2. We performed regression analyses as a first step to address this question. The results showed that, for healthy controls, the magnitude of attentional blink was not correlated (a) with their full-scale IQ (FIQ), $F(1, 15) = 0.007$, $p = .94$, $r^2 = .001$, for Experiment 1; $F(1, 13) = 1.130$, $p = .31$, $r^2 = .08$, for Experiment 2; (b) with their verbal IQ (VIQ), $F(1, 15) = 0.344$, $p = .57$, $r^2 = .022$, for Experiment 1; $F(1, 13) = 0.000$, $p = .10$, $r^2 = .00$, for Experiment 2); or (c) with their performance IQ (PIQ), $F(1, 15) = 1.429$, $p = .25$, $r^2 = .087$, for Experiment 1; $F(1, 13) = 3.526$, $p = .08$, $r^2 = .213$, for Experiment 2. Similar results were obtained for ADHD participants: (a) FIQ, $F(1, 10) = 0.000$, $p = .10$, $r^2 = .000$, for Experiment 1; $F(1, 9) = 0.868$, $p = .38$, $r^2 = .09$, for Experiment 2; (b) VIQ, $F(1, 10) = 0.158$, $p = .70$, $r^2 = .016$, for Experiment 1; $F(1, 9) = 3.871$, $p = .08$, $r^2 = .301$, for Experiment 2; (c) PIQ, $F(1, 10) = 0.000$, $p = .10$, $r^2 = .000$, Experiment 1; $F(1, 9) = 1.109$, $p = .32$, $r^2 = .110$, for Experiment 2.

To evaluate whether the difference in the magnitude of attentional blink between groups could be accounted for solely by their difference in IQ, we then performed an analysis of covariance (ANCOVA) with the participant group as the factor, their IQ score as the covariate, and the magnitude of attentional blink as the dependent variable. We first tested the assumption of the homogeneity of slopes, which was satisfied in each of these ANCOVAs (i.e., the Factor × Covariate interaction was not significant). The results showed that for Experiment 1, the difference in blink magnitude between children with ADHD and healthy controls still held even after their difference in FIQ was accounted for, $F(1, 26) = 4.734$, $p = .04$. Similar results were obtained for VIQ, $F(1, 26) = 4.907$, $p = .04$, and for PIQ, $F(1, 26) = 7.028$, $p = .01$. The same and even stronger results applied to Experiment 2: FIQ, $F(1, 23) = 17.898$, $p = .00$; VIQ, $F(1, 23) = 26.434$, $p = .00$; and PIQ, $F(1, 23) = 11.681$, $p = .00$.

We also examined whether FIQ differed between early and late blinkers. A 3-way ANOVA with participant group, experiment, and blink pattern as factors and FIQ as the dependent measure

showed that, as expected, there was a significant main effect of participant group, $F(1, 47) = 20.187$, $p = .00$. However, the main effect of blink pattern was not significant, $F(1, 47) = 1.079$, $p = .30$, suggesting that the early and later blinkers did not differ in their FIQ. None of the two- or three-way interactions were significant either (all Fs < 1). It thus appeared that the difference in performance in the RSVP task between ADHD and control participants could not be explained by their IQ measures.

Discussion

Extended Attentional Blink in ADHD Participants

A number of models have been proposed to explain attentional blink, and these models share common characteristics (Chun & Potter, 1995; Shapiro et al., 1997). In essence, attentional blink occurs because the resources at a capacity-limited processing stage are not available for the second target until the processing of the first target is completed. As the representations of the second target are held up in the perceptual buffer, they are vulnerable to interference from subsequent stimuli in the stream. An increased and protracted attentional blink thus suggests a less efficient processing mechanism in children with ADHD when they have to deal with rapidly changing inputs.

In the present study, the attentional blink lasted for approximately 500 to 600 msec for both ADHD and healthy children. Moreover, the blink was greater in magnitude and temporally more extended in ADHD than in healthy participants. This result is consistent with those obtained in Hollingsworth et al. (2001) on adult patients. Note that in the current work, when the second target (the XLC) appeared at the last time lag, it was not followed by a distractor and thus was not vulnerable to an interference effect. The detection sensitivity at the last time lag may therefore have been artificially inflated. Further studies with longer time lags are required to ascertain whether the attentional blink in children with ADHD would extend beyond 600 msec, although this measure will also depend on the physical attributes of the stimuli and other timing parameters used in the experiments.

