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Working Memory Capacity and the Antisaccade Task: Individual Differences in Voluntary Saccade Control

Nash Unsworth
Georgia Institute of Technology

Josef C. Schrock
Maryville College

Randall W. Engle
Georgia Institute of Technology

Performance on antisaccade trials requires the inhibition of a prepotent response (i.e., *don't look at the flashing cue*) and the generation and execution of a correct saccade in the opposite direction. The authors attempted to further specify the role of working memory (WM) span differences in the antisaccade task. They tested high- and low-span individuals on variants of prosaccade and antisaccade trials in which an eye movement is the sole requirement. In 3 experiments, they demonstrated the importance of WM span differences in both suppression of a reflexive saccade and generation of a volitional eye movement. The results support the contention that individual differences in WM span are not exclusively due to differences in inhibition but also reflect differences in directing the focus of attention.

As Baddeley and Hitch (1994) pointed out in their review on the progress of working memory (WM), the concept of a central executive is certainly the least well understood component of the WM system. However, individual-differences research in WM span has proven to be a fruitful means for studying the nature of the central executive. Research has demonstrated that WM span is related to real-world cognitive tasks such as reading comprehension (Daneman & Carpenter, 1980), following directions (Engle, Carullo, & Collins, 1991), and reasoning ability (Conway, Cowan, Bunting, Therriault, & Minkoff, 2002; Engle, Tuholski, Laughlin, & Conway, 1999; Kyllonen & Christal, 1990; Oberauer, Süβ, Wilhelm, & Wittmann, 2003). In addition, the concept of working memory capacity (WMC) has become an explanatory mechanism in a diverse array of health-related research areas, including work on alcoholism (Finn, 2002), identifying people who are susceptible to early onset Alzheimer's (Rosen, Bergeson, Putnam, Harwell, & Sunderland, 2002), and life-event stress (Klein & Boals, 2001).

Working Memory Capacity and Executive Attention

We and our colleagues (Engle, 2000; Engle, Kane, & Tuholski, 1999) view WM as a system consisting of a subset of highly activated long-term memory units (see Cowan 1988, 1995), a wide array of processes that achieve and maintain activation of those units, and an executive attention component. The executive attention component (or central executive) of WM is an attentional mechanism used to maintain current task goals, process incoming information, and block external (i.e., environmental distractors) and internal (i.e., other unrelated long-term memory units) interference (Engle, 2000). This idea is similar to that of controlled processing (Posner & Snyder, 1975; Schneider & Shiffrin, 1977) but is probably better conceptualized as a somewhat narrower concept similar to the supervisory attentional system proposed by Norman and Shallice (1986) and the general concept of cognitive control as depicted in computational models of the prefrontal cortex and anterior cingulate regions of the brain (e.g., Botvinick, Braver, Barch, Carter, & Cohen, 2001). Thus, when we refer to WMC, we mean the executive attention component of the broader WM system that is necessary when active maintenance is needed, especially in conditions of interference (Engle, 2000; Engle, Kane, et al., 1999).

Note that the executive attention construct is not needed for all cognitive processing but, rather, comes into play in situations requiring inhibition of prepotent responses, error monitoring and correction, and decision making and planning (Posner & DiGirolamo, 1998; Shallice & Burgess, 1993). This view is compatible with the notion that the control of attention is necessary for correlated mental actions such as inhibition, updating, and time sharing (e.g., Miyake et al., 2000; Salthouse, Atkinson, & Berish, 2003). At the present time, there is not enough evidence to suggest exactly how the central executive can be fractionated. In addition, it is important to note that the limitation in WMC is not necessarily about the number of "chunks" that can be held in memory at any one time (Cowan, 2001; Miller, 1956) but, rather, the limitation is about the extent to which individuals can control the focus of attention in a myriad of different situations. In fact, as we will see

Nash Unsworth and Randall W. Engle, School of Psychology, Georgia Institute of Technology; Josef C. Schrock, Division of Behavioral Sciences, Psychology, Maryville College.

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Correspondence concerning this article should be addressed to Nash Unsworth, School of Psychology, Georgia Institute of Technology, Atlanta, GA 30332–0170, or to Randall W. Engle, School of Psychology, Georgia Institute of Technology, Atlanta, GA 30332–0170. E-mail: gtg039d@prism.gatech.edu or randall.engle@psych.gatech.edu

shortly, models that suggest that WMC limitation is about the number of chunks that can be held in memory would be hard-pressed to predict our results.

We have argued that, like all other cognitive tasks, WM span measures such as reading span and operation span reflect multiple processes and constructs (Engle, Tuholski, et al., 1999). Individuals differ in verbal–phonological skills and knowledge, and this is reflected in all WM span tasks making use of verbal information. Individuals differ in visuospatial skills and knowledge, and this is reflected in all WM span tasks making use of visuospatial information. However, the primary construct responsible for the correlation between complex span measures of WM and tasks of higher order cognition is a domain-general capability to control attention, and this ability is particularly important in situations involving proactive interference or conflict between competing response tendencies. Thus, the variance that is common to all WM span measures such as operation, reading, and counting spans, which all load on the same factor, is a domain-free executive attention ability that is important for predicting performance on higher order cognitive tasks (e.g., Engle, Tuholski, et al., 1999; Kane et al., 2004).

Indeed, Baddeley (1992) made a similar statement, noting: "the central executive clearly reflects a system concerned with the attentional control of behavior, with subsequent developments almost certainly depending on parallel developments in the study of attention and the control of action" (p. 559). We have taken this suggestion and shown in several studies that high- and low-WM span individuals differ in their performance on tasks that require controlled attention with minimal memory requirements but with similar performance on tasks that require little controlled attention. Thus, as we hope to make evident, individual differences in WM span not only correspond to higher level cognitive abilities but also to the lower level capability to control attention.

Conway, Cowan, and Bunting (2001) demonstrated the importance of WMC in controlling selective attention using the classic dichotic listening procedure. In this study, high- and low-span participants performed a dichotic listening task that demonstrated the cocktail party phenomenon (Moray, 1959). Conway et al. (2001) reasoned that if WM span differences are related to attentional control, then low-span individuals should be more susceptible to distracting information than are high-span individuals. It is striking that Conway et al. (2001) found that 65% of low-span participants heard their names in the ignored message, whereas 20% of high-span participants heard their names. Thus, the results suggest that individuals ranked low on measures of WM span are more prone to orienting to a powerful attention-capturing cue than are individuals ranked high on WM span measures. Conway et al. (2001) interpreted these results on the basis of the controlled-attention view of WM, suggesting that high-span participants were more effective at blocking the distracting information than were low-span participants because of the use of controlled attention in maintaining task-relevant information and blocking interfering stimuli.

Kane and Engle (2003) recently demonstrated the importance of WM span differences in another classic selective attention–interference task in a series of studies using the Stroop task. Kane and Engle found that high- and low-span participants differed in their ability to perform the Stroop task if 75% of the trials were congruent as opposed to either 0% or 50% congruent trials. The argument was that in conditions with a high proportion of incongruent trials, the trials themselves served to cue the maintenance of the task set. However, under conditions of frequent congruent trials, there was not the frequent cue to maintain the productions necessary to perform incongruent trials, and low-span individuals were less likely to do the mental work necessary to consistently maintain the task-relevant goals. That is, low-span individuals would show more of what Duncan and others (De Jong, Berendsen, & Cools, 1999; Duncan, 1995) have termed *goal neglect*, or the disregard of task requirements even when the task goal is understood. If the word matched the color on most trials and then suddenly changed so that word and color did not match, low-span participants tended to make more errors than did high-span participants. Thus, the ability to maintain the task goal in mind (i.e., say the color and not the word) decreased as the number of congruent trials increased, resulting in more errors for both groups. However, because of the high-span participants' ability to maintain the task goal (and thus lesser susceptibility to goal neglect), they were less affected than were low-span participants. The authors suggested that the difference between high- and low-span participants was due to the greater use of executive attention by the high-span participants in order to maintain the task goal in the face of conflict.

In our view, individual differences in WM span, and hence executive attention, should be most apparent in situations in which active maintenance is needed, particularly in the face of potent environmental distractors or strong internal interference. That is, interference-rich situations in which representations either irrelevant to or contradictory to the current task capture attention make it more likely that the current task goals and productions will decay from the active state and thus be less likely to control thought and behavior.

Working Memory Span Differences in the Antisaccade Task

One task particularly amenable for studying this situation is the antisaccade task (Hallet, 1978; Hallet & Adams, 1980; see Everling & Fischer, 1998, for a review), in which participants are required to make a saccade either toward (prosaccade) or away (antisaccade) from a flashing cue. Previous work with this task has demonstrated differences in groups thought to differ in WMC such as older and younger adults (Butler, Zacks, & Henderson, 1999; De Jong, 2001), schizophrenics and healthy controls (Fukushima et al., 1988), and patients with lesions in the prefrontal cortex and healthy controls (see Everling & Fischer, 1998, for a review). Kane and colleagues (Kane, Bleckley, Conway, & Engle, 2001) further tested the claim that WM span differences are related to low-level executive attentional abilities using a modified version of this task. In the modified version, participants are required to make either a prosaccade or an antisaccade and identify one of three letters (*B*, *P*, or *R*) that were briefly presented in the correct screen location. Prosaccade trials simply require looking toward the flashing cue, and this response is thought to rely on exogenous, automatic attentional capture and should not require the recruitment of executive control. Antisaccade trials, however, require not only the inhibition of a prepotent response (i.e., *don't look at the flashing box*) but also require the planning and execution of a voluntary saccade in the opposite direction. Antisaccades are essentially voluntary saccades generated via top-down control and thus re-

quire a degree of attention control not apparent in the relatively automatic prosaccades. Therefore, antisaccades but not prosaccades should require executive control, and thus individual differences in WM span should be apparent only in antisaccade trials.

Kane et al. (2001) reasoned that WM span differences would be important in the antisaccade task because it relies on the same domain-general executive attention component needed in both dichotic listening and the Stroop task. To be specific, Kane et al. argued that executive attention would be crucial in the antisaccade task because of the need to actively maintain task goals in the face of a powerful attention-capturing cue leading to a habitual response contradictory to the one required in the task. Roberts and Pennington (1996) advanced a similar view, noting that "the Antisaccade and Stroop tasks have strong prepotent responses but relatively light working memory demands: Thus, even momentary lapses or slight deficiencies in working memory will affect the balance in favor of prepotency" (p. 112). Kane et al. hypothesized that low-span individuals would be worse at maintaining the production in active memory (*if blink left–look right*) than high-span individuals, and thus any lapse in attention (and intention) would result in prepotency guiding behavior and hence the occurrence of a fast error. That is, low-span individuals should be more susceptible to goal neglect than are high-span individuals, resulting in more errors of looking in the wrong direction. Further, these errors should be very fast, because they are functionally prosaccades. In addition, even if the task goal were actively maintained, low-span individuals should be slower at resolving the conflict between the task goal and habit: Low-span individuals would be slower to move the focus of attention to the correct location than high-span individuals, even if the task goal were actively maintained.

This is precisely what Kane et al. (2001) found. High- and low-span participants did not differ on prosaccade trials but did differ on antisaccade trials. Even though both groups performed worse on the antisaccade task compared with the prosaccade task, low-span participants performed much worse than high-span participants on the antisaccade trials. To be specific, high-span participants were 174 ms faster on average to identify the letters than were low-span participants. Further, when eye movements were measured (Experiment 2), the two groups of participants did not differ in the frequency with which they shifted their eyes to the wrong side of the screen in the prosaccade condition. In the antisaccade condition, however, low-span participants were slower to saccade and made many more reflexive saccades to the exogenous cue on the wrong side of the screen.

