Published in final edited form as:

J Abnorm Child Psychol. 2008 August ; 36(6): 903–913. doi:10.1007/s10802-008-9221-0.

The Effects of Incentives on Visual-Spatial Working Memory in Children with Attention-Deficit/Hyperactivity Disorder

Keri Shiels¹, Larry W. Hawk Jr.¹, Cynthia L. Lysczek¹, Rosemary Tannock^{2,3}, William E. Pelham Jr.^{1,4}, Sarah V. Spencer¹, Brian P. Gangloff¹, and Daniel A. Waschbusch⁴

¹ Department of Psychology, University at Buffalo, SUNY, Buffalo, New York

² Institute of Medical Science, University of Toronto, Toronto, Ontario, Canada

³ Brain and Behavior Research Program, Research Institute, The Hospital for Sick Children, Toronto, Ontario, Canada

⁴ Department of Pediatrics, University at Buffalo, SUNY, Buffalo, New York

Abstract

Working memory is one of several putative core neurocognitive processes in Attention-Deficit/Hyperactivity Disorder (ADHD). The present work seeks to determine whether visual-spatial working memory is sensitive to motivational incentives, a laboratory analogue of behavioral treatment. Participants were 21 children (ages 7–10) with a diagnosis of ADHD-Combined type. Participants completed a computerized Spatial Span task designed to assess storage of visual-spatial information (forward span) and manipulation of the stored information (backward span). The Spatial Span task was completed twice on the same day, once with a performance-based incentive (trial-wise feedback and points redeemable for prizes) and once without incentives. Participants performed significantly better on the backward span when rewarded for correct responses, compared to the no incentive condition. However, incentives had no effect on performance during the forward span. These findings may suggest the use of motivational incentives improved manipulation, but not storage, of visual-spatial information among children with ADHD. Possible explanations for the differential incentive effects are discussed, including the possibility that incentives prevented a vigilance decrement as task difficulty and time on task increased.

Keywords

ADHD; working memory; incentives; motivation; cognition; executive function

Introduction

Attention-deficit/hyperactivity disorder (ADHD) is a heterogeneous disorder characterized by pervasive behavioral symptoms of inattention, hyperactivity and impulsivity beginning in childhood (American Psychiatric Association [APA], 1994). The heterogeneity of this disorder poses a major challenge in identifying the causal mechanisms that result in the behavioral symptoms characteristic of ADHD. For example, consistent evidence of executive function deficits in children with ADHD have been shown at the group level but the sensitivity and specificity of any single deficit is not high enough to support any single dysfunction that causes all cases of ADHD (e.g., Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005; Pennington, 2005).

As a result, it seems plausible that multiple causal pathways involving different neurocognitive deficits and environmental factors lead to the behavioral manifestation of ADHD. Recent theories of ADHD implicate both executive and motivational processes in ADHD (Sonuga-Barke, 2002), as well as interactions between these processes (Castellanos, Sonuga-Barke, Milham, & Tannock, 2006). Thus, there has been a shift away from theories of individual core processes towards multi-process models of ADHD (e.g., Castellanos & Tannock, 2002; Nigg, 2003; Sonuga-Barke, 2002).

Although the role of working memory in ADHD has been studied for over a decade (Douglas & Benezra, 1990), it is among the more recent additions to the list of *core* neurocognitive processes implicated in ADHD (e.g., Castellanos & Tannock, 2002). Working memory maintains temporary, active representations of information for further processing or recall and is thought to underlie a wide range of mental activities such as problem-solving, decision-making, reading, and arithmetic (Baddeley, 2003). According to the three-component model of working memory proposed by Baddeley and Hitch (1974), working memory involves separate auditory-verbal and visual-spatial storage systems that are secondary to the central executive domain. The auditory-verbal storage system, referred to as the phonological loop, maintains linguistic information. Deficits in this component of working memory are thought to result in poor vocabulary, difficulty with word decoding, and language acquisition weakness. The visual-spatial system handles the spatial location of items and is associated with literacy, comprehension, and arithmetic. The central executive is a control unit that manipulates information stored in the auditory-verbal and visual-spatial systems and simultaneously accesses long term memory to complete complex cognitive activities (Baddeley, 2003). The storage systems are typically assessed via forward span, which involves maintaining information in memory and recalling that information in the same sequence, whereas backward span requires manipulation of information, and assesses the central executive system (Kaplan, Fein, Kramer, Delis, & Morris, 1999; Milner, 1971).

The empirical evidence for a working memory deficit in ADHD was previously limited to auditory-verbal working memory, with a comprehensive review by Pennington and Ozonoff (1996) concluding that ADHD is not associated with a deficit in auditory-verbal working memory. Measures of visual-spatial working memory were largely unstudied at that time. Although not all studies have found visual-spatial working memory to be deficient in children with ADHD (Cohen et al., 2000; Geurts et al., 2004; Jonsdottir, Bouma, Sergeant, & Scherder, 2005), the results of two recent meta-analytic studies (Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005) suggest that auditory-verbal and visual-spatial working memory impairments are evident in children with ADHD, independent of intelligence and academic achievement, with the most robust deficits in visual-spatial working memory (both the storage and manipulation components). In a more recent study by Martinussen and Tannock (2006), children with ADHD without comorbid language learning disorders exhibited deficits in visual-spatial storage and auditory-verbal and visual-spatial central executive functions that were independent of comorbid psychiatric disorders. Indeed, the findings for visual-spatial working memory deficits constitute the largest effect in the literature for an ADHD neuropsychological weakness, with a pooled effect size of Cohen's $d = 1.06$ (Cohen, 1988; Nigg, 2006). In addition to the empirical evidence for visual-spatial working memory deficits in ADHD, brain regions that support executive functions have been implicated in the pathophysiology of ADHD, with converging evidence for dysfunction of dopaminergic and noradrenergic pathways in the prefrontal cortex and subcortical regions (Biederman & Spencer, 1999; Castellanos, 1997).

