

# Attention control and the antisaccade task: A response time distribution analysis

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## ABSTRACT

In three experiments response time (RT) differences between correct prosaccade and antisaccade trials were examined via distribution analyses by fitting an ex-Gaussian function to individual RT distributions. Experiment 1 demonstrated that antisaccades are slower than prosaccades and this difference is due to both a shift in the overall distribution as well as a lengthening of the tail of the distribution. Experiment 2 demonstrated that manipulating foreperiod duration led to changes in both accuracy and RT for antisaccades but not prosaccades. Furthermore, the change in RT for antisaccades resulted in a lengthening in the tail of the distribution but not a shift in the distribution. Finally, Experiment 3 demonstrated that with sufficient practice performance on antisaccades was equated with performance on prosaccades in terms of both accuracy and RT. An examination of the RT distributions suggested that practice led to parallel changes in both the mean of the distribution and the tail of the antisaccade distribution. These results are interpreted within a two-factor theory of attention control that suggests that performance on antisaccades is driven by both competition resolution and goal-maintenance abilities.

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## 1. Attention control and the antisaccade task: a response time distribution analysis

A major function of our cognitive system is the ability to maintain current goals in the face of interference, especially when these current goals are in opposition to automatic/reflexive behavior (Engle & Kane, 2004). This important function is variously labeled as attention control, controlled attention, cognitive control, executive attention etc. and is thought to be reliant on intact frontal lobe circuitry. Regardless of the specific label, it is clear that this function of the system is needed in a number of real-world behaviors and is important for the successful attainment of goals in a multitude of situations. One task that has been used extensively to examine this function is the antisaccade task (Hallet, 1978). In the present study we examined the entire distribution of responses in this type of task to better understand the underlying processes that lead to successful performance.

### 1.1. Antisaccade

In the antisaccade task (Hallet, 1978; Hallet & Adams, 1980; see Everling & Fischer, 1998 for a review) participants are told to fixate on a central cue and after a variable amount of time, a flashing cue appears either to the right or left of fixation and participants have to shift their attention and gaze to the opposite side of the screen as

quickly and accurately as possible. In the control condition for this task, participants are instructed to shift their attention and gaze to the same side of the screen as the cue (i.e., prosaccade). Typically, participants are slower and more error prone on antisaccade trials compared to prosaccade trials. This has been taken as evidence that antisaccade trials require participants to inhibit reflexive orienting to the cue (i.e., inhibit a prosaccade) and generate a voluntary saccade to the opposite side of the screen.

Given that there is good deal of conflict between the automatic orienting response and the task goal, antisaccades require top-down attention control processes in order to prevent automatic attention capture and to program an endogenous saccade to the opposite side of the screen. Any lapse in attention will lead to the cue capturing attention and the occurrence of a reflexive prosaccade, and hence an error. This is because attention control is theoretically needed to actively maintain the task goal so that the reflexive prosaccade can be inhibited and a correct antisaccade can be generated. Furthermore, even when attention control is appropriately engaged so that the task goal is active, antisaccades will still be slower than prosaccades (i.e., the antisaccade cost, Godijn & Kramer, 2007) due to the time needed to program a voluntary saccade in the opposite direction and deal with lapses in goal maintenance.

Evidence consistent with the view that attention control is needed on antisaccade trials relative to prosaccade trials comes from a number of studies that have demonstrated that certain groups with attention control deficits make more errors and have slower correct antisaccade response times than control groups, but generally equivalent performance on prosaccades. For instance, patients with lesions to the dorsolateral prefrontal cortex typically make more antisaccade errors

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than control participants (e.g., Guitton, Bachtel, & Douglas, 1985) and patients with lesions to the frontal eye fields typically have slower antisaccades response times than control participants (e.g., Gaymard, Ploner, Rivaud, Vermersch, & Pierrot-Deseilligny, 1998). Furthermore, schizophrenic patients typically make more antisaccade errors and have slower antisaccade response times than control participants (e.g., McDowell & Clementz, 2001). Likewise, healthy individuals with low working memory capacities also make more antisaccade errors and have slower antisaccade response times than individuals with high working memory capacities (e.g., Kane, Bleckley, Conway, & Engle, 2001; Unsworth, Schrock, & Engle, 2004). Collectively, these results suggest that the ability to control attention seems to be important in terms of both preventing reflexive eye movements towards the exogenous cue (i.e., prevent erroneous prosaccade) and the ability to effectively generate a saccade in the correct direction (i.e., generate a correct antisaccade).