Thus, participants selected on the basis of the number of words recalled in the operation-span task showed differential performance on a highly attention-demanding task that placed a minimal burden on memory. The authors suggested that the reason high-span participants performed better than low-span participants in the antisaccade condition was that they were better able to maintain the task goal in WM to, in a sense, override the automatic response to look toward the cue. Thus, attention and memory processes seem to work in concert in order to generate the correct response. These results indicate that individual differences in WM span correspond to individual differences in executive attention. In addition, in our view, these results demonstrate that individual differences in WM span are important in a variety of tasks that require the maintenance of information in a highly active and easily accessible state.

However, there is one feature of the Kane et al. (2001) study that could limit their conclusion that span differences in antisaccade performance are due to differences in attentional control. It is possible that the differences found between high- and low-WM span participants in the Kane et al study resulted from a differential load resulting from the letter task. It has been previously demonstrated that a secondary attention-demanding task increases the reflexive errors in the antisaccade task (Roberts, Hager, & Heron, 1994; Stuyven, Van der Goten, Vandierendonck, Claeys, & Crevits, 2000). Thus, one possibility is that the letter-identification task was more demanding for the low-WM span participants, which led to a greater attentional load and therefore a greater decrement in the antisaccade task. In other words, even the eye-movement results may have resulted from a differentially difficult secondary task for the low-WM span participants. If that were the case, then it would be difficult to make the argument that differences in the antisaccade task were the direct result of individual differences in capability for attention control.

In the present study, we attempted to alleviate the shortcoming of the Kane et al. (2001) study by using eye movements as the only required response. Participants fixated in the center of the screen, and two boxes were displayed 11° to each side of fixation. At some point, one of the boxes flickered. In the prosaccade condition, the participants were simply to shift their attention and their gaze to the box that had flickered. They had 600 ms to make that eye movement. In the antisaccade condition, participants were to shift their attention and gaze to the box on the side of the screen opposite the one that flickered. Again, they had 600 ms to respond and have their eye position recorded in that region, and any saccade in the direction of the flashing cue was treated as an error. This task was not confounded by possible differential skill in identifying or discriminating letters as in the Kane et al. study or by any other concurrent task. All that participants had to remember to do was to look toward the flickering box in one condition and to look away from the flickering box in the other condition. If there were differences in performance of high- and low-WM span participants in the antisaccade condition, they were not due to a secondary task.

Furthermore, this study extends the findings of Kane et al. (2001) by demonstrating that span differences can arise even in prosaccade trials under conditions of increased interference or endogenous cuing and thus are more reliant on the executive attention system. In Experiment 1, we presented prosaccade and antisaccade trials in blocks. In Experiment 2, however, we randomly presented both prosaccade and antisaccade trials within the same block, a manipulation designed to increase the demands for cognitive control in the task. In Experiment 3, we had participants perform four separate saccade tasks in order to better determine the roles of endogneous control and suppression in the antisaccade paradigm.

Experiment 1

In Experiment 1, we examined whether individual differences in WM span would be important in the antisaccade task in the absence of a secondary task component. In addition, on the basis of the previous work demonstrating the relationship between per-

formance on the antisaccade task and executive functioning, we made two predictions about the relationship between WM span and the antisaccade task. First, low-WM span participants should be less able to maintain the task goal in active memory, because their attention is more easily captured by distraction. Thus, they should make more errors in the form of reflexive saccades to the exogenous cue, and these errors should be relatively rapid because the participant, at the moment the error occurs, is functionally in the prosaccade condition. Second, even when low-WM span participants are successful in maintaining the task goal in active memory, they will have difficulty implementing the control required to resolve the conflict between the competing response tendencies to, on one hand, make the prepotent response to the movement-affording exogenous cue and, on the other hand, do what the experimenter has asked them to do. This difficulty in control should be reflected in longer latencies on correct trials for low-span participants than for high-span participants.

In contrast, we did not predict span differences in the prosaccade version of the task. In the prosaccade task, the orienting response coincides with the task goal of looking toward a flashing cue on the screen. Because the prosaccade version of the task makes minimal demands on controlled attention, we did not anticipate performance differences between high- and low-span participants.

Method

Participant Screening for Working Memory Span

We prescreened participants using the operation-span task (OSPAN; Turner & Engle, 1989). The OSPAN has demonstrated good reliability and validity (Conway et al., 2002; Engle, Tuholski, et al., 1999; Klein & Fiss, 1999). To be specific, the OSPAN has a test–rest reliability of .88 (Klein & Fiss, 1999) and Cronbach alpha estimates ranging from .61 to .83 (Conway et al., 2002; Engle, Tuholski, et al., 1999; Klein & Fiss, 1999). Furthermore, researchers have demonstrated the validity of OSPAN in numerous contexts by showing that OSPAN correlates well with other measures of WM span, loads highly on a latent variable with other WM span tasks, and predicts performance on a large number of higher order cognitive tasks (Conway et al., 2002; Engle, Tuholski, et al., 1999).

The OSPAN requires participants to solve a series of math operations while trying to remember a set of unrelated words. For example, participants may see: *Is (9/3) − 1 = 1? Dog*. The participant is required to read the operation aloud without pausing and then to verify aloud whether the operation is correct ("yes" vs. "no"). After verification, participants are required to read the word aloud once again without pausing. Once the participant has read the word aloud, the experimenter presses a key to move onto the next operation-word string. The same procedure is repeated until three question marks (*???*) appear, indicating to the participant that it is time to recall the words from that set in the correct order. The operation-word strings can vary from two to five items in length. The OSPAN score is the sum of recalled words for all of the sets in which the entire set is recalled in the correct order. In addition, in order to ensure that participants are not trading off between solving the operations and remembering the words, an 85% accuracy criterion on the math operations is required for all participants.

Participants and Design

Participants were 30 high-span participants and 30 low-span participants, as determined by the OSPAN. Those participants scoring in the upper quartile were deemed high-span participants, whereas those scoring in the lower quartile were considered low-span participants. Participants were recruited from a participant pool at Georgia Institute of Technology and from the Atlanta community through newspaper advertisements. Participants were between the ages of 18 and 35 years and received either course credit or $20 compensation for their participation. Each participant was tested individually in a laboratory session lasting approximately 1 hr. The design was a 2 (WM span: high vs. low) × 2 (saccade type: antisaccade vs. prosaccade) mixed factorial design with saccade type as the within-subjects variable. Four blocks of 75 trials each, two prosaccade and two antisaccade, were counterbalanced in an ABAB–BABA design.

Apparatus and Materials

We recorded eye movements using an Applied Science Laboratories E-5000 eye-tracker (Bedford, Massachusetts). This is an infrared-based eye-tracking system with a sampling frequency of 60 Hz, allowing eye movements to be recorded every 16.7 ms. The eye-tracker has a spatial resolution error of less than 1° of visual angle. We used a magnetic head tracking system (Applied Science Laboratories, Bedford, Massachusetts) in order to coordinate head movements and camera focus on the eye. Only the movements of the left eye were recorded. We calculated eye position, fixation, and fixation duration using the EYENAL software (Eyenal Data Analysis Program, Version 1.0, 2000) provided by Applied Science Laboratories. Stimuli were presented on a 19-in. (48.26-cm) monitor using E-Prime experimental software (Schneider, Eschman, & Zuccolotto, 2002).

Procedure

Participants were seated in a comfortable chair and given an overview of the equipment used for eye tracking. After the lights were dimmed, the eye-tracking apparatus was calibrated for luminance and spatial accuracy. Participants were instructed that their response to each trial in the experiment would be an eye movement, and that they should do their best to respond as quickly and as accurately as possible. Each block of experimental trials was preceded by a practice session of 10 trials of the same type as the upcoming experimental block. Calibration was checked again following the first two blocks of the experiment.

For each trial, participants saw a black screen containing the word *ready* in the center of the screen and 1-cm (0.6° visual angle) white squares positioned at 11.5° of visual angle to the left and right of center. At the start of each trial, the word *ready* was presented in the center of the screen for 1,500 ms to warn the participant that a trial was about to begin. A fixation point (a white plus sign 1 cm × 1 cm) then appeared for a period that varied unpredictably between 600 and 2,200 ms in 100-ms increments. Eye position was monitored to ensure that the participant was looking at the fixation point at the end of the wait period. If the participant was not looking at the fixation point when the wait period ended, the fixation point remained on the screen, and eye position was checked every 50 ms until the participant was focused on the center position. Following the wait period, one of the squares flashed for 600 ms while the fixation point and the other square remained on the screen.

In the antisaccade task, participants were required to make their first eye movement toward the square opposite the flashing square. In the prosaccade task, participants made a saccade toward the flashing square. After the target had flashed for 600 ms, the targets and center fixation point were removed, and the words *left* or *right* appeared at the correct target location. The word remained on the screen for 1,500 ms. Within each 75-trial block, the target position was random and equally likely to occur to the left or right of center. Participants completed 4 sets of 75 trials: two prosaccade sets and two antisaccade sets, so that each participant completed 150 prosaccade and 150 antisaccade trials during the experiment. Trial type alternated between sets, with the order of presentation counterbalanced. In the first block of prosaccade and antisaccade trials, participants were told whether their first eye movement was in the correct direction, with a correct eye movement defined as the initial eye movement from center

going toward the correct target location, and an incorrect saccade as an initial eye movement away from the target location.

Data Preparation

Measures of eye position and pupil diameter were recorded at 60 Hz, or one sample every 16.67 ms. These raw data were reduced into larger fixation units. We defined a fixation as a point of gaze remaining within a 1° × 1° boundary for at least three recording cycles (50 ms). Following classification of the data into fixations, we mapped the location of the fixations onto screen locations. The display screen was divided into three areas of interest. A central fixation area of a 2.6° horizontal visual angle and a 5° vertical visual angle was centered on the screen so that the initial fixation point was in the middle of this area. The left and right areas of interest extended from the edges of the central fixation area to the left or right edges of the screen. To compensate for any calibration differences among participants, we adjusted the fixation area of interest for each participant so that the majority of the center fixations fell within the middle of the central area of interest. To be specific, the areas of interest remained the same but were simply shifted up, down, right, or left a degree or two to compensate for any small calibration differences between participants, such that the central fixation point (as defined by the participant's fixations when on center) was in the center of the central area of interest. This was done for each set of participant data as a whole. Therefore, everyone had the same area to traverse to get from the center fixation point to a fixation point in either the left or right areas of interest.

For each trial, we examined the first saccade after the onset of the flashing cue. If the initial saccade was toward the appropriate target, the trial was classified as correct; if the saccade was in a direction opposite the appropriate location, the trial was classified as a direction error. Trials were discarded if the initial saccade fell outside the areas of interest, the data recording was interrupted because of lost pupil or corneal reflectance, or fixation at the moment of target onset was not within the central area of interest. These criteria eliminated 10.4% of the trials. Trials were also eliminated if the latency to respond was above 1,000 ms or below 100 ms, accounting for an additional 4% of the trials.[1]

Results

Participants

Data for 5 high- and 6 low-span participants were excluded from data analyses because of eye-tracking data-collection problems. The mean OSPAN scores for the final 25 high- and 24 low-span participants were 27.9 ($SD = 7.7$, range 19–50) and 6.1 ($SD = 2.3$, range 2–9), respectively. The mean ages for high- and low-span participants were 24.68 ($SD = 6.03$) and 25.04 ($SD = 5.16$) years, $t(47) = 0.225$, $p > .36$.