If visual-spatial working memory is truly a core process in ADHD, it should be influenced by effective treatments for the disorder, and improvements in visual-spatial working memory should be related to clinical improvement. The most effective clinical treatments for ADHD

are behavior therapy, stimulants, and their combination (American Academy of Pediatrics [AAP], 2001). Initial evidence suggests that stimulant medication improves both visual-spatial and auditory-verbal working memory in children with ADHD (Bedard, Martinussen, Ickowicz, & Tannock, 2004; Kempton et al., 1999; Mehta, Goodyear & Sahakian, 2004; Tannock, Ickowicz, & Schachar, 1995; Zeiner, Bryhn, Bjercke, Truyen, & Strand, 1999). For example, methylphenidate (MPH) significantly improved storage of visual-spatial information on the CANTAB Spatial Span Forward task and the Finger Windows Forward task (Bedard et al., 2004). Although Bedard et al. (2004) did not find MPH to improve manipulation of visual-spatial information (Finger Windows Backward task), such an effect was observed on a different task in a more recent study (Bedard, Jain, Hogg-Johnson, & Tannock, 2007). In addition, Kempton et al. (1999) have found stimulant medication to improve manipulation of visual-spatial information (i.e., updating of memory sequence) in children with ADHD. Bedard and colleagues (2007) suggest that the effects of MPH on visual-spatial working memory may be component-specific, and even instrument-specific.

Despite the large number of studies showing that behavioral treatments improve functioning in ADHD (Pelham & Fabiano, in press), and in contrast to the growing literature on the effects of medication on working memory, there is virtually no research examining the impact of behavior therapy on working memory among children with ADHD. However, Klingberg and colleagues (2005) recently reported that systematic practice of computerized working memory exercises over a 5 to 6-week period improved standard measures of working memory in 7 to 12-year-old boys with ADHD, suggesting that working memory can be improved without medication. Furthermore, children in the treatment group exhibited a significant reduction in parent-reported symptoms of inattention and hyperactivity/impulsivity. These findings are promising but did not address the effects of actual behavioral contingencies on working memory performance but rather the effects of repeated practice on working memory and symptomatology. Contingencies, in the form of reinforcement and response cost, are central in behavioral treatments of ADHD (Pelham & Waschbusch, 1999), and behavior therapy improves the functioning of children with ADHD across home and school settings (Pelham & Fabiano, in press; Pelham, Wheeler, & Chronis, 1998).

In addition to the practical significance of behavior therapy, the role of motivation and sensitivity to reinforcement and punishment in ADHD is of considerable theoretical significance (e.g., Douglas, 1989; Haenlein & Caul, 1987; Quay, 1988). Recent models of ADHD emphasize the timing of reinforcement (Sagvolden, Johansen, Aase & Russell, 2005; Sonuga-Barke, 2002), suggesting that ADHD symptoms result from a steepened delay-of-reinforcement gradient or heightened aversion to delay. According to these theories, deficits in children with ADHD are expected to be ameliorated when reinforcers are powerful, frequent, and relatively immediate (Sagvolden et al., 2005). In addition, children with ADHD may be particularly sensitive to the removal of rewards, resulting in a decrement in performance upon removal of reward possibly due to frustration (Douglas & Parry, 1994). Thus, the question of whether motivational incentives improve working memory has both clinical and theoretical implications.

The motivational and executive processes implicated in ADHD are often framed as independent, but there are important reasons to expect interactions among these processes. It has been suggested that motivation and cognition are integrated via connections between ventral frontal brain regions involved in the processing of reward-related stimuli and dorsolateral prefrontal brain regions associated with cognitive processes (Gilbert & Fiez, 2004; Haber, Fudge, & McFarland, 2000). The interaction between cognition and motivation may be revealed by examining the effects of reinforcement on cognitive processes in the laboratory as an analogue of behavior therapy. Such reinforcement improves several aspects

of cognitive performance in children with ADHD, namely response inhibition and accuracy (see review by Luman, Oosterlaan & Sergeant, 2005).

As noted above, however, none of these studies specifically assessed the effects of reinforcement on working memory. The purpose of this study was to provide an initial test of the hypothesis that continuous reinforcement (points awarded for correct performance, coupled with trial-by-trial feedback) enhances visual-spatial working memory in children with ADHD, relative to performance under typical testing conditions when children are asked to try their best. Because removal of incentives may be a more powerful manipulation than the addition of incentives among children with ADHD, we counterbalanced the order of incentive presentation across participants.

Method

Participants

Participants were 21 children (3 female) between the ages of 7.1 and 10.1 years (mean age of 8.8 years) who received a *DSM-IV* diagnosis of ADHD Combined type. All participants were enrolled in a Summer Treatment Program (STP; Pelham, Fabiano, Gnagy, Greiner, & Hoza, 2005) conducted at the Center for Children and Families at the State University of New York at Buffalo. The STP is a behavioral treatment program in which evidence-based treatments for ADHD are implemented across recreational and academic settings. The children attended the STP from 8:00 a.m. to 5:00 p.m., Monday through Friday, for 4 weeks, and participated in the current study at various points during the 4-week STP. Children were recruited for this STP via radio advertisements, flyers sent to families who participated in previous research, and clinic referrals. Participation in the present study was voluntary, with parental consent and participant assent obtained in accordance with procedures approved by the University at Buffalo and the Women's and Children's Hospital of Buffalo.

During this study, all participants were tested while unmedicated. Fourteen children had discontinued medication for the summer months. Seven children were taking stimulant medication regularly but refrained from taking medication on the day of the assessment. The minimum amount of time since the last dose of medication was approximately 15 hours.

Diagnostic Assessment

All participants had a *DSM-IV* (APA, 1994) diagnosis of ADHD confirmed by a comprehensive clinical diagnostic assessment. The assessment involved a structured clinical interview with one or both parents (Diagnostic Interview Schedule for Children Version IV, DISC-IV; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000). Parents and teachers also completed rating scales on the child's behavior (Disruptive Behavior Disorder (DBD) rating scales; Pelham, Gnagy, Greenslade, & Milich, 1992; Pelham, Fabiano, & Massetti, 2005) as well as reports of functional impairment (Impairment Ratings Scale (IRS); Fabiano et al., 2006). Diagnosis of externalizing disorders was based on the information obtained from these measures and supplemental information from a clinical interview in addition to independent agreement by two diagnosticians. Children met criteria for ADHD-Combined type based on parent and teacher reports indicating at least 6 inattentive symptoms and 6 hyperactive-impulsive symptoms, with at least 1 symptom occurring both at home and at school, each at clinically significant levels (i.e., rating of pretty much or very much), as in earlier work (Masseti et al., 2007; Waschbusch, Craig, Pelham, & King, 2007). Patterns of comorbidity were typical: Twelve participants (57.1%) met criteria for Oppositional Defiant Disorder, two participants (9.5%) met criteria for Conduct Disorder, and eight children (38%) met criteria for one or more anxiety disorders.