More important, we obtained essentially the same results on attentional blink whether the first target was physically highlighted from the distractors or not. The magnitude of attentional blink appeared to be comparable and Lag 1 sparing occurred in both experiments. These findings rule out the alerting effect of the first target as an explanation for Lag 1 sparing and are at odds with the "automatic versus controlled attention" account of the attentional blink in ADHD, as proposed by Hollingsworth and colleagues.

The impairment of attention in the temporal domain is reminiscent of the slower early visual information processing demonstrated in ADHD participants with the backward masking paradigm (Rund, Oie, & Sundet, 1996). However, the two phenomena address different aspects of temporal information processing. In visual backward masking, patients with ADHD identify fewer target stimuli than healthy controls when the target is briefly obscured by a second visual stimulus. They also have difficulty identifying target stimuli at interstimulus intervals that do not affect the performance of healthy controls. In RSVP, participants exhibit an attentional blink only in the dual-task condition, when they are required to identify two targets concurrently, but not in the single-task condition, although visual stimuli are presented at the same rate in both conditions. It thus appears that whereas the masking paradigm demonstrates an impairment in early visual processing, the RSVP task showcases a cognitive deficit in participants with ADHD.

Can the difference in attentional blink between ADHD and healthy children be explained in terms of their disparity in general intelligence? After all, the finding that the children with ADHD overall demonstrate a greater attentional blink seems, prima facie, to agree well with their lower average IQ score. Our regression and covariance analyses on their attentional performance and IQ suggest otherwise. The magnitude of attentional blink is not correlated with IQ measures for either ADHD or healthy children. Nor does the difference between the two groups disappear when IQ is taken into account. The poor performance of the children with ADHD in this RSVP task may thus reflect a distinct deficit in their attention functions. On the other hand, one should note that because only approximately two thirds of our participants had an IQ score entered in the analyses, these results should be considered preliminary.

The Attention Gating Model

By contrasting the temporal pattern of attentional blink between control and ADHD children, we observe that other than of a larger magnitude, the attentional blink in ADHD children occurs later than it does in control children. A small number of the patient participants have an attentional blink that is normal in magnitude but with a peak that is temporally displaced. These findings lead us to propose an attention gating model and to hypothesize a psychophysical mechanism for the attention impairment in ADHD participants (Figure 4).

Attention gating has been proposed in earlier studies to account for a variety of observations relating to the processing of the RSVP stimuli in visual short-term memory (Reeves & Sperling, 1986) and to account for Lag 1 sparing (Raymond et al., 1992), although the dynamics of gating were not elaborated in the latter work. Along with these suggestions, we propose here a model to account for the experimental findings obtained in the current study. In this model, an attention gate controls the flow of information from a perceptual buffer to a level where the cognitive processing required for target detection and identification takes place. Because processing capacity is limited, attention gating facilitates perceptual and cognitive processing by allowing only an adequate amount of information to access this level at a given time. A deficit or a blink occurs when the processing resources are not available or when incoming stimuli saturate those processing resources. In this conceptual framework, Lag 1 sparing occurs when the attention gate closes just fast enough for T_2 (XLC in the present experiment) to "sneak in" and be processed together with T_1 (the number) and only when resources are available to process this extra bit of information.

It is observed in the present work and that of Hollingsworth and colleagues that the attentional blink is more protracted in ADHD patients. Although limited capacity can account for the wider attentional blink obtained in these patients, the attention gating hypothesis provides another perspective to address this issue. The model suggests that their attention gate is slower in re-opening to allow for the processing of the stimuli that follow T_1. The stimuli are blocked and blinked out if they appear at a time when the