Correct Trial Latency

Our measure of latency is the elapsed time between the onset of the flashing target and the beginning of the fixation in the area of interest. Only latencies for saccades made in the correct direction were included in the analysis. The results suggest that participants demonstrated longer latencies on antisaccade than on prosaccade trials. In addition, as seen in Figure 1, in terms of WM span differences, the results suggest that high- and low-span participants do not differ on prosaccade trials but that low-span participants demonstrated significantly longer latencies for saccades in the correct direction on antisaccade trials.

These observations were supported by a 2 (saccade type) × 2 (span) mixed analysis of variance (ANOVA), with saccade type as the within-subjects variable. The ANOVA yielded main effects of both saccade type, $F(1, 47) = 32.30$, $p < .01$; and span, $F(1, 47) = 4.14$, $p < .05$. However, both of these effects were qualified by a significant two-way interaction, $F(1, 47) = 6.49$, $p < .05$. This interaction suggests that high- and low-span individuals do not differ in latency on the relatively automatic prosaccade trials, $F(1, 47) < 1$, but that low-span participants had significantly longer latencies on antisaccade trials than did high-span participants, $F(1, 47) = 6.20$, $p < .05$. Thus, although both span groups' performance was hurt on antisaccade trials relative to prosaccade trials, low-span participants' performance was hurt much more.

Direction Errors

Direction errors represent those trials in which the first saccade outside the fixation area of interest after the cue was in the direction opposite the target. For the analyses, the dependent measure is the percentage of direction errors. The results suggest that participants made more errors on antisaccade trials than on prosaccade trials and that low-span participants made more errors overall than did high-span participants. In addition, recall that in prosaccade trials, the correct response was simply an eye movement toward a flashing cue, so we expected high- and low-span participants to have relatively few errors for these trials. Indeed, as seen in Figure 2, both high- and low-span participants were correct more than 95% of the time on the prosaccade trials, and no WM span differences in error rates were found on the prosaccade trials. Although low-span participants made more errors overall, the span groups did not differ in the percentage of errors on the relatively automatic prosaccade trials. In contrast, a correct response in the antisaccade trials was a saccade in the direction opposite that of the flashing cue. Here, low-span participants tended to make many more errors than did high-span participants.

These conclusions were supported by a 2 (saccade type) × 2 (span) mixed ANOVA, with saccade type as the within-subjects variable. The analysis suggested that the percentage of direction errors was higher for antisaccades than for prosaccades, $F(1. 47) = 45.98$, $p < .01$, and that it was higher for low-span participants than for high-span participants, $F(1, 47) = 6.43$, $p < .05$. In addition, these two factors interacted with each other, $F(1, 47) = 6.62$, $p < .05$, indicating that high- and low-span participants do not differ in the percentage of direction errors on prosaccades, but that low-span participants were much more likely to make an initial eye movement toward the flashing cue, $F(1, 47) = 8.39$, $p < .01$.

Error Latency

Recall that, in our view, errors reflect those trials in which the task goal has been temporarily lost from active memory and behavior is guided in favor of prepotency. The response is, functionally, a prosaccade response. In these situations, one would expect such errors to occur relatively rapidly. To test this prediction, we examined error latencies for those trials in which the participant's first saccade and fixation were in the incorrect direc-

[1] Note that in all three experiments, there were no differences between high- and low-WM span individuals in the percentage of trials eliminated because of these criteria (i.e., all ps $> .24$).

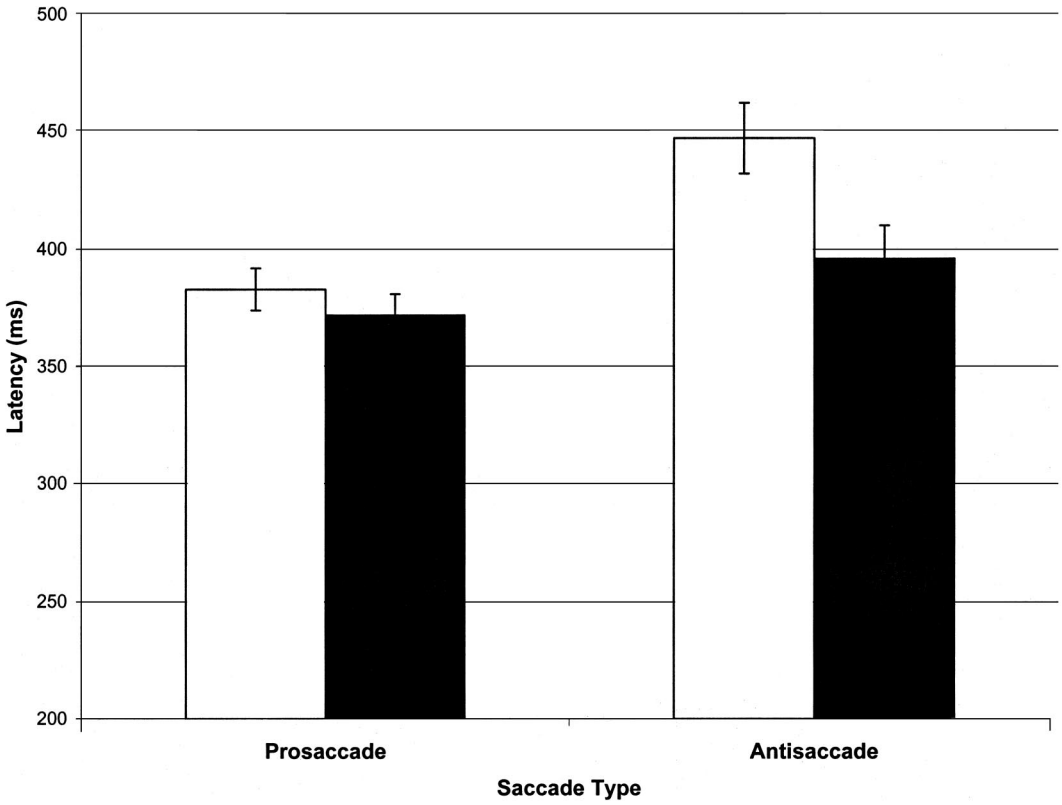

Figure 1. Mean latency for correct trials as a function of working memory span and saccade type for Experiment 1. Open bars indicate low-span participants; solid bars indicate high-span participants. Error bars represent one standard error of the mean.

tion. Thus, error latency reflects the time between the onset of the flashing cue and the beginning of fixation in the area of interest opposite the target. As shown in Table 1, the results suggest that latencies on incorrect trials were shorter than those on correct trials, and there were no span differences in latencies for incorrect trials. Span differences in latency occurred only for correct trials.

A 2 (accuracy: correct vs. incorrect) $\times$ 2 (span) mixed ANOVA, with accuracy as the within-subjects variable, confirmed these impressions. The ANOVA yielded a main effect of accuracy, $F(1, 47) = 269.98$, $p < .01$, suggesting that latencies on incorrect trials were shorter ($M = 265$ ms, $SE = 9$) than they were on correct trials ($M = 399$ ms, $SE = 8$). In addition, there was a significant Accuracy $\times$ Span interaction, $F(1, 47) = 4.73$, $p < .05$. The interaction suggests, similar to the correct trial latency analysis, that significant span differences emerge for correct trials, $F(1, 47) = 4.14$, $p < .05$, with low-span participants responding more slowly than high-span participants. For incorrect trials, however, no span differences emerge, $F(1, 47) < 1$.

Discussion

The idea that prosaccades are prepotent, largely automatic responses to exogenous stimuli was supported by the results from Experiment 1. Both groups were faster and more accurate on prosaccade trials than they were on antisaccade trials. In addition, no group differences in prosaccade performance were found on the basis of measures of WM span. On the other hand, WM span measures such as OSPAN appear to tap at least some of the same processes required for performing the more attention-demanding antisaccade task. High-WM span participants performed the antisaccade task faster and were better able to resist being drawn to the attention-capturing cue than were low-span participants. These findings nicely replicated the basic findings from Kane et al. (2001), demonstrating that span differences are observed in the antisaccade task but not in the prosaccade task. It seems, therefore, that the span differences observed in the Kane et al. study were not solely a result of the secondary letter-identification task they used.

Furthermore, the results support the prediction that WM span differences will appear in conditions in which top-down control of attention is required, particularly when task goals must be actively maintained in the face of interference. High- and low-span participants only differed on antisaccade trials on which the task goal is to move attention and eyes to a location opposite the flashing box. If for any reason the task goal is temporarily lost, the cue will capture attention and control behavior, and a fast error will be made. Referring back to Figure 2, one can see that low-span participants make many more reflexive saccades in antisaccade trials than do high-span participants. In addition, when either group makes an error, the error tends to occur relatively rapidly (e.g., roughly 134 ms faster than in correct trials). If the span differences observed on antisaccade trials are reflective of defi-

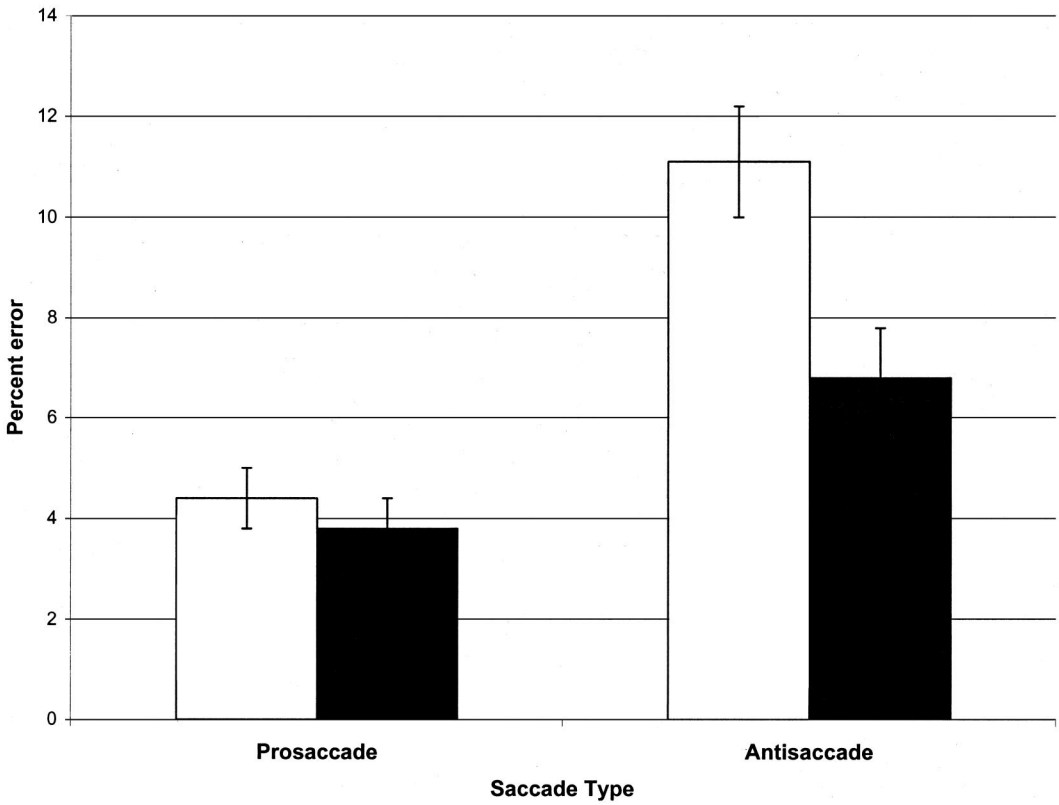

Figure 2. Mean percentage of direction errors as a function of working memory span and saccade type for Experiment 1. Open bars indicate low-span participants; solid bars indicate high-span participants. Error bars represent one standard error of the mean.

ciencies in attentional control, then we should be able to demonstrate similar differences on prosaccade trials by increasing the need for such control. This was precisely our aim in Experiment 2.