Standardized measures of intellectual ability and achievement included the Vocabulary and Block Design subtests from the Wechsler Intelligence Scale for Children, Third (WISC-III; Kaplan et al., 1999) and Fourth (WISC-IV Integrated; Kaplan, Fein, Kramer, Delis, & Morris, 2004) Editions and the Letter-Word Identification, Calculation, and Spelling subtests from the Woodcock-Johnson Test of Achievement, Third Edition (WJTA-III; Woodcock, McGrew, & Mather, 2001). Participants were classified as learning disabled if they received a score of less than 80 on any of the achievement measures, which resulted in the classification of one child. The mean estimated IQ for this sample was 115.3 (SD = 18.8). Participants also passed routine audiometric and visual screening. Exclusionary criteria included the following: (1) an estimated Full Scale IQ score of less than 80, (2) history of seizures or other neurological problems, (3) history of other medical or psychological problems for whom withdrawal of psychostimulant treatment for testing involved considerable risk, (4) hearing or vision problems, (5) childhood history of concurrent diagnosis of pervasive developmental disorder, schizophrenia or other psychotic disorder, eating disorder, or any other psychiatric illness requiring medication other than ADHD, and (6) children who did not exhibit functional impairment.

Spatial Span Task

Visual-spatial working memory was assessed via a computerized adaptation of the Corsi's block tapping task (Milner, 1971) and Spatial Span subtest from the WISC-IV Integrated (Kaplan et al., 2004) that incorporated features of the Spatial Span task from the CANTAB (Luciana, 2003). The task was programmed in E-Prime 1.1 (Psychology Software Tools, Pittsburgh, PA; the control file is available from the authors). In this task, an array of ten white squares on a black background is presented on the computer screen (see Fig. 1a.). On each trial, a yellow smiley face appears in two to eight of the squares at a rate of one square per second. For forward span, which assesses short-term storage or maintenance of visual-spatial information (Kaplan et al., 1999), children were instructed to use a computer mouse to click on the squares in the same order in which the smiley face appeared. For backward span, participants were asked to click on the squares in the reverse order in which the smiley face appeared. Backward span measures the manipulation of visual-spatial information (Kaplan et al., 1999), requiring participants to update and reorder the stimuli, whereas manipulation of information is not necessary for forward span.

Forward span was always administered before backward span, as with the Spatial Span subtest from the WISC-IV Integrated (Kaplan et al., 2004). For each direction (forward and backward), there were two trials at each level of difficulty, beginning with 2-location sequences and advancing to a maximum of 8-location sequences. The task terminated when both trials within a difficulty level were incorrect. Performance on both forward and backward Spatial Span tasks was determined by calculating the total number of trials completed correctly.

During the incentive condition, visual feedback was presented after each trial in the form of words indicating a correct or incorrect response, the amount of points earned for that trial, and the total points earned for all previous trials (see Fig. 1b). Children received 25 points for each correct trial and zero points for incorrect trials. Each point had an approximate value of one U.S. cent. The no-incentive condition did not provide feedback on performance or points after each trial and simply prompted the child to begin the next trial.

Procedure

Children were escorted from a non-preferred STP activity to the testing room. (Children were not removed from their preferred activities in order to avoid frustration.) The following laboratory rules were described and operationalized: follow directions, stay in assigned area, use materials appropriately, and try your best. Children were awarded a 50-point behavioral

bonus at the end of the testing session for compliance with these rules. Children lost 25 points per rule violation after an initial warning. The laboratory point store was initially introduced to the child during the assent procedure and was mentioned again prior to testing to remind children that all points earned could be exchanged for prizes in the store.

The Spatial Span task (forward and backward) was administered twice in one laboratory session, once with feedback and incentives and once without. Participants were seated at eye level with the computer monitor and were instructed to respond with their dominant hand and to place the other hand in their lap. Children were not allowed to use their hand as an aid in tracking the sequence of target boxes, and the mouse pointer did not appear on the screen during sequence presentations. A research assistant remained seated behind the participant throughout the session.

Participants were randomly assigned to receive either the incentive or no-incentive condition first since the order in which the incentives are presented may influence performance (Corr, 2002; Douglas & Parry, 1994). Table 1 presents sample characteristics for each incentive order. To ensure that participants understood the task, forward and backward span each began with a 2-location practice sequence, repeated until accurately completed, in both the incentive and no-incentive conditions. During the practice sequence prior to the incentive condition, participants received feedback and points although they did not keep these points, whereas they did not receive reinforcement during the practice prior to the no-incentive condition. Within an incentive condition, the same set of location sequences (from 5 possible sets) was employed for forward and backward span, as is typical for Spatial Span test from the WISC-IV Integrated (Kaplan et al., 2004). Location sequence was changed from one incentive condition to the next to minimize practice effects.

The average time for completion of the Spatial Span task was fifteen minutes. Following the task, points were exchanged for toys, games, or gift cards at a small laboratory “point store”. Trial-by-trial feedback and the rapid conversion of points were considered important in light of hypothesized problems with delay of reinforcement in ADHD (Sagvolden et al., 2005; Sonuga-Barke, 2002).

Data Analysis

Separate 2×2 ANOVAs were used for forward and backward span to assess the impact of the within-subjects Incentive factor (incentive v. no-incentive) and the between-subjects Incentive Order factor (incentive first v. no-incentive first). As a measure of effect size, Cohen's d (Cohen, 1988) was reported. The primary dependent measure was the number of trials correct, with possible scores ranging from 0 to 14.