Clearly, deficits in attention control can lead to an increase in error rates and to slower correct antisaccade response times. However, it is not clear why or how attention control affects correct antisaccade response times. Recent work has suggested that antisaccades are slower than prosaccades (the antisaccade cost) because antisaccades require both the inhibition of the prepotent prosaccade response as well as the generation of a correct antisaccade response (e.g., Massen, 2004; Olk & Kingstone, 2003). Specifically, Olk and Kingstone (2003) demonstrated a markedly reduced antisaccade cost when inhibition was theoretically matched. That is, in conditions where endogenous prosaccade and antisaccade were used, the antisaccade cost was significantly reduced. These authors suggested that the need for inhibition was a major reason that antisaccades are slower than prosaccades. Other recent work has suggested that the antisaccade cost is partially due to differential demands on attention control and active maintenance between prosaccades and antisaccades (Godijn & Kramer, 2007, 2008). Specifically, Godijn and Kramer suggested that in order to generate a correct antisaccade the task goal must be actively maintained in working memory (Kane et al., 2001; Unsworth et al., 2004). Variation in attention demands (and hence attention control) can lead to variation in the extent to which the goal is properly maintained. When the goal is fully maintained, a relatively fast correct antisaccade will be generated. When the goal is not properly maintained, an erroneous prosaccade will be generated. However, when the task goal is only partially maintained, relatively slow, but correct antisaccades will be generated. Thus, Godijn and Kramer suggest that the need to inhibit prosaccades likely accounts for some of the antisaccade cost, but, attention control and working memory demands also likely account for some of the antisaccade cost. One issue with these studies, however, is that only mean response times (or latencies) are examined. As will be seen next, an examination of the full distribution of responses can be informative in terms of elucidating the underlying mechanisms of interest.

### 1.2. Response time distributions and attention control

Although an examination of mean response time (RT) has long been the primary means of examining differences in attention control in a variety of tasks, recent work has suggested that an examination of the full response time distribution can also be very informative (Heathcote, Popiel, & Mewhort, 1991; Hohle, 1965; Ratcliff, 1979; Rohrer & Wixted, 1994; Spieler, Balota, & Faust, 1996, 2000). For example, it is well known that RT distributions tend to be positively skewed (Luce, 1986). As such, a given manipulation might lead to a shift in the overall distribution, a lengthening of the tail of the distribution, or some combination of both. Thus, without an examination of the full RT distribution it is difficult to ascertain what led to an overall change in mean RT.

One way to examine these issues is to fit a mathematical function to the entire distribution and see if experimental manipulations lead

to a change in a particular parameter or set of parameters. One popular mathematical function that has been used extensively to examine changes in RT distributions is the ex-Gaussian. As its name suggests, the ex-Gaussian function is a convolution of an exponential and a Gaussian distribution which has been found to provide an accurate description of RT distributions and has been used as a tool in examining group and experimental differences in RT distributions (Heathcote et al., 1991; Hohle, 1965; Ratcliff, 1979; Rohrer & Wixted, 1994; Spieler et al., 1996, 2000). The ex-Gaussian has three parameters that describe the distribution:  $\mu$  (the mean of the Gaussian),  $\sigma$  (the standard deviation of the Gaussian), and  $\tau$  (the mean and standard deviation of the exponential). The mean of the ex-Gaussian distribution is simply  $\mu + \tau$ . Importantly, although none of these parameters reflect an underlying cognitive process, research has shown that certain parameters are affected more by some manipulations than others and that group differences can be localized to specific parameters (e.g., aging, West, 2001; ADHD, Leth-Steensen, Elbaz King, & Douglas, 2000). That is, manipulations that lead to a shift in the entire distribution should lead to a change in  $\mu$ , whereas manipulations that increase the tail of the distribution should lead to an increase in  $\tau$ .

Utilizing the ex-Gaussian, a number of studies have demonstrated that attention control manipulations have differential effects on the various parameters. Several studies have found that interference effects (i.e., the RT difference between congruent and incongruent trials) in a number of tasks is due to differences in  $\mu$ , with little or no differences in  $\tau$ , thus suggesting that interference was due to an overall shift in the distribution rather than a change in the tail of the distribution (e.g., Mewhort, Braun, & Heathcote, 1992; Spieler et al., 2000). For instance, both Mewhort et al. (1992) and Spieler et al. (2000) found that interference in a local-global task was primarily due to an increase in  $\mu$  in the incongruent condition. Likewise, Spieler et al. (2000) found that interference in two versions of the flanker task was due to an increase in  $\mu$ , with little or no change in  $\tau$ . Thus, increases in mean RT in these attention control tasks is typically due to a shift of the distribution rather than a lengthening of the tail.

An important exception to this trend is found in the Stroop task. Several studies (e.g., Heathcote et al., 1991; Spieler et al., 1996, 2000; see also De Jong, Berendsen, & Cools, 1999) have found that Stroop interference effects are due to increase in both  $\mu$  and  $\tau$ , suggesting that the increase in mean RT on incongruent Stroop trials is due to both a shifting of the distribution and a lengthening of the tails. To account for these effects, Kane and Engle (2003) suggested that the overall shift in the distribution reflected the fact that all incongruent trials require some degree of competition resolution compared to congruent trials. Thus, all incongruent trials are increased by a fixed amount of time relative to congruent trials. In accounting for the change in the tail of the distribution, Kane and Engle suggested that some small proportion of trials reflect failures of goal maintenance (i.e., goal-neglect, Duncan, 1995) whereby the task goal was temporarily lost but then recovered prior to an error actually being made. Thus, some trials were much slower than normal due to fluctuations in goal maintenance.