Experiment 2

In Experiment 2, high- and low-span participants were given a mixed-trial version of the prosaccade and antisaccade tasks. In Experiment 1, participants completed 75 trial blocks of either prosaccade or antisaccade trials; thus, each individual trial in a block of trials was of the same type (either prosaccade or antisaccade). In Experiment 2, prosaccade and antisaccade trials were presented randomly in a mixture in the same block. We predicted that the mixed-trial design would increase the demands to control attention on both the antisaccade and prosaccade trials for two reasons. First, the previous trial type no longer predicted the upcoming trial, and this should place a premium on active maintenance of the task goal for each trial. Second, because in many instances prosaccade trials followed antisaccade trials or vice versa, active suppression of the goal in the previous trial should play an important role, unlike in Experiment 1, in which the task goal for each trial reinforced the task goal for the next trial. Because the increased attentional demands should apply for both prosaccade and antisaccade trials, we in effect created a prosaccade task that required attentional control and active maintenance of task goals, and for this reason, we expected low-span participants to show performance differences for both prosaccade and antisaccade trials in the mixed-trial design.

Table 1
Mean Latency (in ms) for Correct and Incorrect Trials by Span

	Latency			
	Correct		Incorrect	
Working memory span	*M*	*SE*	*M*	*SE*
High	384	11	267	12
Low	415	11	262	12

Method

Participants and Design

We selected 25 new high- and low-span participants, as determined by the OSPAN, from the same population as in Experiment 1 to participate. The design was a 2 (WM span: high vs. low) × 2 (saccade type: antisaccade vs. prosaccade) mixed factorial design with saccade type as the within-subjects variable.

Procedure

The stimulus display and eye-tracking equipment were the same as those used in Experiment 1. We used a modified version of the prosaccade and

antisaccade tasks from Experiment 1. As before, each trial began with the word *ready* displayed for 1,000 ms, followed by two 1-cm square targets flanking a central fixation point. Rather than using a plus sign as the fixation point, we displayed either a white diamond or a white circle for 1,000 ms. Both symbols subtended 0.6° of visual angle and provided instruction for the type of eye movement to be performed on that trial. For example, the white circle might indicate a prosaccade trial and the diamond an antisaccade trial. At the end of 1,000 ms, the symbol disappeared, and 200 ms later, one of the two displayed boxes on the screen began flashing. Participants were required to make a saccade toward the flashing box if the symbol indicated a prosaccade trial and a saccade toward the box opposite the flash if the symbol indicated an antisaccade trial. The box flashed for 600 ms, and then the word *correct* replaced the box at the appropriate target location for 1,000 ms, followed by feedback indicating whether the participant's initial eye movement was in the correct direction.

Following instructions, an initial calibration, and 20 trials of practice (10 practice trials with each symbol), participants completed 240 experimental trials. After every 80 trials, the symbol meaning was switched (e.g., a circle indicated prosaccade in the first 80 trials, antisaccade for the next 80 trials, and prosaccade for the last 80 trials), and the calibration was rechecked. Participants were given 16 practice trials using the new symbol mapping after each pause in the experiment. Within each 80-trial block, 40 prosaccade and 40 antisaccade trials were presented in random order. The same data-screening criteria were used as in Experiment 1. We eliminated 3.5% of the trials because either pupil reflectance was lost during the trial, or the first saccade was to a point outside the target areas. We removed 1.6% of the trials because the initial saccade was either slower than 1,000 ms or faster than 100 ms.

Results

Participants

Data for 5 high- and 4 low-span participants were excluded from analyses because of eye-tracking data-collection problems. In addition, one low-span participant was removed from the data set because of excessive (approximately 50%) errors, a likely indication that the participant had failed to understand the instructions. The mean OSPAN scores for the final 20 high- and 20 low-span participants were 26.5 ($SD = 7.8$, range 19–44) and 5.3 ($SD = 2.5$, range 2–9), respectively. The mean ages for high- and low-span participants were 22.25 ($SD = 4.45$) and 21.68 ($SD = 5.32$), $t(37) = -0.361$, $p > .72$.

Correct Trial Latency

The elapsed time between the onset of the flashing target and the start of a fixation in the left or right area of interest represents the latency measure. We included only latencies for saccades made in the correct direction in the analysis. As seen in Figure 3, the results suggest that, like in Experiment 1, antisaccade trials were performed more slowly than were prosaccade trials. In addition, low-span participants had slightly longer latencies than did high-span participants on both types of trials, and unlike in Experiment 1, the span difference in latency was not a function of saccade.

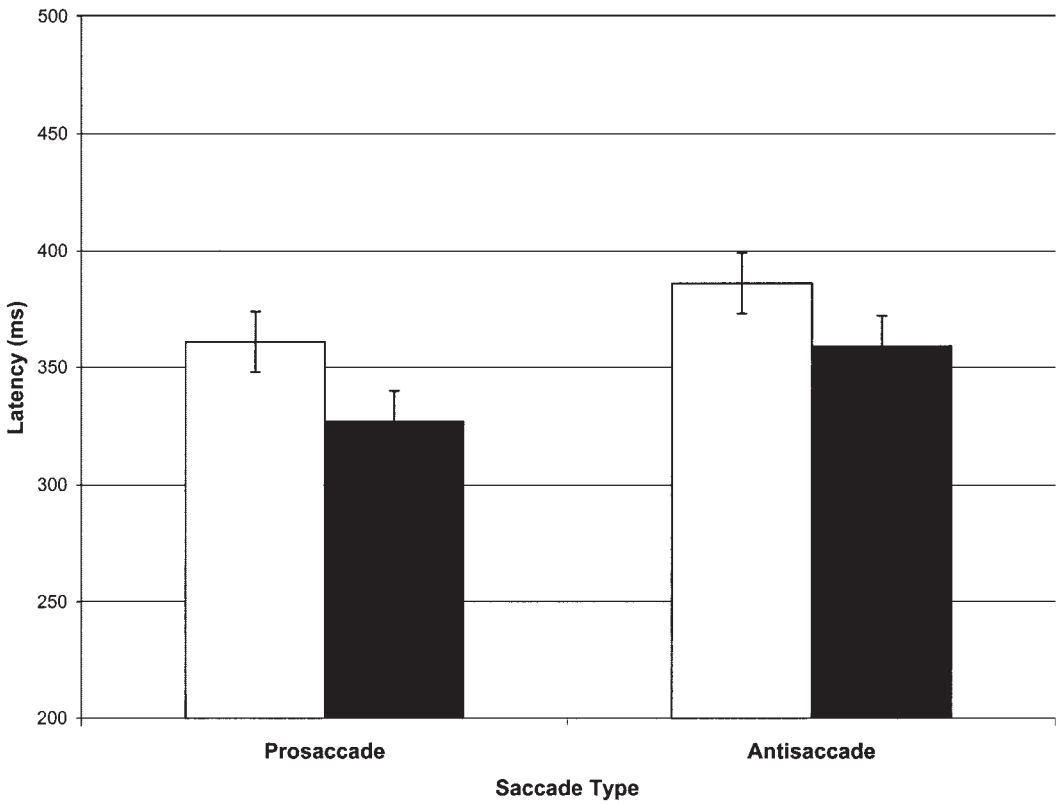

Figure 3. Mean latency for correct trials as a function of working memory span and saccade type for Experiment 2. Open bars indicate low-span participants; solid bars indicate high-span participants. Error bars represent one standard error of the mean.

That is, low-span participants had longer latencies on both prosaccade and antisaccade trials than did high-span participants.

These impressions were confirmed by a 2 (saccade type) × 2 (span) mixed ANOVA with saccade type as the within-subjects variable. The ANOVA revealed a main effect of saccade type, $F(1, 38) = 52.7$, $p < .01$, suggesting that antisaccade trials had longer latencies ($M = 372$ ms, $SE = 9$) than prosaccade trials ($M = 344$ ms, $SE = 9$). In addition, the ANOVA yielded a marginal main effect of span, $F(1, 38) = 2.95$, $p = .09$, suggesting that low-span participants were slower in responding ($M = 373$ ms, $SE = 12$) than were high-span participants ($M = 343$ ms, $SE = 12$). Furthermore, unlike that in Experiment 1, the Saccade Type × WM Span interaction did not approach significance, $F(1, 38) < 1$.

Direction Errors

As in Experiment 1, we counted initial saccades made to the area of interest opposite the correct target location as direction errors. As expected, low- and high-span participants no longer performed equally well on prosaccade trials. As can be seen in Figure 4, low-span participants were more likely than were high-span participants to make their initial saccade in the wrong direction for both antisaccade trials and prosaccade trials. In addition, unlike the results of Experiment 1, span differences in error rates did not seem to be a function of saccade type.

A 2 (saccade type) × 2 (span) mixed ANOVA with saccade type as the within-subjects variable supported these observations. To be specific, the ANOVA yielded main effects of both saccade type, $F(1, 38) = 7.87$, $p < .01$; and span, $F(1, 38) = 14.49$, $p < .01$, indicating that more errors were made in antisaccade trials than prosaccade trials and that low-span participants were more error-prone than high-span participants. However, the interaction between these two variables did not reach significance, $F(1, 38) = 1.82$, $p = .19$, suggesting that although low-span participants made more errors than high-span participants, this was not a function of saccade type.

Previous Trial Influence

Because we intermixed prosaccade and antisaccade trials within the same experimental block, we wondered whether the previous trial influenced responding on the current trial. That is, would switching to a new trial type influence performance and if so, would this be a function of WM span? Therefore, we analyzed trials that were either the same as the previous trial (a no-switch trial) or trials that were different from the previous trial (a switch trial). As shown in Figure 5, the results suggest that switching to a new trial type substantially increased error rates. However, this increase in error rates was not different for high- and low-span individuals.