Preliminary analyses indicated that, consistent with random assignment to incentive order, the subgroups by incentive order did not reliably differ in the proportion of subjects who were female or who had co-occurring ODD, CD, or an Anxiety disorder, χ^2 s < 2.3, ps > .12, nor did they differ in age IQ, or achievement scores, F s < 1. In addition, the subgroups by incentive order did not differ in the number of ADHD symptoms endorsed by parents and teachers on the DBD rating scale, F s < 1.3.

Results

Task Performance

As can be seen in Figure 2, performance on forward span was not enhanced in the incentive compared to the no-incentive condition. In fact, the means were in the direction opposite to the predicted direction, $F(1, 19) = 1.6$, $p > .5$, $d = .27$. The order in which the participants

experienced the incentive conditions had no discernable effect on forward span performance, incentive order and Incentive Order X Incentive $F_s < 1$, $d's < .17$.

In contrast, performance on backward span was significantly enhanced under incentive compared to the no-incentive condition, $F(1, 19) = 4.6$, $p < .05$, $d = .47$, as shown in Figure 3. Although this incentive effect for backward span did not reliably depend on the order in which incentives were presented, Incentive Order X Incentive $F(1, 19) = 2.0$, $p = .17$, unprotected post-hoc tests suggested that the enhancement by incentive was driven by participants who received the incentive first, $F(1, 19) = 6.0$, $p < .05$, $d = .70$, relative to those who received no-incentive first, $F < 1$, $d = .19$.

In sum, the presence of a performance-based incentive improved performance on backward span, measuring manipulation of visual-spatial information, whereas performance on forward span was not affected.

Supplementary analyses: Performance over time

Although the finding of an incentive effects on backward span but not forward span could be due to a specific effect on the manipulation component of working memory, it is also the case that backward span always came after forward span. Thus, it is possible that incentives prevented a decline in performance over time, commonly referred to as a vigilance decrement. Although the Spatial Span task is not a traditional vigilance task, examining performance over time allows us to better evaluate this alternative hypothesis. To this end, the number of trials completed correctly in each block, regardless of incentive condition or span direction, was analyzed using repeated measures ANOVA with Block (4 levels) as a within-subjects factor and Incentive Order as a between-subjects factor. Orthogonal polynomial contrasts were employed to examine the linear, quadratic and cubic trends in performance across blocks.

Fig. 4 shows the mean number of trials correct for each block within each incentive order group. The number of trials correct decreased linearly across blocks, $F(1, 19) = 6.6$, $p < .05$, $d = .55$, and the multiple reversals of span direction resulted in a marginal cubic change across blocks, $F(1, 19) = 4.0$, $p = .06$, $d = .42$. Furthermore, the decline in performance over time depended on the order of the incentive condition, Incentive Order x Block quadratic, $F(1, 19) = 4.9$, $p < .05$. For participants in the incentive first condition, the quadratic effect of time is marginal, $F(1, 19) = 3.5$, $p = .08$, with pairwise comparisons showing that the last block is reliably different from the three previous blocks, $ps = .007$, $.024$, $.016$, for blocks 1, 2, and 3, respectively. Participants in the no-incentive first condition, did not demonstrate a quadratic change in performance across blocks, $F < 1$; unprotected tests revealed that although there was a significant decrease from block 1 to block 2, $p = .02$, none of the remaining blocks reliably differ from one another, $ps > .20$. The supplementary analyses suggest that the presence of a performance-based incentive prevented a vigilance decrement that occurred in the absence of this incentive.

Behavior Bonus

The majority of children were well-behaved during the testing session. Four of the children (19%) lost 25 behavior bonus points, and one child lost all 50 points, for violations of the basic lab rules. All of the participants who lost behavior bonus points for rule violations received the no-incentive condition first, resulting in a reliable difference between incentive order groups in the number of children who lost behavior points, $\chi^2 = 6.0$, $p < .02$.

Discussion

Working memory has been increasingly implicated as a core process in ADHD, with recent reviews focusing on impairments in visual-spatial working memory (Martinussen et al., 2005; Willcutt et al., 2005). Several studies have demonstrated that stimulant medication improves working memory in children with ADHD, whereas the impact of behavioral interventions has not previously been evaluated. The current study provides the first test of whether incentives, a laboratory analogue of behavior therapy, improve visual-spatial working memory among children with ADHD. The results suggest support for this hypothesis because incentives improved performance on a component of visual-spatial working memory in unmedicated children with ADHD. Importantly, this improvement with incentives was limited to backward span, which involves manipulation of information and is thought to reflect visual-spatial central executive functioning, whereas performance on forward span, a measure of short-term storage, did not improve with trial-wise feedback and rewards for performance. Moreover, the present study provides preliminary evidence that incentives interact with time on task and task difficulty to prevent a predictable decline in performance over time, or a vigilance decrement, particularly when the task is most difficult.

These findings are broadly consistent with research on the impact of reinforcement contingencies on other cognitive processes (see Luman et al., 2005). Research has shown that rewards improve performance of children with ADHD on measures of response inhibition and accuracy, with some studies demonstrating that children with ADHD perform as well as control children when rewards are present. The Spatial Span task used in this study is thought to assess separate components of working memory, specifically visual-spatial storage and manipulation. Interestingly, incentives were only found to improve the manipulation component of working memory, which is considered to reflect executive system functioning. Thus, these findings provide further support for the interaction of motivational and executive processes as well as the effectiveness of behavioral interventions on improving a putative core deficit in ADHD.