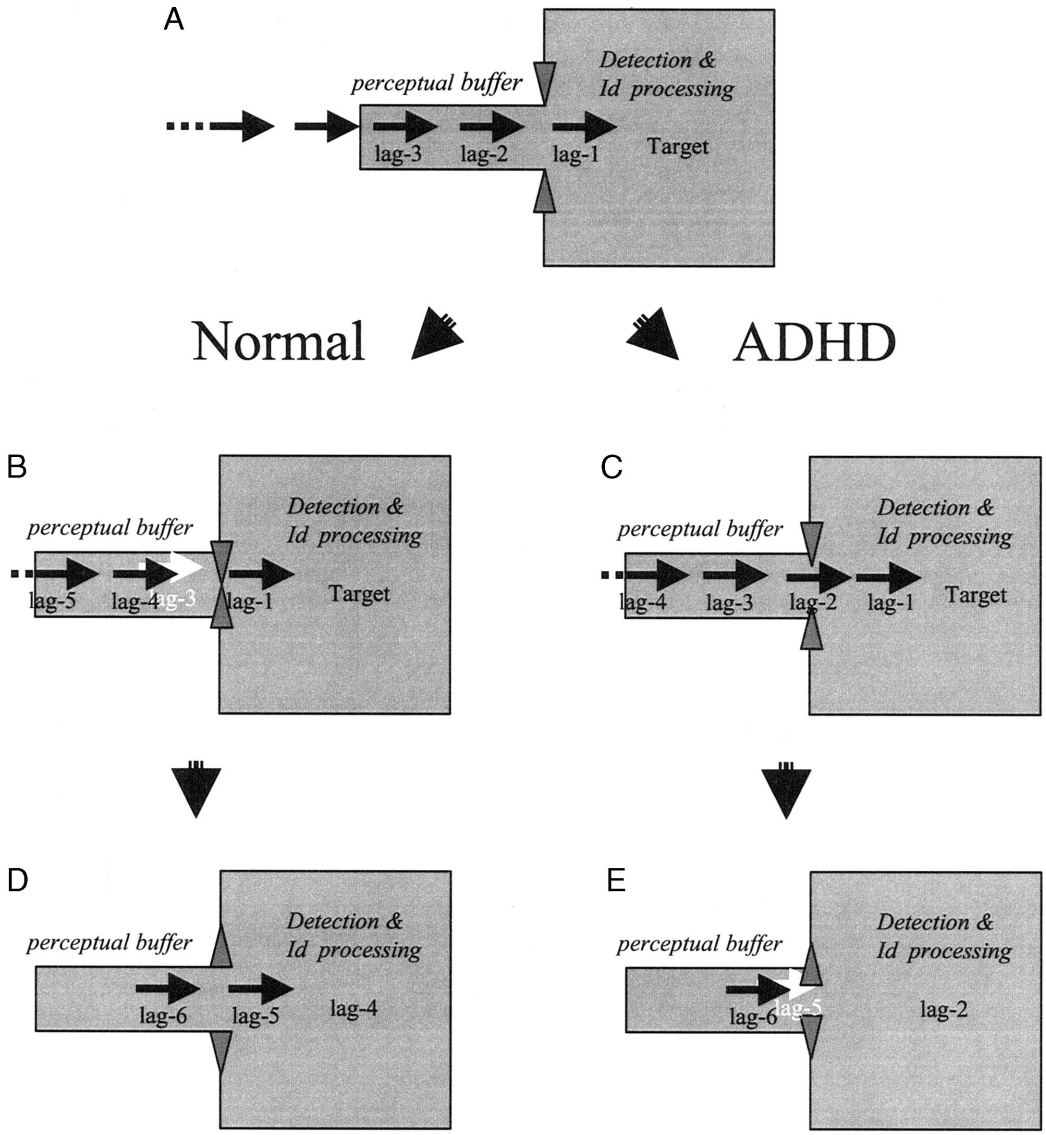

Figure 4. The attention-gating model. A: Stimuli early in the rapid serial visual presentation sequence are processed at a capacity-limited stage while the later ones are held up in the perceptual buffer. To allow for efficient processing of the stimuli, detection of a potential target triggers gate closing, which requires time to complete. B: The Lag 1 stimulus can sneak in before the gate completely closes. The stimuli in the buffer are eliminated from temporary storage by those that come after. C: The gate opens again once the identification (Id) processing for the target is complete. D: In a participant with attention-deficit/hyperactivity disorder (ADHD), the attention gate takes longer to close after target detection. As a result, more trailing stimuli access the capacity-limited stage than in the case of a control participant. This in turn can lead to two possible outcomes. If the participant has adequate processing resources, Lag 2 or even Lag 3 stimuli can be processed, resulting in multiple early-lag sparing. If the participant does not have adequate resources for concurrent processing, this will lead to a deterioration in identification for all of the stimuli. E: Moreover, gate re-opening is also slower in ADHD participants, resulting in more trailing stimuli being obliterated in the perceptual buffer.