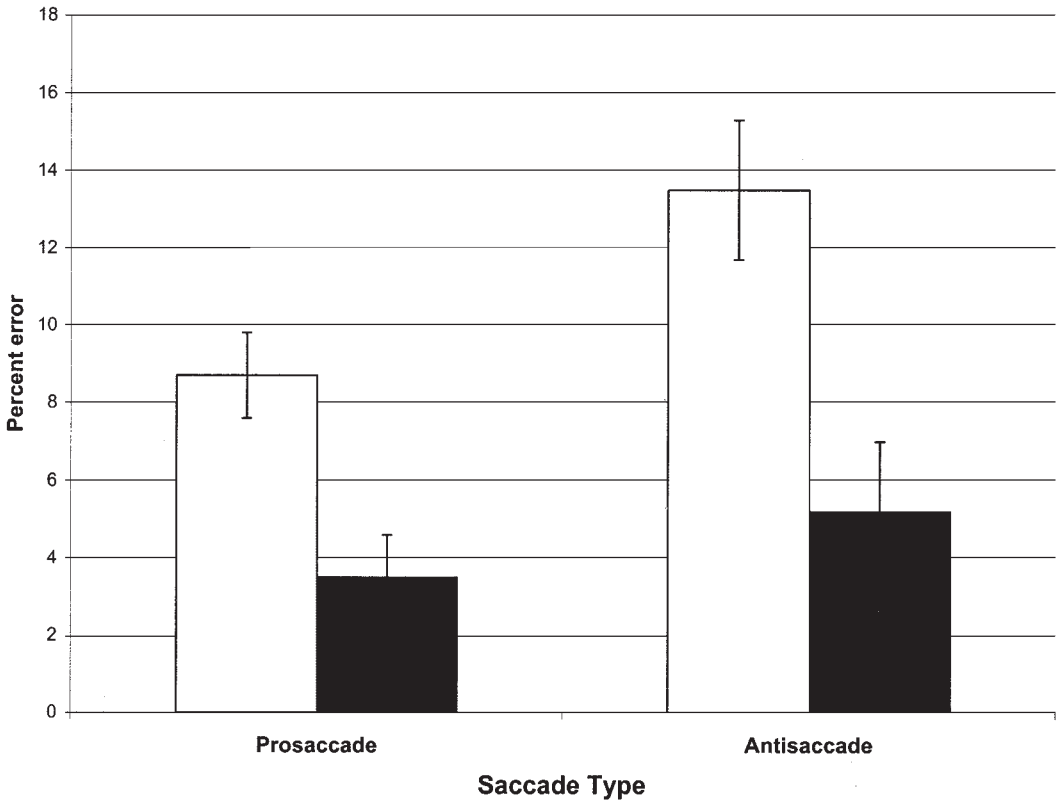

Figure 4. Mean percentage of direction errors as a function of working memory span and saccade type for Experiment 2. Open bars indicate low-span participants; solid bars indicate high-span participants. Error bars represent one standard error of the mean.

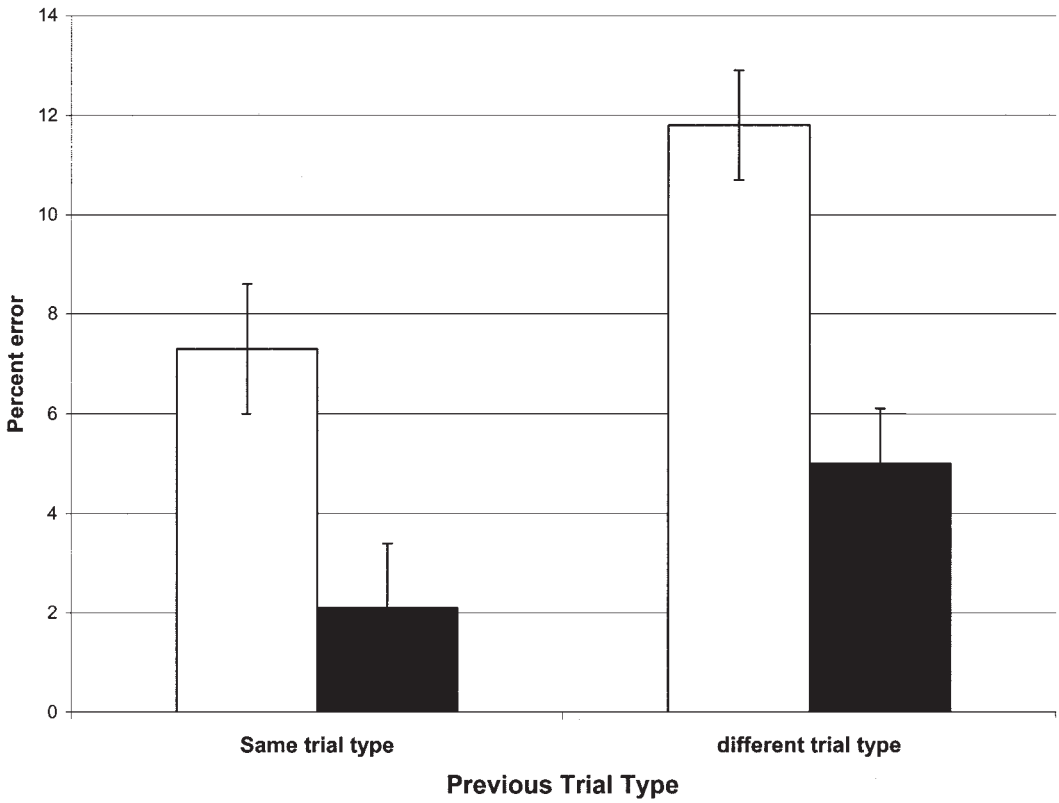

Figure 5. Mean percentage of direction errors as a function of working memory span and previous trial for Experiment 2. Open bars indicate low-span participants; solid bars indicate high-span participants. Error bars represent one standard error of the mean.

We conducted a 2 (previous trial type: same vs. different) × 2 (span) mixed ANOVA with previous trial type as the within-subjects variable on the percentage of direction errors to confirm these observations. The ANOVA demonstrated a main effect of previous trial type, $F(1, 38) = 28.73$, $p < .01$, suggesting that error rates were larger when switching to a new trial type ($M = 8.44$ %) than when performing the same trial type ($M = 4.73$ %). However, this effect did not interact with WM span, $F(1, 38) = 1.32$, $p = .26$.

Discussion

As expected, the results from Experiment 2 demonstrate that prosaccades are executed faster and more accurately than are antisaccades. However, unlike those in Experiment 1, low-span participants made more errors on both antisaccades and prosaccades than did high-span participants. In addition, low-span participants were marginally slower on both saccade tasks than were high-span participants. Thus, by intermixing prosaccade and antisaccade trials within the same block of trials, we found that span differences now emerged in prosaccade trials as well. We argue that this manipulation put a premium on active maintenance and attentional control, and that this increased need for control adversely impacted low-span participants more so than it did high-span participants.

It is important to note that the relationship between performance on the prosaccade task in Experiment 2 and WM span is unlikely due to our simply having increased the complexity of the task. In numerous studies that combine the prosaccade with concurrent WM intensive tasks (e.g., solving math equations, shadowing speech, or tapping fingers in a regular pattern), researchers have found that these concurrent tasks adversely impact antisaccade performance but not prosaccade performance. Furthermore, in those studies that do include WMC measures, researchers have not reported a relationship between WMC and the prosaccade task, even when there is a concurrent task (Kane et al., 2001; Roberts et al., 1994). Much the same has been found with studies focusing on other group differences in the prosaccade and antisaccade task, including age, prefrontal cortex damage or lesion and schizophrenia—the prosaccade task is, for the most part, immune to just those factors that predict antisaccade performance. So why does the prosaccade task in Experiment 2 lead to performance differences based on WM span?

We argue that the use of the mixed-trial design encouraged participants to rely more on control of attention during the prosaccade task for both maintenance and, possibly, suppression. Because the trials switched randomly from prosaccade to antisaccade, the task goal was no longer necessarily reinforced by the previous trial and, in fact, it was as likely that the goal from the previous trial (e.g., *look to the opposite side of the flash*) proac-

tively interfered with the goal for the trial that followed. Indeed, when the trials switched from prosaccades to antisaccades, or vice versa, both span groups made more errors than when the trial remained the same as the previous trial. Attention control should be important when responding to situations that involve competing responses and proactive interference. In these situations of increased interference, the need to actively maintain the task goal is of critical importance. The finding that low-span participants made more errors on both prosaccades and antisaccades suggests that they were deficient in their ability to keep the task goal actively maintained and thus were more susceptible to goal neglect than were high-span participants. In addition, the finding that low-span participants were now marginally slower on prosaccade trials suggests that these prosaccades were no longer being executed in a purely automatic fashion but, rather, were being executed via voluntary control.

Experiment 3

In our third and final experiment, we questioned whether the suppression of a reflexive saccade is important for differentiating high- and low-WM span participants. As noted previously, the antisaccade requires two processes over and above those required in the prosaccade: the generation and execution of a voluntary saccade and the suppression of a reflexive saccade. Thus, the differences found between high- and low-span participants in Experiments 1 and 2 (especially latency differences) may be due to differences in the generation of a correct voluntary saccade, the inhibition of a prepotent response, or both. On the basis of previous research, we had reason to believe that differences in latency are reflective of differences in the ability to generate a voluntary saccade. Indeed, as demonstrated in Experiment 2, low-span participants were marginally slower than high-span participants in their execution of prosaccades, $F(1, 38) = 3.46$, $p = .071$. Thus, low-span participants were slower to execute a saccade that required some form of control but that did not require the inhibition of a prepotent response. In addition, the work of Stuyven et al. (2000) has suggested that endogenously cued prosaccades are both slower than the typical exogenously cued prosaccades and more susceptible to interference (their Experiment 3). Stuyven et al. argued that endogenous prosaccades acted similarly to antisaccades and thus concluded: "no evidence was found for interference due to inhibition of a prepotent response over and above the interference effect found on planned saccades" (p. 82). Based on this evidence, we had high- and low-span participants perform four separate saccade tasks. Two of the tasks were exactly the same as those performed in Experiment 1 (i.e., prototypical exogenous prosaccade and antisaccade). In addition, participants performed an endogenous prosaccade and an endogenous antisaccade task. If the inhibition of a prepotent response is necessary for differentiating highs and lows, then we should only see differences in the two antisaccade tasks. If, however, the generation of a voluntary saccade is important, then we should see that low-span participants are slower on those saccade tasks that require control but do not differ on the relatively automatic exogenous prosaccade task. To be specific, we should see differences in the endogenous prosaccade task that requires a voluntary saccade but does not require the suppression of a reflexive saccade.

Method

Participants and Design

We selected 36 new high- and low-span participants, as determined by the OSPAN, from the same population as those in Experiments 1 and 2. The design was a 2 (WM span: high vs. low) × 2 (saccade type: antisaccade vs. prosaccade) × 2 (cue type: endogenous vs. exogenous) × 4 (task order) mixed factorial design with saccade type and cue type as the within-subjects variables. We counterbalanced task order using a Latin square with the stipulation that prosaccade and antisaccade trials alternated with either exogenous or endogenous tasks (see Table 2), respectively.

Procedure

The stimulus display and eye-tracking equipment were the same as those used in Experiment 1. Before receiving any experimental trials, participants received 6 trials of each saccade task in the order of exogenous prosaccade, exogenous antisaccade, endogenous prosaccade, and endogenous antisaccade, to become familiar with the tasks. In addition, each block of experimental trials was preceded by a practice session of 15 trials of the same type as the upcoming experimental block. Calibration was checked again following the first two blocks of the experiment.

For each trial, participants saw a black screen containing the word *ready* in the center of the screen and 1-cm (0.6° visual angle) white squares positioned at 11.5° of visual angle to the left and right of center. At the start of each trial, the word *ready* was presented in the center of the screen for 1,500 ms to warn the participant that a trial was about to begin. A fixation point (a white plus sign 1 cm × 1 cm) then appeared for a period that varied unpredictably between 600 and 2,200 ms in 100-ms increments. In the exogenous conditions, following the wait period, one of the squares flashed for 600 ms while the fixation point and the other square remained on the screen. In the endogenous conditions, however, the fixation point was replaced by an arrow pointing to either the left or the right of the screen for 600 ms.