The differential incentive effects for forward and backward span were unexpected and the reasons for these findings remain unclear. In order to understand these results, it is necessary to consider the cognitive processes required for each of these tasks. Forward span only requires storage and recall of the information whereas the backward span has an additional component of manipulation of the stored information. According to Martinussen et al. (2005), several studies have shown that children with ADHD exhibit large impairments in both the visual-spatial storage as well as the visual-spatial central executive domains (i.e., manipulation). Therefore, we predicted that incentives would improve both types of processing. However, it may be that the processes required for manipulation of information are more sensitive to effort and motivation than those required for memory storage. Conversely, Bedard and colleagues (2004) found that methylphenidate improved performance on a measure of visual-spatial storage but not manipulation in children with ADHD. Similarly, in a very recent study, Bedard and colleagues (2007) found that methylphenidate improved storage of visual-spatial information irrespective of the instrument used to assess working memory, whereas the impact of stimulant medication on visual-spatial manipulation was less consistent (i.e., significantly improved performance on one measure of visual-spatial manipulation but not another). Thus, it is possible that stimulant medication and incentives preferentially enhance different components of visual-spatial working memory, such that incentives improve central executive functioning whereas medication improves storage. Accordingly, it seems plausible that the combination of stimulant medication and incentives would result in the greatest overall improvement in visual-spatial working memory in children with ADHD. Of course, this hypothesis is speculative, but it seems important more generally to test the separate and interactive effects of medication and incentives, given the theoretical and clinical relevance of combined interventions. Indeed, surprisingly few studies have examined the concurrent effects

of incentives and medication on cognitive performance in ADHD (c.f., Solanto, Wender, & Bartell, 1997), and future work should aim to fill this important gap in the literature.

Additional issues that may be contributing to the differential incentive effects include task difficulty, span order, and incentive order. Intuitively, it seems that more difficult tasks would require more extrinsic reinforcement (i.e., incentives for performance) since easier tasks can be accomplished with minimal effort. Although more difficult tasks have also been conceptualized as more intrinsically motivating than those that are less demanding among healthy adults (Tomporski & Tinsley, 1996), the current findings provide initial support for the hypothesis that extrinsic reinforcement results in greater improvement on more difficult tasks, at least among children with ADHD.

Now that we have demonstrated that some aspects of working memory are influenced by incentives among children with ADHD, future work should include control groups to determine whether these effects are specific to ADHD. Perhaps children without ADHD have higher levels of intrinsic motivation, and are therefore sufficiently motivated by the increased task demands whereas children with ADHD are not. Furthermore, perhaps task difficulty increases intrinsic reinforcement to certain point, but once that threshold is crossed extrinsic reinforcement is necessary to avoid performance declines and/or frustration. More generally, the addition of control groups to future work on both incentives and medication will aid in determining the degree to which such treatments normalize working memory among children with ADHD.

We examined whether incentive effects were dependent upon the order in which incentive and no-incentive conditions were provided. Although such effects are rarely tested (or at least rarely reported), recent findings of incentive order effects on inhibitory control among children with ADHD (Huang-Pollock, Mikami, Pfiffner, & McBurnett, 2007) call attention to the methodological and theoretical importance of such effects in ADHD research. Though the interaction of incentives and incentive order fell short of statistical significance, close inspection of the data suggested that the incentive effect on the backward span task was driven primarily by those who experienced the incentive condition first. In the incentive first group, the no-incentive block may be subjectively experienced as a form of punishment since children were initially rewarded for certain behaviors that they subsequently were asked to perform without reward. According to Gray (1982) and others (Corr, 2002; Douglas & Parry, 1994), removal of reward (i.e., extinction) is essentially equivalent to punishment. Thus, the decline in performance in children with ADHD when rewards are omitted may reflect frustration, either due to excessive sensitivity to punishment or reward, or possibly an interaction between these motivational systems postulated by Gray (Corr, 2002; Gray, 1982; see Quay, 1988, for a discussion of motivational systems in disruptive behavior disorders). In order to determine if the decline in performance is due to frustrative nonreward, it would be important to reintroduce incentives in a later block to see if performance improves once again.

Notably, the behavior bonus data do not support a frustrative nonreward explanation, since only the participants in the no-incentive first group lost points for rule violations during the testing session. If frustrative nonreward was a factor that contributed to a decline in performance during the no-incentive block for the incentive first group, we would have also expected a greater number of rule violations in this group.

Another plausible explanation is that span order may have contributed to the differential effect of incentives. The computerized Spatial Span task used in this study was designed to resemble the Spatial Span subtest from the WISC-IV Integrated (Kaplan et al., 2004), in which forward span is always administered first, followed by backward span. Since span direction (i.e., forward or backward) was confounded with time on task (i.e., backward span always came

after forward span), the presence of a motivational incentive may have prevented a vigilance decrement, or a decrement in performance due to the time spent on a task. Deficits in vigilance or sustained attention in ADHD are debatable. Although several studies suggest a significant decline in performance when prolonged attention is required (Corkum, Schachar, & Siegel, 1996; Solanto et al., 1997), and that this decline is greater in children with ADHD than control children (Huang-Pollack, Nigg, & Halperin, 2006), others have concluded that sustained attention is intact in ADHD (Nigg, 2006). Though the Spatial Span task is not intended to serve as a measure of vigilance, the way in which the task was structured suggests that incentives may have prevented a decrease in performance during the backward span (since it always occurred after the forward span) that would typically occur in the absence of that incentive. This would also be consistent with the Block x Incentive Order interaction from the supplementary analyses. If incentives did prevent a vigilance decrement, this may provide further support for cognitive-energetic deficits in children with ADHD (Sergeant, Oosterlaan, & van der Meere, 1999; Sergeant, 2005). According to the cognitive-energetic model (Sanders, 1983; Sergeant et al., 1999), performance deficits in ADHD children may be linked to *state* factors, including effort, arousal, and activation. Effort in this model refers to the necessary energy to meet the demands of the task and encompasses terms such as motivation and response to contingencies. Thus, a vigilance decrement may have been due to diminished effort as a function of time on task which was improved with incentives. In future research, it will be important to counterbalance the order in which the span tasks are completed to disentangle the role of working memory processing requirements and vigilance (i.e., time on task).

Future work in this area should address several additional issues. First, as noted above, the inclusion of a control group is necessary to determine whether incentives differentially improve performance for children with ADHD and would provide a standard against which to evaluate the importance of the incentive effect. Second, replication of these findings with multiple measures of working memory will reveal whether the incentives improve working memory in general, or whether the effects seen in this study are specific to this task (c.f., studies of medication; Bedard et al., 2007). More broadly, including measures of additional core constructs (e.g., sustained attention) would be helpful given the substantial overlap in these constructs (Castellanos & Tannock, 2002). Third, the present work employed a compound reinforcer, contingent feedback and points, to ensure that the incentive manipulation was relatively strong and immediate. However, work that examines the separate impact of reinforcement from feedback and points will be interesting, particularly in light of the putative role of delay-of-reinforcement gradients in ADHD (e.g., Sagvolden et al., 2005). Finally, the present findings are based on a small sample and replication within a larger sample is important. A larger sample would also permit the examination of differential incentive effects as a function of symptom dimensions or subtypes, as well as comorbidity.