Support for this view comes from a study by De Jong et al. (1999). In this study, participants performed congruent and incongruent Stroop trials with either a long (2000 ms) or a short (200 ms) response-stimulus interval (RSI). De Jong et al. reasoned that the fast pace of the short RSI would keep attention tightly focused on the task goal, thereby preventing goal-neglect. The long RSI, however, should induce more goal-neglect as participants would have ample time between trials to think about things unrelated to the task at hand and thus the goal would not be as actively maintained. Thus, there should be a large Stroop effect with the long RSI, but little or no effect with a short RSI. Overall the results were consistent with these notions such that with a short RSI the Stroop effect was a non-significant 11 ms. With a long RSI the Stroop effect was 47 ms. Furthermore, rank

ordering the RTs from fastest to slowest suggested that the difference in the magnitude of the Stroop effect between the two RSI conditions was localized primarily in the slowest RTs. Thus, a manipulation thought to affect goal maintenance abilities lead to a change in the tail of the distribution but little or no shift in the distribution. These results suggest that in tasks such as Stroop, the increase in mean RT is likely due to two processes: one dealing with overall competition resolution on each trial (possibly inhibition) and another due to fluctuations in goal maintenance.

### 1.3. The present study

The goal of the present study was to examine the RT difference between prosaccade and antisaccade trials by examining the full distribution of responses. If the RT difference between prosaccade and antisaccade trials is due to differences in the need to inhibit a prosaccade and generate a voluntary antisaccade on every trial, one would expect that the entire antisaccade distribution should be shifted compared to the prosaccade distribution leading to a change in  $\mu$ . If, however, the RT difference is due to periodic failures of goal maintenance then one would expect that the tail of the antisaccade distribution should be lengthened compared to the prosaccade distribution leading to a change in  $\tau$ , but no change in  $\mu$ . Finally, if like the Stroop task, the RT difference is due to two processes (competition resolution and goal maintenance), then one would expect both a shift and a lengthening of the antisaccade distribution compared to the prosaccade distribution leading to an increase in both  $\mu$  and  $\tau$ .

These possibilities were examined in three experiments in which participants performed a large number of either prosaccade or antisaccade trials. As with prior antisaccade studies, mean RT and accuracy were examined for prosaccade and antisaccade trials. Furthermore, ex-Gaussian functions were fit to each individual's distribution of responses and the overall parameters were examined for differences between conditions. An examination of the entire distribution of responses should help elucidate the reason(s) why antisaccades are slower than prosaccades.

Before we present the experiments a brief note on the present antisaccade task is in order. In the current experiments we utilized a button press version of the antisaccade task (Kane et al., 2001; see also Guitton et al., 1985; Nieuwenhuis, Broerse, Nielen, & de Jong, 2004; Roberts, Hager, & Heron, 1994). In this version a white equal sign (=) appears to blink on one side of the screen and participants are instructed to move their attention and their eyes to the opposite side of the screen to identify a briefly presented target letter. Thus, in the current task we are not directly measuring eye movement responses but rather are inferring the presence of a saccade based on the need to quickly move the eyes to the correct side of the computer screen to identify the target letter. Prior work with this version of the task has shown that RTs for the target identification task and the saccade latencies for the actual eye movements are very similar and index the same overall information (e.g., Kane et al., 2001). Furthermore, this version of the task has been utilized frequently to examine individual differences in attention control and has been shown to have desirable psychometric properties (i.e., good reliability and validity; Unsworth, Redick, Lakey, & Young, 2010; Unsworth & Spillers, 2010). Thus, although this version of the antisaccade is slightly different than more traditional versions, the overall task structure, task requirements, and theoretical mechanisms involved are the same. That is, just as there are a number of ways to measure RTs (e.g., vocal, button press, etc.) and infer the processes underlying performance in other attention control tasks (e.g., Stroop), there are a number of ways to measure performance and mechanisms in the antisaccade task that should provide the same general information. For instance, in both traditional versions of the antisaccade and the button press version used here, participants theoretically have to maintain the task goal (look away

from the flashing cue) and program a correct saccade in the opposite direction in order to move their eyes to the correct location and identify the correct target letter in the button press version. Thus, goal maintenance requirements are likely very similar across versions leading to similar results. We further discuss these issues in the General discussion.

## 2. Experiment 1

The purpose of Experiment 1 was to examine RT differences between prosaccade and antisaccade trials via RT distribution analyses. Participants performed 250 prosaccade or 250 antisaccade trials. The ex-Gaussian function was fit to each individual's correct RT distribution and the parameters were examined for differences between prosaccade and antisaccade trials. As noted previously, if the increase in RT for antisaccades is due to a shift in the distribution we should expect to see differences in  $\mu$ . If the difference is due to a lengthening of the tail of the distribution we should see differences in  $\tau$ . Finally, if the difference is due to both a shift and a lengthening of the tail, differences should emerge in both  $\mu$  and  $\tau$ .

### 2.1. Method

#### 2.1.1. Participants and design

Participants were 50 undergraduate students recruited from the subject-pool at the University of Georgia. Participants were between the ages of 18 and 35 and received course credit for their participation. Twenty-five participants were randomly assigned to the antisaccade condition and 25 were randomly assigned to the prosaccade condition.