In the exogenous antisaccade task, participants were required to make their first eye movement toward the square opposite the flashing square. In the exogenous prosaccade task, participants made a saccade toward the flashing square. In endogenous antisaccade task, participants were required to make a saccade to the box in thedirection opposite to that in which the arrow was pointing. In the endogenous prosaccade task, the participant made a saccade toward the box toward which the arrow was pointing. After the target flashed for 600 ms or the arrow remained on screen for 600 ms, the targets and center fixation point were removed, and the words *left* or *right* appeared at the correct target location. The target word then remained on the screen for 1,500 ms. Following presentation of the target word, the participant was given feedback on whether his or her first eye movement

Table 2
Task Order for Experiment 3

Order			
1	2	3	4
Tasks			
Ex. pro.	Ex. anti.	En. anti.	En. pro.
Ex. anti.	Ex. pro.	En. pro.	En. anti.
En. pro.	En. anti.	Ex. anti.	Ex. pro.
En. anti.	En. pro.	Ex. pro.	Ex. anti.

Note. Ex. pro. = exogenous prosaccade; Ex. anti. = exogenous antisaccade; En. anti. = endogenous antisaccade; En. pro. = endogenous prosaccade.

from the center was correct. After 1,500 ms of feedback, the next trial began.

Within each 75-trial block, the target position was random and equally likely to occur to the left or right of center. Participants completed four sets of 75 trials: two prosaccade sets, two antisaccade sets, two exogenous sets (one for prosaccade and one for antisaccade), and two endogenous sets, so that each participant completed 150 prosaccade and 150 antisaccade trials as well as 150 endogenous and 150 exogenous trials during the experiment. The same data-screening criteria were used as in Experiment 1. We eliminated 3.45% of the trials because either pupil reflectance was lost during the trial, or the first saccade was to a point outside the target areas. We removed 1.76% of the trials because the initial saccade was either slower than 1,000 ms or faster than 100 ms.

Results

Participants

Data for 4 low-span participants and 4 high-span participants were excluded from data analyses because of eye-tracker calibration problems. The mean OSPAN scores for the final 32 high-span and 32 low-span participants were 24.72 ($SD = 5.19$, range 19–37) and 5.91 ($SD = 2.79$, range 0–9), respectively. The mean ages for high- and low-span participants were 23.22 ($SD = 3.92$) and 22.75 ($SD = 4.64$) years, $t(62) = 0.437$, $p > .65$.

Correct Trial Latency

Latency was computed as the amount of time between the onset of the cue and the start of fixation in the area of interest. Only latencies of correct saccades were included in the analyses. The correct trial latency results are broken up into three sections: omnibus results, results contrasting automatic and controlled saccades, and results contrasting exogenous and endogenous prosaccades. Note that in all analyses, many of the effects of interest were qualified by significant interactions involving task order. However, all of these interactions took the same general form, suggesting that performance on exogenous prosaccade changes as a function of when it was performed. That is, when exogenous prosaccade is the first task performed, performance latencies are shorter than the other saccade tasks, and no individual differences appear. As more tasks are performed before exogenous prosaccade, performance on exogenous prosaccade becomes more like the other saccade tasks, with latencies getting longer and individual differences beginning to appear.

Omnibus results. The overall results suggest that endogenously cued saccades took longer to execute than exogenously cued saccades, and antisaccades took longer to execute than prosaccades. Furthermore, the results suggest that the largest WM span differences occurred in the endogenous cue condition. However, as noted above, most of these effects were qualified by significant interactions involving task order; therefore, only the higher order interactions are reported (see Appendixes A–C for the complete ANOVA results).

These observations were supported initially by a 2 (span) × 2 (cue type: exogenous vs. endogenous) × 2 (saccade type: prosaccade vs. antisaccade) × 4 (task order) mixed ANOVA with cue type and saccade type as the within-subjects variables. As expected, the analysis revealed main effects of cue type, $F(1, 56) = 47.69$, $p < .01$; and saccade type, $F(1, 56) = 45.81$, $p < .01$, indicating that performance on endogenously cued saccades took longer than performance on exogenously cued saccades ($M = 455$ ms, $SE = 6$ and $M = 423$ ms, $SE = 7$, respectively) and that antisaccades had longer latencies than prosaccades ($M = 451$ ms, $SE = 7$ and $M = 427$ ms, $SE = 6$, respectively). In addition, these two factors interacted with each other as indicated by the significant Cue Type × Saccade Type interaction, $F(1, 56) = 5.05$, $p < .05$. This interaction suggests that the performance differences between prosaccades and antisaccades are largest under exogenous conditions rather than endogenous conditions. That is, the mean difference between prosaccades and antisaccades under exogenous conditions was 33 ms, whereas the difference between prosaccades and antisaccades under endogenous conditions was only 16 ms.

However, each of the main effects as well as the two-way interaction were qualified by significant interactions involving task-order effects. To be specific, they were qualified by a significant three-way Cue Type × Saccade Type × Task Order interaction, $F(3, 56) = 7.49$, $p < .01$. Decomposition of this interaction, as shown in Table 3, suggests that the difference between exogenous prosaccade and antisaccade trials is the largest when exogenous tasks come before endogenous tasks (i.e., Task Orders 1 and 2), but that the difference is reduced the later the exogenous prosaccade is performed in the task.

Table 3
Mean Saccade Latency (in ms) by Task Order, Cue Type, and Saccade Type for Experiment 3

Cue and saccade type	Task order							
	1 (Expro. 1st)		2 (Exanti. 1st)		3 (Enanti. 1st)		4 (Enpro. 1st)	
	M	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*
Exogenous								
Prosaccade	373	14	405	14	420	14	431	14
Antisaccade	433	15	450	15	446	15	429	15
Endogenous								
Prosaccade	445	13	468	13	444	13	431	13
Antisaccade	476	15	456	15	453	15	467	15

Note. Expro. 1st = Exogenous prosaccade first task performed; Exanti. 1st = Exogenous antisaccade first task performed; Enanti. 1st = Endogenous antisaccade first task performed; Enpro. 1st = Endogenous prosaccade first task performed.

In terms of WM span differences, the analysis revealed a Span × Cue Type interaction, $F(1, 56) = 3.99$, $p = .051$, that approached conventional significance (partial $\eta^2 = .07$), indicating that there was no difference between high- and low-span individuals in the exogenous condition but that low-span participants were slower in the endogenous condition (e.g., mean differences between exogenous and endogenous were 41 ms for low-span participants and 23 ms for high-span participants, respectively). However, this effect was qualified by a significant three-way Span × Cue Type × Task Order interaction, $F(3, 56) = 3.66$, $p < .05$. As shown in Table 4, this interaction suggests that there are significant differences between exogenous and endogenous trials for low-span participants, with endogenous trials having longer latencies than exogenous trials in all task orders except for Task Order 4, although even here endogenous tasks are slightly slower (all ps $< .01$ except Task Order 4, $p > .19$). For high-span participants, however, the main differences between exogenously and endogenously cued trials occurred for Task Order 1 ($p < .01$). Thus, low-span participants showed differences regardless of task order, but high-span participants showed differences between exogenous and endogenously cued trials only when exogenous prosaccade was the first task performed.

Automatic versus controlled saccades. The finding that high- and low-span participants differed on endogenously cued saccades but not on exogenously cued saccades is interesting, because the exogenous cue condition is a mixture of putatively automatic and controlled saccades (i.e., exogenous prosaccade and exogenous antisaccade), whereas the endogenous cue condition is more reflective of controlled saccades. This is suggested by the fact that the Cue Type × Saccade Type interaction was significant, showing larger differences in the exogenous than in the endogenous cue conditions. Therefore, as a more sensitive test of the hypothesis that high- and low-span individuals differ only on controlled saccades, we examined WM span differences on saccades that required voluntary generation versus saccades that could be executed relatively automatically. Note that when we refer to voluntary (endogenous) control, we mean those situations in which interpretation of a cue is needed to direct the focus of attention. To be specific, in the context of the current study, endogenous control refers to situations in which a cue is presented that is informative about the location where the target will occur but that is not actually in that location (e.g., Klein & Shore, 2000). Hence, exogenous antisaccade requires endogenous control because the cue is informative about the where the target will appear (on the opposite side of the screen) but is not in that location. In addition, endogenous prosaccade and antisaccade require endogenous control because the cue indicates the target location but is presented at fixation. Thus, all of these conditions require the interpretation of the cue as well as the direction of the focus of attention to the correct location. Automatic responding on exogenous prosaccade trials, on the other hand, is accomplished because the cue is informative about the location of the target and is already in the location of the target. Thus, little cognitive work is needed to direct the focus of attention to the location of the target.

In order to confirm these impressions, we first looked at whether the saccade could be executed relatively automatically (e.g., exogenous prosacccade) or required endogenous control based on the criteria specified above (e.g., exogenous antisaccade, endogenous prosaccade, and endogenous antisaccade). Therefore, we compared automatic saccades with voluntary saccades by collapsing on exogenous antisaccade, endogenous prosaccade, and endogenous antisaccade, with a 2 (control type: automatic vs. voluntary) × 2 (span: high vs. low) × 4 (task order) mixed ANOVA with control type as the within-subjects variable. As expected, the analysis revealed a main effect of control type, $F(1, 56) = 102.2$, $p < .01$, suggesting that latency for automatic saccades was shorter ($M = 407$ ms, $SE = 7$) than it was for voluntary saccades ($M = 450$ ms, $SE = 6$). However, this effect was qualified by a significant interaction involving task order, $F(3, 56) = 11.90$, $p < .01$. Decomposition of this interaction suggests that latency for automatic saccades was shortest when exogenous prosaccade was the first task performed, and then latency linearly increased across the other task orders, $F(3, 60) = 2.78$, $p < .05$ ($p < .01$ for the linear contrast). For the voluntary saccades, however, task order did not have an effect, $F(3, 60) < 1$. Thus, as noted previously, exogenous prosaccade is affected by when in the experiment it is performed, with shorter latencies occurring the earlier in the experiment it was performed. For the other saccade tasks, order does not seem to have an effect.

In terms of WM span differences, the ANOVA yielded a significant Control Type × Span interaction, $F(1, 56) = 5.76$, $p < .05$, suggesting that the difference between automatic and volun-

Table 4
Mean Saccade Latency (in ms) by Task Order, WM Span, and Cue Type in Experiment 3

WM span and cue type	Task order							
	1 (Expro. 1st)		2 (Exanti. 1st)		3 (Enanti. 1st)		4 (Enpro. 1st)	
	M	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*
High								
Exogenous	398	20	453	20	447	20	395	20
Endogenous	468	18	461	18	443	18	412	18
Low								
Exogenous	408	20	402	20	419	20	464	20
Endogenous	454	18	463	18	454	18	485	18

Note. WM = working memory; Expro. 1st = exogenous prosaccade first task performed; Exanti. 1st = exogenous antisaccade first task performed; Enanti. 1st = endogenous antisaccade first task performed; Enpro. 1st = endogenous prosaccade first task performed.

tary saccades was greater for low-span participants (i.e., *M* difference = 53 ms) than for high-span participants (i.e., *M* difference = 33 ms). There was also a Span × Task Order interaction, $F(3, 56) = 3.11$, $p < .05$, indicating that high- and low-span participants differed in latency in the four different task orders.

Exogenous versus endogenous prosaccades. Finally, in order to better answer our question of primary interest (i.e., Is the voluntary generation of a saccade important in differentiating high- and low-WM span participants?), we conducted a repeated measures analysis on exogenous and endogenous prosaccade tasks only. Thus, if the voluntary generation of a saccade is important in differentiating highs and lows, we should see span differences in the endogenous prosaccade condition, when controlled saccades are made without the added complication of the need to suppress a reflexive saccade. We conducted a 2 (span) × 2 (task: exogenous prosaccade vs. endogenous prosaccade) × 4 (task order) ANOVA with task as the only repeated measures variable to test this idea. The ANOVA revealed a main effect of task, $F(1, 56) = 49.41$, $p < .01$, as well as a significant Task × Task Order interaction, $F(3, 56) = 8.81$, $p < .01$.