The finding that reinforcement contingencies improve visual-spatial working memory among children with ADHD may have important treatment implications. For example, Milch-Reich and colleagues (Milch-Reich, Campbell, Pelham, Connelly, & Geva, 1999) found that children with ADHD had difficulty telling stories about social interactions, relative to controls, only when they were required to use working memory (i.e., cues presented sequentially rather than simultaneously) and this deficit was not alleviated with medication. In addition, among psychosocial interventions, only contingency management programs have been shown to change social behaviors in children with ADHD (Pelham & Fabiano, in press), suggesting that incentive programs in real life might have effects comparable to those obtained on laboratory tasks. Furthermore, improvement in working memory among children with ADHD was shown to be related to a significant reduction in parent-reported symptoms of inattention and hyperactivity/impulsivity (Klingberg et al., 2005). In sum, the role of working memory in social functioning as well as the relationship between improvements in working memory and reduced

symptomatology suggests that working memory may serve as a potential mediator in treatment response.

Summary

The use of motivational incentives in the form of continuous reinforcement and feedback improved manipulation of visual-spatial information (backward spatial span), though not storage (forward spatial span), in unmedicated children with ADHD. Incentives may selectively improve certain components of working memory in ADHD, though additional work is needed to rule out alternative vigilance or frustration interpretations. More generally, the present study provides initial evidence that motivational incentives have a beneficial impact on some aspects of visual-spatial working memory among children with ADHD, opening the door to future work on the role of this putative core process in treatment response.

Acknowledgements

We thank Mark Kutowski for programming the task and Rebecca Ashare for providing feedback on drafts of the manuscript. This research was supported by grant MH069434 from the National Institute of Mental Health.

References

- American Academy of Pediatrics. Clinical practice guidelines: treatment of the school-age child with attention-deficit/hyperactivity disorder. *Pediatrics* 2001;108:1–16. [PubMed: 11433046]
- American Psychiatric Association. Diagnostic and statistical manual of mental disorders. Vol. 4th. Washington, DC: Author; 1994.
- Baddeley A. Working memory: Looking back and looking forward. *Nature Reviews Neuroscience* 2003;4(10):829–839.
- Baddeley, A.; Hitch, GJ. Recent Advances in Learning and Motivation. Bower, GA., editor. New York: Academic; 1974. p. 47–89.
- Bedard AC, Jain U, Hogg-Johnson S, Tannock R. Effects of methylphenidate on working memory components: influence of measurement. *Journal of Child Psychology and Psychiatry* 2007;48:872–880. [PubMed: 17714372]
- Bedard AC, Martinussen R, Ickowicz A, Tannock R. Methylphenidate improves visual spatial memory in children with Attention-Deficit/Hyperactivity Disorder. *Journal of the American Academy of Child and Adolescent Psychiatry* 2004;43:260–268. [PubMed: 15076258]
- Biederman J, Spencer T. Attention-deficit/hyperactivity disorder (ADHD) as a noradrenergic disorder. *Biological Psychiatry* 1999;46(9):1234–1242. [PubMed: 10560028]
- Castellanos FX. Toward a pathophysiology of attention-deficit/hyperactivity disorder. *Clinical Pediatrics* 1997;36(7):381–393. [PubMed: 9241475]
- Castellanos FX, Sonuga-Barke EJS, Milham MP, Tannock R. Characterizing cognition in ADHD: beyond executive dysfunction. *Trends in Cognitive Sciences* 2006;10(3):117–123. [PubMed: 16460990]
- Castellanos FX, Tannock R. Neuroscience of attention-deficit/hyperactivity disorder: the search for endophenotypes. *Nature Reviews Neuroscience* 2002;3(8):617–628.
- Cohen NJ, Vallance DD, Barwick M, Im N, Menna R, Horodezky NB, et al. The interface between ADHD and language impairment: an examination of language, achievement, and cognitive processing. *Journal of Child Psychology and Psychiatry* 2000;41(3):353–362. [PubMed: 10784082]
- Cohen, J. Statistical power analysis for the behavioral sciences. Vol. 2nd. Hillsdale, NJ: Erlbaum; 1988.
- Corkum PV, Schachar R, Siegel LS. Performance on the continuous performance task and the impact of reward. *Journal of Attention Disorders* 1996;1(2):114–121.
- Corr PJ. J.A. Gray's reinforcement sensitivity theory and frustrative nonreward: a theoretical note on expectancies in reactions to rewarding stimuli. *Personality and Individual Differences* 2002;32(7):1247–1253.
- Douglas, VI. Can Skinnerian theory explain attention deficit disorder? A reply to Barkley. In: Bloomington, LL.; Sergeant, J., editors. *Attention Deficit Disorder: Current concepts and emerging*

trends in attentional and behavioral disorders of childhood. Oxford: Pergamon Press; 1989. p. 235-254.

Douglas VI, Benezra E. Supraspan verbal memory in attention deficit disorder with hyperactivity normal and reading-disabled boys. *Journal of Abnormal Child Psychology* 1990;18(6):617–638. [PubMed: 2074344]

Douglas V, Parry P. Effects of reward and nonreward on frustration and attention in attention deficit disorder. *Journal of Abnormal Child Psychology* 1994;22(3):281–302. [PubMed: 8064034]

Fabiano GA, Pelham JWE, Waschbusch DA, Gnagy EM, Lahey BB, Chronis AM, et al. A Practical Measure of Impairment: Psychometric Properties of the Impairment Rating Scale in Samples of Children With Attention Deficit Hyperactivity Disorder and Two School-Based Samples. *Journal of Clinical Child and Adolescent Psychology* 2006;35(3):369–385. [PubMed: 16836475]

Gray, JA. The neuropsychology of anxiety: an enquiry into the functions of the septo-hippocampal system. New York: Oxford University Press; 1982.