attention gate is closed. An intriguing possibility is that if the dynamics of attention gating are affected as a whole, we would expect gate closing to be slower in ADHD children as well. More stimuli following T_1 will pass the gate before it closes, which could lead to two possible outcomes. First, if there are not enough resources, these stimuli will not be efficiently processed and a deeper and wider blink will thus be in place. Second, if there are adequate resources, these stimuli will be processed and identified and a blink will therefore not be visible at the earlier time lags occupied by these stimuli. This leads us to predict that, whereas most of the children with ADHD will demonstrate an attentional blink deeper and wider than normal participants, some of them

with adequate processing resources may exhibit a pattern of attentional blink with a largely normal magnitude but that is temporally displaced toward later time lags. In other words, compared with healthy controls, these children with ADHD will be able to process and detect stimuli that closely follow T_1, resulting in Lag 1, Lag 2, and perhaps even Lag 3 sparing, at least relatively speaking.

Indeed, we observe that although children with ADHD have significantly deeper and wider attentional blinks overall than healthy controls, some of these children have a blink with a normal magnitude but one that is temporally displaced toward a later time lag. These results support a deficit in the temporal gating of attention in ADHD. On the other hand, one must note that our statistical analysis does not allow the isolation of a subgroup of ADHD children without further qualifications. This particular finding is thus at the present time only descriptive in nature. Moreover, the attention gating model posits that the ADHD children whose blink is normal in magnitude but temporally displaced are equipped with more processing resources than those whose attentional blink is both deep and wide. We do not have independent evidence to substantiate this prediction. In fact, our analysis on IQ does not reveal a difference between these children. Given our relatively small sample of participants, more studies clearly are warranted to investigate this issue further and to explore if there are any differences in clinical characteristics among them.

Specificity of the Attention Gating Deficit in ADHD Participants

Evidence supporting attention impairment in people with ADHD has mostly come from studies using the Continuous Performance Tests (see Barkley et al., 1992; Corkum & Siegel, 1993, for a review). A recent study attempted to ascertain the specificity of sustained attention deficits in children with ADHD (Swaab-Barneveld et al., 2000). Children with a variety of different psychiatric diagnoses, including conduct disorder, mood disorder, and pervasive developmental disorder, as well as healthy and ADHD children were engaged in a sustained attention task. The results showed that sustained attention deficit is to a certain extent common to all children with psychiatric disorders. These sustained attention tests are thus sensitive but lacking in specificity in relation to participants with ADHD (Riccio & Reynolds, 2001).

In the clinical population, an increased attentional blink has been demonstrated in RSVP-related tasks in patients with dyslexia, with schizophrenia, and with depression as well as in patients with focal brain lesions (Hari, Valta, & Uutela, 1999; Husain et al., 1997; Li et al., 2002; Rizzo et al., 2001; Rokke et al., 2002). An increased attentional blink is thus not a finding specific to ADHD patients. Instead, it may simply reflect limited attention capacity, which could occur in a variety of neurological conditions. Could the slower dynamics in attention gating be a finding more specific to ADHD participants? In an earlier study of patients with schizophrenia, we observed that although they demonstrated an extended attentional blink, none of them exhibited a blink of normal magnitude that was temporally displaced, as can occur in children with ADHD (Li et al., 2002; Li, Yang, & Lin, unpublished results). In other words, the attentional blink in schizophrenia patients, though seemingly similar to that observed in ADHD patients, can be explained entirely by means of limited attentional or processing resources. It remains to be seen whether the same applies to other neurological conditions.

In conclusion, a gating deficit may be central to the attention impairment in ADHD. Together with the RSVP or related behavioral paradigms, the attention gating hypothesis is worth pursuing in order to unravel the underlying mechanism of the attention impairment in ADHD.

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