In terms of the primary question of interest, there was a significant Task × Span interaction, $F(1, 56) = 5.02$, $p < .05$, indicating that the difference between exogenous and endogenous prosaccade was greater for low-span participants (*M* difference = 52 ms) than it was for high-span participants (*M* difference = 27 ms). Thus, high- and low-span differences in saccade latency occur even when the inhibition of a prepotent response is not necessary. However, this effect, as well as those above, was qualified by a three-way interaction involving task order: Task × Span × Task Order, $F(3, 56) = 2.62$, $p = .06$, that approached conventional significance (partial $\eta^2 = .12$). This interaction is the same three-way interaction as the omnibus three-way reported previously.

Direction Errors

Direction errors represent those trials in which the participants moved their eyes in the direction opposite that of the target. For the analysis, the dependent measure is the percentage of direction errors for each task, respectively. The results suggest that overall error rates were quite low, but more errors occurred in both of the antisaccade tasks than in either of the prosaccade tasks. In addition, low-span participants made more errors than high-span participants on antisaccade trials with little to no differences occurring on prosaccade trials.

These observations were supported by a 2 (span) × 4 (task order) × 2 (cue type: exogenous vs. endogenous) × 2 (saccade type: prosaccade vs. antisaccade) mixed ANOVA with cue type and saccade type as the repeated measures. In terms of cue type, the only significant effect was a significant Cue Type × Span × Task Order interaction, $F(3, 56) = 3.15$, $p < .05$. This interaction suggests that high- and low-span participants do not differ in percentage of errors on exogenous trials but that significant differences appear for endogenous trials. To be specific, low-span participants make more errors on endogenous tasks than do high-span participants, but only when exogenous tasks are performed first.

Looking at differences between prosaccade and antisaccade trials, as expected, more errors were made for antisaccade trials than for prosaccade trials, $F(1, 56) = 74.91$, $p < .01$. In addition, a Span × Saccade Type interaction, $F(1, 56) = 3.56$, $p = .064$, that approached conventional significance ($\eta^2 = .06$), suggests that high- and low-span participants did not differ on the amount of errors made on the prosaccade tasks but that difference between percentage of errors for prosaccade and antisaccade trials was greater for low- than for high-span participants. That is, the mean difference between percentage of errors for prosaccade and antisaccade trials was greater for low-span participants (*M* difference = 3.96%) than it was for high-span participants (*M* difference = 2.54%). The only other effect to approach conventional significance was a four-way Cue Type × Saccade Type × Span × Task Order interaction ($p = .10$) that approached conventional significance (partial $\eta^2 = .11$).

Discussion

These results suggest that the latency differences observed between high- and low-span participants are not necessarily a function of the added time needed to suppress a reflexive saccade but, rather, are reflective of the time needed to engage in voluntary processing. Recall that if the suppression of a reflexive saccade is important for differentiating highs and lows, then differences should only emerge in the two antisaccade tasks. However, if the generation of a voluntary saccade is important, the differences should only emerge when voluntary saccade control is required. Thus, the findings support the notion that span differences in latency are reflective of differences in voluntary saccade control that is not necessarily reliant on the need to suppress a reflexive saccade. That is, low-span participants were hurt worse than high-span participants on those saccade tasks that required voluntary saccade generation, compared with exogenous prosaccade that could be executed relatively automatically. Furthermore, the finding that low-span participants were hurt worse on voluntary saccade generation than were high-span participants on the endogenous prosaccade supports the notion that the differences in latency are reflective of differences in voluntary processing but are not necessarily limited to differences in suppression. Low-span participants were hurt worse than high-span participants on a task that required voluntary processing but that did not require the simultaneous need to suppress a reflexive saccade.

We must be cautious in interpreting these results, however, because most of them were qualified by significant interactions involving task order. What are we to make of these order effects? These order effects suggested that latencies on exogenous prosaccade are affected by when in the experiment the exogenous prosaccade task was performed. To be specific, latencies on exogenous prosaccade are generally very short when it is the first task performed. However, the later in the experimental session the exogenous prosaccade task is performed, the more the latencies resemble those of the other saccade tasks. Thus, performance on exogenous prosaccade changes as a function of when in the experimental session it was performed, with task order having minor effects on the other saccade tasks.

One way of thinking about this is in terms of the degree of control that had been required previously. For the most part, exogenous prosaccades can be executed fairly automatically by simply allowing automatic attentional capture to do the work. The other saccade tasks all require that a controlled saccade be made. However, exogenous prosaccades are affected by the degree of

control that was used previously. To be specific, exogenous prosaccade had the shortest latencies when it was the first task performed, and then latency got progressively longer as the number of preceding trials that required controlled saccades increased. That is, in Task Order 2, only exogenous antisaccade precedes exogenous prosaccade, and thus only 75 trials requiring controlled saccades precede it. However, in both Task Orders 3 and 4, in which 225 and 150 controlled saccades were required, respectively, there is an almost 60-ms increase in saccade latency. Requiring a participant to engage in repeated controlled saccades seems to promote the use of controlled saccades even when they are not needed for accurate performance (i.e., exogenous prosaccade). This finding is not a new one. In fact, Kane et al. (2001, Experiment 2) found that low-span participants were slower on prosaccades than high-span participants if the prosaccade task followed many blocks of antisaccade trials (e.g., 360 trials). Like the results of the present study, performing many trials under attentional control seems to foster the use of control on those trials that do not require it, and WM span differences begin to emerge once attentional control is required for accurate responding.

Furthermore, the lack of a reliable difference between high- and low-span participants in latency for the exogenous antisaccade condition is surprising, given the results of Experiment 1 and previous research. However, WM span differences do emerge when you take into account the task-order effects. That is, looking at those conditions in which exogenous antisaccade follows exogenous prosaccade (i.e., Task Orders 1 and 4), there are significant differences, with low-span participants demonstrating longer antisaccade latencies than did high-span participants ($M = 455$ ms, $SE = 12$; $M = 407$ ms, $SE = 12$, respectively), $F(1, 30) = 7.55$, $p < .01$. However, when exogenous antisaccade trials came before exogenous prosaccade trials (i.e., Task Orders 2 and 3), no significant differences emerged, $F(1, 30) = 2.06$, $p > .16$. Thus, the task-order effects obscured WM differences on exogenous antisaccade trials. This is not a new finding. Indeed, in the Kane et al. (2001) study, task-order effects also obscured WM span differences in some conditions. Thus, it would seem that WM span differences on antisaccade trials are in part determined by the order in which the tasks are performed. It is clear that these task-order effects present a confusing picture of the relationship between performance on the antisaccade and individual differences in WM span. Understanding the reasons for these task-order effects represents an important research endeavor for the future.

General Discussion

In Experiments 1–3, we investigated the link between WM span and attentional control using variants of the antisaccade paradigm. Across the three experiments, it was shown that both processes needed for correct performance in the antisaccade (suppression of a reflexive saccade and generation of a correct voluntary saccade) are related to WM span. In Experiment 1, we demonstrated that high- and low-span participants do not differ on relatively automatic prosaccades in either errors or latency, but that low-span participants are hurt worse on antisaccade trials in terms of both errors and latencies even when the sole requirement is an eye movement. These results suggest that tasks like the OSPAN tap some of the same processes required in antisaccade trials but not in prosaccade trials. Furthermore, the results suggest that WM span differences emerge in conditions in which active goal maintenance is needed, especially in the face of potent distraction. That is, on antisaccade trials, if the task goal is not actively maintained (*if blink left—look right*), then any momentary lapse in attention will result in attention being captured by the salient cue, and thus prepotency will guide before. For prosaccades, however, both the task goal and the prepotent response coincide, and thus any lapse in attention will result in the correct behavior—and hence attentional control is not needed for accurate performance. Therefore, the finding that high- and low-span participants differed only on antisaccade trials suggests that low-span individuals are deficient in their ability to actively maintain task goals in the face of interference and thus are more susceptible to what Duncan (1995) has termed *goal neglect*.

Indeed, Experiment 2 furthered the notion that active maintenance and attentional control are important for differentiating between high- and low-span participants. Recall in Experiment 2 that prosaccade and antisaccade trials were no longer presented in a block format, but rather were intermixed within the same block of trials. According to our rationale, the intermixing of prosaccade and antisaccade trials within the same block served to increase the need for goal maintenance because the previous trial no longer predicted the upcoming trial and thus increased the likelihood of an occurrence of goal neglect. Therefore, in order to generate the correct response, the task goal was not continually reinforced on each and every trial, and thus participants were required to keep the task goal active on each and every trial. In such a situation, low-span participants not only performed worse on antisaccades in terms of both latency and errors, but now, they also performed worse on prosaccade trials. Here, if task goals are not actively maintained, then disorganized behavior will result, and an inappropriate response will occur. Notice that in such a situation, not all errors are reflective of differences in suppressing automatic attentional capture. That is, the finding that low-span individuals make more errors on prosaccade trials than do high-span individuals implies that low-span participants were actually making antisaccades on prosaccade trials. Thus, on these trials, low-span participants were not deficient in their ability to resist attention-capturing cues but, rather, were deficient in their ability to engage in the task-appropriate behavior.

Furthermore, the finding that low-span participants were marginally slower than high-span participants to generate correct prosaccades and antisaccades in Experiment 2 suggests that the latency differences observed previously may not be due exclusively to differences in suppression. In Experiment 3, we demonstrated that high- and low-span participants differ in latency on those tasks that require voluntary saccade control but do not differ on relatively automatic saccade generation (see Table 5 for an overview of latency and errors for Experiment 3). In addition, the finding that high- and low-span participants did not differ on exogenous prosaccades but that low-span participants were significantly hurt on endogenous prosaccades suggests that at least some of the latency differences observed are not reliant on the need to suppress a reflexive saccade. That is, low-span participants were hurt worse than high-span participants on a task that required voluntary processing but was not simultaneously reliant on the need to inhibit a prepotent response. In addition, although we observed significant latency differences between the two prosaccade tasks, no differences in errors emerged. Thus, executive

Table 5
Mean Saccade Latency (in ms) and Percentage of Direction Errors by Task and Span for Experiment 3

	Task															
	Ex. pro.				Ex. anti.				En. pro.				En. anti.			
	Latency		Error		Latency		Error		Latency		Error		Latency		Error	
Span	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*
High	410	13.00	1.3	0.31	437	13.00	4.3	0.63	437	9.00	.92	0.35	454	11.00	3.0	0.97
Low	404	9.00	.58	0.19	444	9.00	4.8	0.63	456	9.00	.88	0.22	472	11.00	4.6	0.81

Note. Ex. pro. = exogenous prosaccade; Ex. anti. = exogenous antisaccade; En. pro. = endogenous prosaccade; En. anti. = endogenous antisaccade.

attention is not only important for the inhibition of prepotent responses (suppression of reflexive saccades) but is also important for the endogenous control of the focus of attention.