#### 2.1.2. Saccade task

In this task (Kane et al., 2001; see also Guitton et al., 1985; Nieuwenhuis et al., 2004; Roberts et al., 1994 for similar button press versions of this task) participants were instructed to stare at a fixation point which was onscreen for a variable amount of time (200–1800 ms). A flashing white “=” was then flashed either to the left or right of fixation (11.33° of visual angle) for 100 ms. This was followed by a 50 ms blank screen and a second appearance of the cue for 100 ms making it appear as though the cue (=) flashed onscreen. Following another 50 ms blankscreen the target stimulus (a B, P, or R) appeared onscreen for 100 ms followed by masking stimuli (an H for 50 ms and an 8 which remained onscreen until a response was given). The participants' task was to identify the target letter by pressing a key for B, P, or R (the keys 1, 2, or 3) as quickly and accurately as possible. In the prosaccade condition the flashing cue (=) and the target appeared in the same location. In the antisaccade condition the target appeared in the opposite location as the flashing cue. Participants received, in order, 10 practice trials to learn the response mapping, 15 practice trials of that particular saccade task (either pro or antisaccade), and 250 real trials of that particular saccade task.

### 2.2. Results

For all the RT results reported, only correct RTs were examined. In addition, RTs that fell below 200 ms or 3 SDs below the individual's mean or above 2000 ms or 3 SD above the individual's mean were excluded from all RT analyses. This resulted in the exclusion of less than 2% of the responses.

Consistent with prior work, performance on the antisaccade task was poorer than that on the prosaccade task. Specifically, as shown in Table 1, performance on the prosaccade task was more accurate and correct saccade RTs were faster than on the antisaccade task, both  $t$ 's > 5.03, both  $p$ 's < .01. Furthermore, overall accuracy and RTs for both conditions were very similar to values reported by Kane et al. (2001) using the exact same task. Thus, we replicated the well known

**Table 1**

Accuracy and correct response time (in ms) as a function of saccade task and Experiment 1.

	Accuracy	Response time
Prosaccade	.92 (.01)	545 (26)
Antisaccade	.62 (.03)	737 (28)

Note. Values in parentheses reflect one standard error of the mean.

difference between prosaccade and antisaccade trials in terms of both accuracy and RT.

Next, we examined the full RT distributions for both tasks in order to examine our question of primary interest. That is, what accounts for the large (i.e., 192 ms) RT difference between prosaccade and antisaccade trials? To examine this, we fit an ex-Gaussian function to each individual's raw RT distribution using QMLE (Brown & Heathcote, 2003). All fits successfully converged within 250 iterations. For each individual we obtained the three parameters of the ex-Gaussian. These parameters were then examined as a function of saccade task (i.e., prosaccade vs. antisaccade). Shown in Fig. 1a are the RT distributions for prosaccade and antisaccade trials. As can be seen, the distribution for antisaccades is both shifted and has an increased tail relative to prosaccades. These impressions were confirmed by examining each parameter separately as a function of saccade task (see Table 2). As can be seen, there were differences between prosaccade and antisaccade trials on all three ex-Gaussian parameters. Specifically, the difference in  $\mu$  between prosaccade and antisaccade trials (73 ms) was significant,  $t(48) = 3.13$ ,  $p < .01$ , as was the difference in  $\sigma$  (63 ms),  $t(48) = 2.95$ ,  $p < .01$ , and the difference in  $\tau$  (120 ms),  $t(48) = 3.36$ ,  $p < .01$ . Thus, the RT difference between prosaccade and antisaccade trials seems to be due to both a shift in the

**Table 2**

Ex-Gaussian parameter estimates as a function of saccade task for Experiment 1.

	$\mu$	$\sigma$	$\tau$
Prosaccade	322 (10)	65 (12)	223 (24)
Antisaccade	395 (21)	128 (17)	343 (26)

Note. Values in parentheses reflect one standard error of the mean.

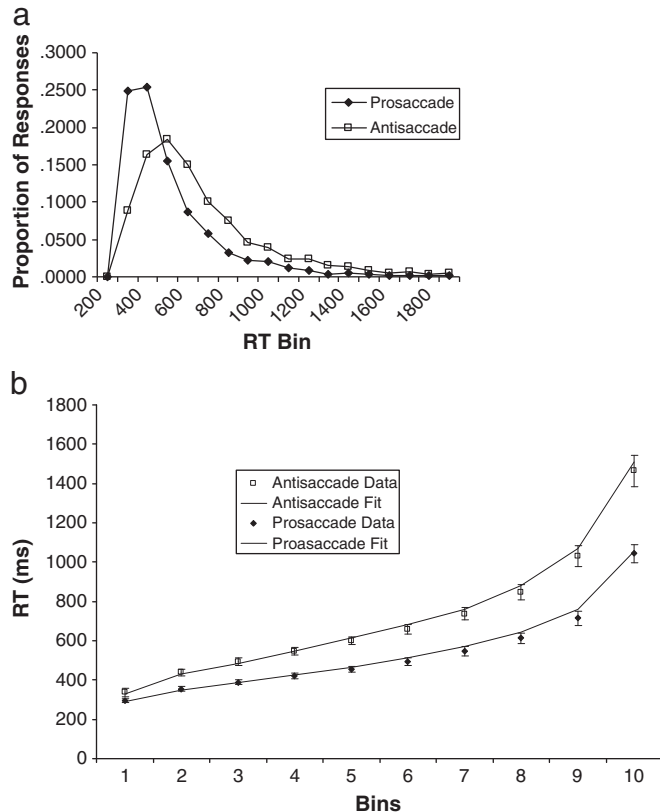
distribution (and subsequent increase in spread) as well as a lengthening of the tail. Furthermore, these two factors seem to be fairly independent given that the correlation between  $\mu$  and  $\tau$  (for both prosaccade and antisaccade trials together) was close to zero,  $r(50) = .01$ ,  $p > .95$ .