Gilbert AM, Fiez JA. Integrating rewards and cognition in the frontal cortex. *Cognitive, Affective, & Behavioral Neuroscience* 2004;4(4):540–552.

Geurts HM, Verte S, Oosterlaan J, Roeyers H, Hartman CA, Mulder EJ, et al. Can the Children's Communication Checklist differentiate between children with autism, children with ADHD, and normal controls? *Journal of Child Psychology and Psychiatry* 2004;45(8):1437–1453. [PubMed: 15482504]

Haber SN, Fudge JL, McFarland NR. Striatonigrostriatal pathways in primates form an ascending spiral from the shell to the dorsolateral striatum. *The Journal of Neuroscience* 2000;20(6):2369–2382. [PubMed: 10704511]

Haenlein M, Caul WF. Attention deficit disorder with hyperactivity: a specific hypothesis of reward dysfunction. *Journal of the American Academy of Child and Adolescent Psychiatry* 1987;26(3):356–362. [PubMed: 3597291]

Huang-Pollock CL, Mikami AY, Pfiffner L, McBurnett K. ADHD Subtype Differences in Motivational Responsivity but not Inhibitory Control: Evidence From a Reward-Based Variation of the Stop Signal Paradigm. *Journal of Clinical Child and Adolescent Psychology* 2007;36(2):127–136. [PubMed: 17484686]

Huang-Pollock CL, Nigg JT, Halperin JM. Single dissociation findings of ADHD deficits in vigilance but not anterior or posterior attention systems. *Neuropsychology* 2006;20(4):420–429. [PubMed: 16846260]

Jonsdottir S, Bouma A, Sergeant JA, Scherder EJ. The impact of specific language impairment on working memory in children with ADHD combined subtype. *Archives of Clinical Neuropsychology* 2005;20(4):443–456. [PubMed: 15896559]

Kaplan, E.; Fein, D.; Kramer, J.; Delis, D.; Morris, R. WISC-III PI Manual. San Antonio, TX: The Psychological Corporation; 1999.

Kaplan, E.; Fein, D.; Kramer, J.; Delis, D.; Morris, R. WISC-IV Integrated. San Antonio, TX: The Psychological Corporation; 2004.

Kempton S, Vance A, Maruff P, Luk E, Costin J, Pantelis C. Executive function and attention deficit hyperactivity disorder: stimulant medication and better executive function performance in children. *Psychological Medicine* 1999;29(3):527–538. [PubMed: 10405075]

Klingberg T, Fernell E, Olesen PJ, Johnson M, Gustafsson P, Dahlstrom K, et al. Computerized training of working memory in children with ADHD—a randomized, controlled trial. *Journal of the American Academy of Child and Adolescent Psychiatry* 2005;44(2):177–186. [PubMed: 15689731]

Luciana M. Practitioner review: computerized assessment of neuropsychological function in children: clinical and research applications of the Cambridge Neuropsychological Testing Automated Battery (CANTAB). *Journal of Child Psychology and Psychiatry* 2003;44:649–663. [PubMed: 12831110]

Luman M, Oosterlaan J, Sergeant J. The impact of reinforcement contingencies on AD/HD: A review and theoretical appraisal. *Clinical Psychology Review* 2005;25:183–213. [PubMed: 15642646]

Martinussen R, Hayden D, Hogg-Johnson S, Tannock R. A Meta-Analysis of Working Memory Impairments in Children with Attention-Deficit/Hyperactivity Disorder. *Journal of the American Academy of Child and Adolescent Psychiatry* 2005;44(4):377–384. [PubMed: 15782085]

- Martinussen R, Tannock R. Working memory impairments in children with attention-deficit hyperactivity disorder with and without comorbid language learning disorders. *Journal of Clinical and Experimental Neuropsychology* 2006;28(7):1073–1094. [PubMed: 16840237]
- Massetti GM, Lahey BB, Pelham WE, Loney J, Ehrhardt A, Lee SS, et al. Academic Achievement Over 8 Years Among Children Who Met Modified Criteria for Attention-deficit/Hyperactivity Disorder at 4–6 Years of Age. *Journal of Abnormal Child Psychology*. 2007
- Mehta MA, Goodyer IM, Sahakian BJ. Methylphenidate improves working memory and set-shifting in AD/HD: relationships to baseline memory capacity. *Journal of Child Psychology and Psychiatry* 2004;45(2):293–305. [PubMed: 14982243]
- Milch-Reich S, Campbell SB, Pelham WE, Connelly L, Geva D. Developmental and individual differences in children's on-line representations of dynamic social events. *Child Development* 1999;70:413–431. [PubMed: 10218263]
- Milner B. Interhemispheric differences in the localization of psychological processes in man. *British Medical Bulletin* 1971;27:272–277. [PubMed: 4937273]
- Nigg JT. Response inhibition and disruptive behaviors: toward a multiprocess conception of etiological heterogeneity for ADHD combined type and conduct disorder early-onset type. *Annals of the New York Academy of Science* 2003;1008:170–182.
- Nigg JT, Willcutt EG, Doyle AE, Sonuga-Barke EJS. Causal heterogeneity in ADHD: Do we need neuropsychologically impaired subtypes? *Biological Psychiatry* 2005;57:1224–1230. [PubMed: 15949992]
- Nigg, JT. *What Causes ADHD? Understanding what goes wrong and why*. New York: Guilford Press; 2006.
- Pelham WE Jr, Fabiano GA. Evidence-based psychosocial treatment for attention-deficit/hyperactivity disorder: An update. *Journal of Clinical Child and Adolescent Psychology*. in press
- Pelham, WE.; Fabiano, GA.; Gnagy, EM.; Greiner, AR.; Hoza, B. The role of summer treatment programs in the context of comprehensive treatment for ADHD. In: Hibbs, E.; Jensen, P., editors. *Psychosocial treatments for child and adolescent disorders: Empirically based strategies for clinical practice*. Washington D.C.: APA Press; 2005. p. 377–410.
- Pelham WE Jr, Fabiano GA, Massetti GM. Evidence-based assessment of attention deficit hyperactivity disorder in children and adolescents. *Journal of Clinical Child and Adolescent Psychology* 2005;34(3):449–476. [PubMed: 16026214]
- Pelham WE Jr, Gnagy EM, Greenslade KE, Milich R. Teacher ratings of DSM-III-R symptoms for the disruptive behavior disorders. *Journal of the American Academy of Child and Adolescent Psychiatry* 1992;31(2):210–218. [PubMed: 1564021]
- Pelham, WE.; Waschbusch, DA. Behavioral intervention in attention-deficit/hyperactivity disorder. In: Quay, HC.; Hogan, AE., editors. *Handbook of Disruptive Behavior Disorders*. New York: Kluwer Academic/Plenum; 1999. p. 255–278.
- Pelham WE Jr, Wheeler T, Chronis A. Empirically supported psychosocial treatments for attention deficit hyperactivity disorder. *Journal of Clinical Child Psychology* 1998;27(2):190–205. [PubMed: 9648036]
- Pennington BF. Toward a new neuropsychological model of Attention-Deficit/Hyperactivity Disorder: Subtypes and multiple deficits. *Biological Psychiatry* 2005;57:1221–1223. [PubMed: 15949991]
- Pennington BF, Ozonoff S. Executive functions and developmental psychopathology. *Journal of Child Psychology and Psychiatry* 1996;37(1):51–87. [PubMed: 8655658]
- Quay HC. Theories of ADDH. *Journal of the American Academy of Child and Adolescent Psychiatry* 1988;27(2):262–263. [PubMed: 3360739]
- Sagvolden T, Johansen EB, Aase H, Russell VA. A dynamic developmental theory of attention-deficit/hyperactivity disorder (ADHD) predominantly hyperactive/impulsive and combined subtypes. *Behavioral and Brain Sciences* 2005;28(3):397–419. [PubMed: 16209748]discussion 419–368
- Sergeant JA. Modeling attention-deficit/hyperactivity disorder: a critical appraisal of the cognitive-energetic model. *Biological Psychiatry* 2005;57:1248–1255. [PubMed: 15949995]
- Sergeant, JA.; Oosterlaan, J.; van der Meere, J. Information processing and energetic factors in Attention-Deficit/Hyperactivity Disorder. In: Quay, HC.; Hogan, AE., editors. *Handbook of disruptive behavior disorders*. Vol. 1999. New York, NY: Kluwer Academic/Plenum Publishers; 1999. p. 75–104.