Working Memory Span, Suppression, and the Voluntary Control of Attention

Previous researchers have demonstrated the importance of WM span differences in the suppression of information. For instance, Rosen and Engle (1998) found that low-span participants experienced more first-list intrusions on second-list learning in a paired-associate task than did high-span participants. In line with Wegner's theory of mental control, Brewin and Beaton (2002) demonstrated that a measure of WM span (operation span) was negatively related to the number of intrusions of thoughts of a white bear when participants were instructed not to think of a white bear. Thus, high-WM span participants are more proficient at suppressing task-irrelevant information. As discussed in the introduction, high- and low-WM span participants also differ on tasks in which environmental distractors must be suppressed in order for correct responding to occur. In both the dichotic listening study of Conway et al. (2001) and the Stroop study of Kane and Engle (2003), a prepotent response had to be inhibited for task-relevant responding to occur.

Like the results of the present study, these results suggest that high- and low-WM span individuals differ in their ability to effectively suppress task-irrelevant information and behavior. Low-span participants made more errors on antisaccade trials than did high-span participants. In addition, as Experiment 2 demonstrated, when prosaccade and antisaccade trials are intermixed within the same block of trials, low-span participants make more errors on prosaccades than do high-span participants. As we have argued, this difference on prosaccade errors reflects low-span participants' inability to effectively maintain task goals in active memory. On antisaccade trials (in the blocked format), a momentary loss of goal maintenance results in the execution of the prepotent response. In the intermixed design, a loss of goal maintenance will result in a global control deficit whereby prepotency may sometimes win out over control (i.e., executing a prosaccade on an antisaccade trial), but such a loss may also result in a more general disorganization of behavior where task-irrelevant responses are being executed, despite the nature of prepotency (executing an antisaccade on a prosaccade trial).

However, the fact that high- and low-span individuals differ in a variety of situations that require suppression does not mean that the sole differentiation of these individuals is the ability to effectively inhibit. Unlike Hasher, Zacks, and colleagues (Hasher & Zacks, 1988; Hasher, Zacks, & May, 1999), we do not consider the limiting function of WM span to necessarily be an inhibitory one but, rather, an attentional one, which could be oriented to maintenance or suppression. Although individuals will differ in their ability to effectively inhibit, we argue that this ability is due to differences in attentional control (e.g., Engle, Conway, Tuholski, & Shisler, 1995). Thus, one prediction from this framework is that individual differences in WM span will appear on tasks that require the endogenous control of attention but in the absence of the need to inhibit. Indeed, several studies have shown that high- and low-span individuals differ on tasks in which suppression of task-irrelevant information is not imperative for correct responding. Rather, these studies have shown WM span differences when endogenous or voluntary control is required but little to no differences under conditions of automatic or reflexive responding. For instance, Unsworth and Engle (in press) recently demonstrated that high- and low-span participants do not differ in learning on the serial reaction time task under unintentional (implicit) learning conditions, but that high-span participants demonstrate more learning under intentional learning conditions. Thus, on the face of it, the serial reaction time task does not require suppression of reflexive responding, but WM span differences still emerge when intentional, voluntary processing is required.

In addition, and more relevant to the current discussion, Bleckley and Engle (2002) explored the link between individual differences in WMC and the flexible allocation of visual attention. In a variant of Egly and Homa's (1984) visual attention task, Bleckley and Engle demonstrated that low-span participants used a diffuse spotlight-like allocation of attention, whereas high-span participants used a more flexible allocation of attention when endogenous cues were used (their Experiment 1). Bleckley and Engle argued that the endogenous cues taxed low-span participants' attentional capacity, resulting in a spotlight-like allocation. In order to test this, Bleckley and Engle had participants engage in the same task but with exogenous cues. Because exogenous cues capture attention automatically, low-span participants' attention should be freed, allowing them to engage in a more flexible allocation of attention. Indeed, their Experiment 2 showed that low-span participants began to show a more flexible allocation of attention similar to that of high-span participants with exogenous cues. Thus, like the present experiments, high- and low-span individuals differ only in situations in which attention needs to be

allocated voluntarily. Under automatic attentional capture, however, span differences do not emerge, because attentional control is not required for accurate performance. Executive attention is of critical importance where task goals and habitual responses are pitted against each other, and thus endogenous control of behavior is required for accurate performance.

This notion is similar to Cowan's theory of attention and memory (1988, 1995), in which the focus of attention is controlled conjointly by both a central executive component and automatic attentional capture. In situations in which the central executive and automatic attentional capture are in opposition, the central executive will direct the focus of attention only when the intent to do so is actively maintained. A momentary lapse in intention will result in automatic attentional capture. Within the antisaccade task, the central executive is needed not only to block automatic attentional capture, but also to effectively direct the focus of attention to the correct location. We argue that individual differences in WM span are reflective of differences in central executive functioning and, thus, the differences found on antisaccade trials reflect low-span participants' inability to prevent automatic attentional capture as well as their inability, relative to high-span participants, to effectively direct the focus of attention. Thus, the finding that low-span participants are more error-prone on antisaccade trials suggests that they are less effective at actively maintaining task goals and suppressing irrelevant responses. However, even when task goals are actively maintained and attentional capture is prevented, low-span participants will still be slower on trials that require endogenous control because they are slower to flexibly direct the focus of attention. Indeed, this notion is supported by the fact that low-span participants are hurt worse than high-span participants, in terms of latency, on endogenous prosaccades compared with exogenous prosaccades. According to this view, then, WM span differences should emerge only in conditions requiring blockage of automatic attentional capture by external and internal distractors or when the focus of attention needs to directed via endogenous control.

Limitations, Alternative Explanations, and Future Directions

One important limitation of the present work is that when dealing with quasi-experimental designs with individual-differences variables, one must be mindful that the study is essentially correlational in nature, and that, thus, causation cannot be inferred directly. For example, in Experiment 1, we found a WM Span $\times$ Saccade Type interaction such that differences appeared in the antisaccade condition but not in the prosaccade condition. From this, we might infer that differences in WM span caused performance differences in antisaccade tasks but not prosaccade tasks. However, because we are dealing with individual-differences variables, the correct inference is that variability in a WM span task covaries with variability in antisaccade but not prosaccade performance. Thus, what all three experiments suggest is that a putative measure of WM is related to putative measures of attentional control. Because the results of this study and similar studies suggest that WM span is related to attentional control, other variables may mediate or moderate this relationship.

One possible alternative explanation to our results is that high- and low-span participants simply differ in general speed of processing and thus, the differences observed here may be due to differences in speed-of-processing abilities rather than attentional control. One problem with this interpretation is that the construct of speed of processing is generally left underspecified and the question becomes "speed of what?" That is, what exactly is meant by speed of processing? In the present article, we have suggested that the speed differences observed between high- and low-span individuals in both the antisaccade and endogenous prosaccade tasks are, in part, a result of differences in the speed of moving the focus of attention to the correct location under controlled conditions. Thus, instead of suggesting that high- and low-span participants differ in global speed of processing, we have specified that span differences in these tasks are due to the efficiency of controlling the focus of attention. Indeed, Heitz and Engle (2004) recently demonstrated, via the Eriksen flanker paradigm, a similar notion by suggesting that high- and low-span participants differ in the rate at which they can constrain the focus of attention. Thus, it is not simply the case that high- and low-span individuals will always differ in the speed with which they can accomplish some task but, rather, differences will occur when the task requires that the focus of attention be moved rapidly, constrained rapidly, or even possibly switched rapidly (see also Heitz & Engle, 2004; Heitz, Unsworth, & Engle, in press).

Although we have suggested that high- and low-span participants differ in the speed of moving the focus of attention, this does not necessarily mean that these same participants do not also differ in global speed of processing, which may affect the results. For instance, several studies have shown that basic speed-of-processing tasks are sometimes related to WM span tasks (e.g., Ackerman, Beier, & Boyle, 2002; Conway et al., 2002). Thus, it is possible that whatever basic speed-of-processing tasks measure may mediate the relationship between WM spans and latency differences on antisaccade and endogenous prosaccade tasks. In order to better understand the possible role of speed of processing, it seems that two paths of inquiry need to be undertaken. One is a task analysis of speed-of-processing tasks to see what aspects of these putative measures of speed of processing are important to differences in WM span. Another path of inquiry is an examination of the relationship among WM span, attentional control, and speed of processing via a large-scale correlational study with multiple measures of each construct in order to fully understand the relationships among these constructs. We hope that, by examining both paths, we may be able to better understand the role of attentional control in WM spans and the possible role that differences in speed of processing may have in mediating or moderating this relationship.

Conclusion

In three experiments, we tested high- and low-WM span individuals on variants of the antisaccade paradigm. The experiments demonstrated that high- and low-span participants differed in their ability to effectively suppress reflexive saccades, with low-span participants making more reflexive saccade errors on antisaccade trials. Furthermore, when the need for attentional control was increased via a random mixing of prosaccade and antisaccade trials within the same block, low-span participants showed increased error rates on both antisaccades and prosaccades. Intermixing prosaccade and antisaccade trials was also shown to increase

low-span participants' correct trial latency on prosaccades compared with high-span participants' trial latency, suggesting that these saccades were no longer performed in a purely automatic fashion. Indeed, prosaccades generated under exogenous cuing versus endogenous cuing resulted in a larger increase in latency for low-span participants than for high-span participants. These results suggest that WM span is related to performance in the antisaccade paradigm when both the suppression of a reflexive saccade and the generation of volitional eye movement in the correct direction need to be controlled.

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Appendix A

Complete Analysis of Variance Results for Omnibus Latency Test From Experiment 3

Effects and interactions	*F*	*MSE*	*p*
Main effects			
S	0.52[a]	.005	.47
C	47.69[a]	.064	.00
SC	45.81[a]	.038	.00
TO	0.18[b]	.002	.91
Two-way interactions			
S × C	3.99[a]	.005	.05
S × SC	0.53[a]	.000	.48
S × TO	2.80[b]	.029	.05
C × SC	5.05[a]	.004	.03
C × TO	4.41[b]	.006	.01
SC × TO	3.81[b]	.003	.02
Three-way interactions			
S × C × SC	0.95[a]	.001	.33
S × C × TO	3.66[b]	.005	.02
S × SC × TO	0.78[b]	.001	.51
C × SC × TO	7.45[b]	.006	.00
Four-way interaction			
S × C × SC × TO	0.86[b]	.001	.47

Note. S = span; C = cue; SC = saccade; TO = task order.
[a] $df = 1, 56$
[b] $df = 3, 56$

Appendix B

Complete Analysis of Variance Results for Automatic and Controlled Saccades Latency Test From Experiment 3

Effects and interactions	*F*	*MSE*	*p*
Main effects			
S	0.102[a]	.001	.75
CT	102.20[a]	.058	.00
TO	0.73[b]	.004	.54
Two-way interactions			
S × CT	5.76[a]	.033	.02
S × TO	3.11[b]	.016	.03
C × TO	11.90[b]	.007	.00
Three-way interactions			
S × CT × TO	1.21[b]	.001	.31

Note. S = span; CT = control type; TO = task order.
[a] $df = 1, 56$
[b] $df = 3, 56$

Appendix C

Complete Analysis of Variance Results for Exogenous and Endogenous Prosaccade Latency Test From Experiment 3

Effects and interactions	*F*	*MSE*	*p*
Main effects			
S	0.287[a]	.001	.59
T	49.41[a]	.050	.00
TO	0.97[b]	.005	.42
Two-way interactions			
S × T	5.02[a]	.005	.03
S × TO	2.31[b]	.011	.09
T × TO	8.81[b]	.009	.00
Three-way interactions			
S × T × TO	2.62[b]	.003	.06

Note. S = span; T = task; TO = task order.
[a] $df = 1, 56$
[b] $df = 3, 56$

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