In addition to examining fits of the ex-Gaussian to the RT distributions, we also examined the raw distribution via vincentizing (e.g., Ratcliff, 1979). Specifically, we ranked ordered each individuals RTs from fastest to slowest (for both the prosaccade and antisaccade conditions) and then created 10 vincentile bins in which the fastest 10% of responses were included in the first bin, the next fastest 10% in the next bin and so on. Importantly, vincentizing allows for an examination of the raw RT distributions without making assumptions about the underlying shape of the distribution. Furthermore, vincentizing can be used to check the degree of fit between the ex-Gaussian and the raw distribution by plotting both and looking for any discrepancies between the raw data and the fit of the model (see Balota, Yap, Cortese, & Watson, 2008; Yap, Balota, Tse, & Besner, 2008). Shown in Fig. 1b are the resulting vincentiles for prosaccade and antisaccade conditions. The symbols represent the raw data (with associated standard errors) and the lines represent the fit of the ex-Gaussian. As can be seen, the antisaccade RT distribution is both shifted over and there is a lengthening of the tail compared to the prosaccade distribution. Furthermore, note that the ex-Gaussian fits the overall data quite well with only minor divergences between the fit and the raw data. Thus, fitting the ex-Gaussian to the data and as well as examining the overall distributions via vincentizing results in the same overall conclusions.

One potential problem with the results thus far is that it is possible that the difference seen in  $\tau$  could be due to differences in posterror slowing. That is, prior research has suggested that participants slow down following an error (Laming, 1979; Rabbitt, 1966), and given that accuracy was lower on antisaccade trials, it is possible that these slow responses in the tail of the distribution simply reflect posterror slowing trials. To examine this we eliminated all correct trials following errors and once again fit the ex-Gaussian to each individual's corresponding RT distribution. Similar to the overall analyses there were differences between prosaccade and antisaccade trials on all three ex-Gaussian parameters. Specifically, the difference in  $\mu$  between prosaccade and antisaccade trials (87 ms) was significant,  $t(48) = 3.78$ ,  $p < .01$ , as was the difference in  $\sigma$  (44 ms),  $t(48) = 2.02$ ,  $p < .05$ , and the difference in  $\tau$  (95 ms),  $t(48) = 2.58$ ,  $p < .05$ . Thus, distributional differences between prosaccade and antisaccade trials were not due to differences in posterror slowing.<sup>1</sup>

### 3. Discussion

The results from Experiment 1 were fairly straightforward. Replicating prior research, performance was better on prosaccade trials than antisaccade trials in terms of both accuracy and RT. Like previous work, correct antisaccades were much slower than correct prosaccades. An RT distribution analysis suggested that this RT



**Fig. 1.** (a) Response time distributions for prosaccade and antisaccade tasks for Experiment 1. (b) Vincentile plots of the antisaccade and prosaccade distributions for Experiment 1. Data points and standard errors represent participants' mean vincentiles. Best fitting ex-Gaussian vincentiles are represented by lines.

<sup>1</sup> Note, similar analyses were conducted for Experiments 2 and 3 in which posterror trials were eliminated from the distribution and ex-Gaussian for the resulting distribution was examined. Eliminating posterror trials led to virtually identical results as those reported for both experiments.



difference was due to both a shift in the antisaccade distribution and a lengthening of the tail of the antisaccade distribution relative to the prosaccade distribution. Thus, like similar work with the Stroop task (Heathcote et al., 1991; Spieler et al., 1996, 2000), it seems as if the difference in RT is due to multiple factors. In particular, as suggested by prior work, it is possible that the difference in RT between prosaccades and antisaccades is due to the fact that antisaccades require a constant amount of time over and above that required by prosaccades in order to inhibit the prepotent response and generate a correct antisaccade (i.e., competition resolution). In addition, as suggested by prior work, it is possible that antisaccades are also slower due to periodic goal-neglect that influences a small proportion of trials. In combination these two factors can account for the overall shift in the distribution (competition resolution) and the lengthening of the tail (goal-neglect). This suggests that the difference in RT (the antisaccade cost) is due to a mixture of factors that are possibly independent. Indeed, as noted previously  $\mu$  and  $\tau$  were uncorrelated. In order to examine this claim more thoroughly one would need to demonstrate that certain manipulations affect one parameter (i.e.,  $\tau$ ) without affecting the other parameter (i.e.,  $\mu$ ). The second experiment was aimed at examining this notion more thoroughly.