- Shaffer D, Fisher P, Lucas CP, Dulcan MK, Schwab-Stone ME. NIMH Diagnostic Interview Schedule for Children Version IV (NIMH DISC-IV): description, differences from previous versions, and reliability of some common diagnoses. *Journal of the American Academy Child and Adolescent Psychiatry* 2000;39(1):28–38.
- Solanto MV, Wender EH, Bartell SS. Effects of methylphenidate and behavioral contingencies on sustained attention in attention-deficit hyperactivity disorder: a test of the reward dysfunction hypothesis. *Journal of Child and Adolescent Psychopharmacology* 1997;7(2):123–136. [PubMed: 9334897]
- Sonuga-Barke EJ. Psychological heterogeneity in AD/HD--a dual pathway model of behaviour and cognition. *Behavioural Brain Research* 2002;130(1–2):29–36. [PubMed: 11864715]
- Tannock R, Ickowicz A, Schachar R. Differential effects of methylphenidate on working memory in ADHD children with and without comorbid anxiety. *Journal of the American Academy Child and Adolescent Psychiatry* 1995;34(7):886–896.
- Waschbusch DA, Craig R, Pelham WE Jr, King S. Self-handicapping prior to academic-oriented tasks in children with attention deficit/hyperactivity disorder (ADHD): medication effects and comparisons with controls. *Journal of Abnormal Child Psychology* 2007;35(2):275–286. [PubMed: 17195950]
- Willcutt EG, Doyle AE, Nigg JT, Faraone SV, Pennington BF. Validity of the Executive Function Theory of Attention-Deficit/Hyperactivity Disorder: a meta-analytic review. *Biological Psychiatry* 2005;57:1336–1346. [PubMed: 15950006]
- Woodcock, RW.; McGrew, KS.; Mather, N. Woodcock-Johnson III Tests of Achievement. Riverside Publishing; Houghton Mifflin, Itasca Illinois: 2001.
- Zeiner P, Bryhn G, Bjercke C, Truyen K, Strand G. Response to methylphenidate in boys with attention-deficit hyperactivity disorder. *Acta Paediatrica* 1999;88(3):298–303. [PubMed: 10229041]

Figure 1a. *Figure 1b.* **Figure 1.**

a. Spatial Span Task screen layout. b. Spatial Span Task feedback screen.

Figure 2. Number of forward span trials correct (and standard errors) for all Incentive x Incentive Order conditions.

Figure 3. Number of backward span trials correct (and standard errors) for all Incentive x Incentive Order conditions.

Figure 4.
Number of trials correct (and standard errors) for all Incentive Order x Task Block conditions.

Table 1

Sample characteristics for each incentive order subgroup.

	Incentive Order	
	Incentive 1 st	No Incentive 1 st
Age, mean (SD)	8.8 (.95)	8.9 (.96)
Gender (male:female)	9:1	9:2
WISC Full Scale IQ, mean (SD)	115 (17)	116 (21)
WJ Test of Achievement, mean (SD)		
Letter-Word Identification	106 (17)	105 (15)
Calculation	107 (15)	113 (16)
Spelling	110 (15)	104 (15)
ADHD symptoms *		
Hyp/Imp, mean (SD)		
Parent report	6.0 (2.0)	5.7 (2.9)
Teacher report	5.2 (3.3)	4.6 (3.5)
Inattentive, mean (SD)		
Parent report	7.1 (1.5)	7.0 (1.8)
Teacher report	5.6 (3.1)	6.1 (2.7)
Comorbid diagnoses (number subjects)		
ODD:CD:Anxiety	4:2:5	8:0:3

* Note. These values represent the number of DSM-IV ADHD symptoms endorsed (i.e., rated as 'pretty much' or 'very much') on the DBD rating scale.