## 4. Experiment 2

The purpose of Experiment 2 was to examine the extent to which RT differences between prosaccades and antisaccades in  $\mu$  and  $\tau$  are due to different factors. Specifically, if estimates of  $\mu$  and  $\tau$  reflect different processes (competition resolution and goal-neglect, respectively) in the current situation, then it should be possible to dissociate the two such that manipulations affect one estimate but have no influence on the other. To test this, we decided to examine the effect of manipulating foreperiod on prosaccade and antisaccade RTs. Manipulating foreperiod has long been known to affect subsequent RTs (e.g., Hohle, 1965; Jennings & van der Molen, 2005; Niemi & Näätänen, 1981). Specifically, it has been found that when the foreperiod (i.e., inter-trial interval) is varied randomly from trial to trial, RTs are slowest for the shortest foreperiod and then asymptote for longer foreperiods (Niemi & Näätänen, 1981). Although there are a number of factors that influence this result (i.e., foreperiod expectancy and prior foreperiod length) and a number of potential reasons for it, one long standing explanation is that it takes time to properly focus attention and with short foreperiods attention is not adequately engaged leading to longer RTs (e.g., Woodrow, 1914). From a goal maintenance perspective this would mean that during the foreperiod attention is needed to activate and maintain the task goal in order to properly respond on the upcoming trial (e.g., De Jong et al., 1999; Kane & Engle, 2003). With a short foreperiod it is possible that the task goal is not properly maintained, leading to a greater likelihood of goal-neglect, and thus a greater likelihood of making an error or having an especially slow RT. As foreperiod increases, the likelihood of adequately activating and maintaining the task goal should increase leading to less goal-neglect. Thus, this suggests that foreperiod effects should selectively influence  $\tau$  and have little or no effect on  $\mu$ . To examine this notion, participants performed 350 prosaccade or antisaccade trials with variable foreperiods (i.e., 200, 600, 1000, 1400, or 1800 ms).

### 4.1. Method

#### 4.1.1. Participants and design

Participants were 44 undergraduate students recruited from the subject-pool at the University of Georgia. Participants were between the ages of 18 and 35 and received course credit for their participation. Twenty participants were randomly assigned to the antisaccade condition and 24 were randomly assigned to the prosaccade condition.

### 4.1.2. Saccade task

The same tasks as Experiment 1 were used. The only difference was that we increased the number of trials from 250 to 350 for both saccade tasks to ensure that there would be enough trials at each foreperiod to examine the resulting RT distributions.

## 5. Results

First we examined accuracy for prosaccade and antisaccades as a function of foreperiod. As can be seen in Fig. 2, prosaccades were more accurate than antisaccades,  $F(1, 42) = 96.34$ ,  $MSE = .04$ ,  $p < .01$ ,  $\eta_p^2 = .70$ . Furthermore, accuracy tended to increase as foreperiod increased,  $F(4, 168) = 8.67$ ,  $MSE = .002$ ,  $p < .01$ ,  $\eta_p^2 = .17$ . Importantly, as shown in Fig. 2, the effect of foreperiod interacted with the saccade task,  $F(4, 168) = 5.60$ ,  $MSE = .002$ ,  $p < .01$ ,  $\eta_p^2 = .12$ . Specifically, there was no effect of foreperiod on prosaccades,  $F(4, 92) = 1.13$ ,  $MSE = .001$ ,  $p > .34$ ,  $\eta_p^2 = .04$ , but there was an effect of foreperiod on antisaccades,  $F(4, 76) = 7.86$ ,  $MSE = .003$ ,  $p < .01$ ,  $\eta_p^2 = .29$ . Thus, at short foreperiods, antisaccade errors were more likely than at longer foreperiods. However, foreperiod duration had no effect on prosaccades.

Next, we examined RTs for prosaccade and antisaccades as a function of foreperiod. For all the RT results reported, only correct RTs were examined. In addition, RTs that fell below 200 ms or 3 SDs below the individual's mean or above 2000 ms or 3 SD above the individual's mean were excluded from all RT analyses. This resulted in the exclusion of less than 2% of the responses. As shown in Fig. 3, prosaccades were faster than antisaccades,  $F(1, 42) = 27.68$ ,  $MSE = 60896$ ,  $p < .01$ ,  $\eta_p^2 = .40$ . Furthermore, RT tended to decrease as foreperiod increased,  $F(4, 168) = 5.81$ ,  $MSE = 3171$ ,  $p < .01$ ,  $\eta_p^2 = .12$ . However, like the accuracy results, foreperiod duration and saccade task interacted,  $F(4, 168) = 5.46$ ,  $MSE = 3171$ ,  $p < .01$ ,  $\eta_p^2 = .12$ . Specifically, like the accuracy results, foreperiod duration had no effect on prosaccades,  $F(4, 92) = .14$ ,  $MSE = 1249$ ,  $p > .96$ ,  $\eta_p^2 = .006$ , but foreperiod duration did have an effect on antisaccades,  $F(4, 76) = 5.94$ ,  $MSE = 5497$ ,  $p < .01$ ,  $\eta_p^2 = .24$ . Thus, antisaccade costs were largest at the shortest foreperiod, but there was still an antisaccade cost even at the longest foreperiod. Furthermore, as shown in Fig. 3 the effect of foreperiod duration on antisaccades was isolated to antisaccade RTs for the shortest foreperiod (i.e., 200 ms). Consistent with prior work on foreperiod effects, the largest effect of foreperiod occurred at the shortest duration (e.g., Niemi & Näätänen, 1981).

In order to examine RT differences in more detail, we next examined the full RT distributions for both tasks. Like Experiment 1, we fit an ex-Gaussian function to each individual's raw RT distribution using QMLE (Brown & Heathcote, 2003). All fits successfully converged within 250 iterations. For each individual we obtained

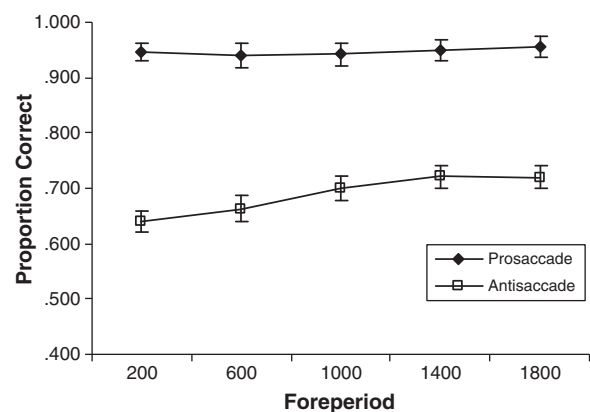
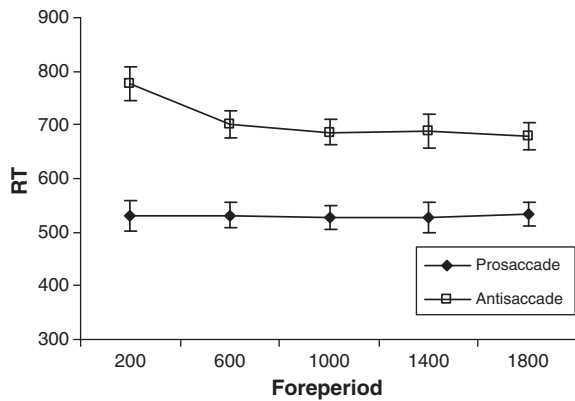


Fig. 2. Proportion correct for prosaccade and antisaccade tasks as a function of foreperiod duration for Experiment 2. Error bars represent one standard error of the mean.



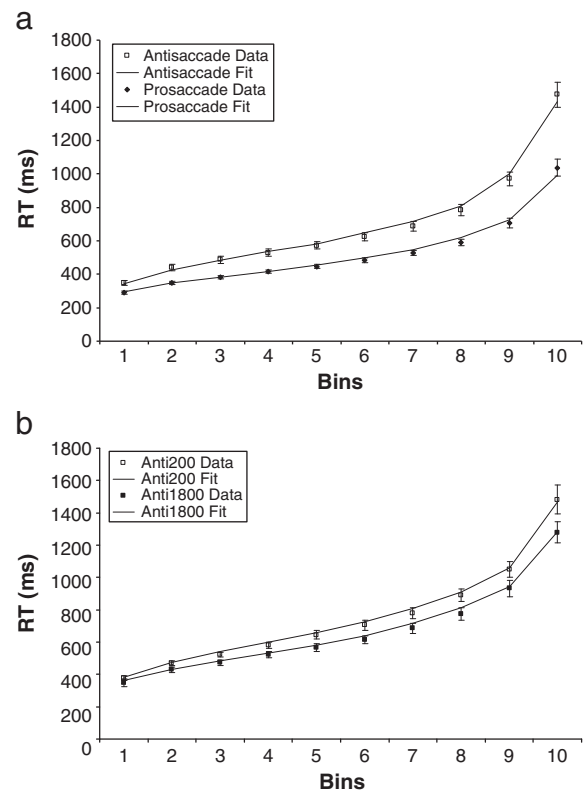
**Fig. 3.** Correct response times for prosaccade and antisaccade tasks as a function of foreperiod duration for Experiment 2. Error bars represent one standard error of the mean.

the three parameters of the ex-Gaussian. These parameters were then examined as a function of saccade task (i.e., prosaccade vs. antisaccade).

Similar to Experiment 1, an examination of the three ex-Gaussian parameters as a function of saccade task suggested differences in all three parameters. Specifically, as shown in Table 3, the difference in  $\mu$  between prosaccade and antisaccade trials (73 ms) was significant,  $t(42) = 3.61$ ,  $p < .01$ , as was the difference in  $\sigma$  (37 ms),  $t(42) = 2.26$ ,  $p < .05$ , and the difference in  $\tau$  (101 ms),  $t(42) = 3.56$ ,  $p < .01$ . Furthermore, these two factors seem to be fairly independent given that the correlation between  $\mu$  and  $\tau$  (for both prosaccade and antisaccade trials together) was close to zero,  $r(44) = .10$ ,  $p > .50$ . Like Experiment 1, these results suggest that the RT difference between prosaccade and antisaccade trials seems to be due to both a shift in the distribution (and subsequent increase in spread) as well as a lengthening of the tail.

Similar to Experiment 1 we also examined the distributions via vincentizing. Shown in Fig. 4a are the resulting vincentile plots for the prosaccade and antisaccade distributions and the resulting fits of the ex-Gaussian. Consistent with Experiment 1, the antisaccade distribution was both shifted over and had an increased tail compared to the prosaccade distribution. Furthermore, like Experiment 1, the data was well fit by the ex-Gaussian.

For our final set of analyses we examined what aspect of the overall antisaccade distribution would be affected by varying foreperiod duration. Specifically, as shown in Fig. 3 there was a clear RT difference (98 ms) between the shortest foreperiod and the longest foreperiod. If this RT difference is due periodic failures of goal maintenance that occur at the shortest foreperiod, we should see that this difference is due to an increase in the tail of the distribution with no shifting of the overall distribution. If, however, this RT difference is due to differences in the time needed to program an accurate antisaccade, we should see a shift in the distribution without necessarily seeing a change in the tail. To examine this, we fit an ex-Gaussian to each individual's antisaccade RT distribution for both the 200 ms foreperiod duration and the 1800 ms foreperiod duration and compared the resulting parameter estimates. As can be seen in Table 4, foreperiod duration had no effect on  $\mu$ ,  $t(19) = .64$ ,  $p > .52$ , but did effect both  $\sigma$ ,  $t(19) = 3.05$ ,  $p < .01$ , and  $\tau$ ,  $t(19) = 2.35$ ,  $p < .05$ . Thus, the RT difference between the shortest and the longest foreperiod



**Fig. 4.** (a) Vincentile plots of the antisaccade and prosaccade distributions for Experiment 2. Data points and standard errors represent participants' mean vincentiles. Best fitting ex-Gaussian vincentiles are represented by lines. (b) Vincentile plots of the 200 ms and 1800 ms foreperiod duration antisaccade distributions for Experiment 2. Data points and standard errors represent participants' mean vincentiles. Best fitting ex-Gaussian vincentiles are represented by lines.

duration resulted in an increase in the tail of the distribution, but did not shift the distribution.

Similar to the prior analyses we also examined the distributions via vincentizing. Shown in Fig. 4b are the resulting vincentile plots for 200 ms foreperiod duration and the 1800 ms foreperiod duration antisaccade distributions and the resulting fits of the ex-Gaussian. Consistent with the prior analyses, the main difference between the 200 ms and 1800 ms foreperiod durations was a lengthening of the 200 ms duration compared to the 1800 ms duration. Indeed, as shown in Fig. 4b, this effect was mainly localized to the slowest vincentile. Furthermore, similar to the prior analyses the data was well fit by the ex-Gaussian.

## 6. Discussion

The results from Experiment 2 wholly replicated those of Experiment 1 suggesting that the RT difference between prosaccade and antisaccade trials is due to both a shifting and lengthening of the tail of the antisaccade distribution relative to the prosaccade distribution. Furthermore, an examination of foreperiod duration effects suggested that performance at the shortest foreperiod duration was both less accurate and slower than performance at the longest

**Table 3**  
Ex-Gaussian parameter estimates as a function of saccade task for Experiment 2.

	$\mu$	$\sigma$	$\tau$
Prosaccade	325 (9)	54 (3)	203 (14)
Antisaccade	398 (19)	91 (17)	304 (26)

Note. Values in parentheses reflect one standard error of the mean.

**Table 4**  
Ex-Gaussian parameter estimates as a function of foreperiod duration for antisaccades for Experiment 2.

	$\mu$	$\sigma$	$\tau$
200 ms foreperiod	390 (28)	201 (46)	387 (57)
1800 ms foreperiod	405 (22)	98 (17)	276 (17)

Note. Values in parentheses reflect one standard error of the mean.

foreperiod duration, but only for antisaccade trials. That is, foreperiod had no effect on prosaccade trials, but on antisaccade trials the shortest foreperiod resulted in more errors and slower RTs. Furthermore, for antisaccade trials, the effect of foreperiod duration was due to an increase in the tail of the distribution without shifting the distribution over. These results are consistent with the notion that the RT difference in  $\tau$  between prosaccade and antisaccade trials is due to periodic lapses in goal maintenance (i.e., goal-neglect). Furthermore, these results are consistent with the notion that it takes time to properly activate the task goal in working memory and keep it properly maintained. When there is little time for preparatory attention to activate the task goal, goal neglect is more likely to occur resulting in either more errors or in longer RTs on a subset of trials (i.e., and increase in  $\tau$ ). Overall, these results are consistent with the notion that the RT difference between prosaccade and antisaccade trials is due to a mixture of processes. Differences in  $\mu$  seem to be due to differences in the need to inhibit a prosaccade and program a correct antisaccade, whereas differences in  $\tau$  seem to be due to differences in goal maintenance requirements between the tasks. The results from Experiment 2 suggest that these two processes are, at least, partially dissociable.

### 7. Experiment 3

The purpose of Experiment 3 was to examine how practice or training on the antisaccade would change performance. Specifically, prior work has suggested that inhibition of a prosaccade and programming of antisaccade as well as goal maintenance are controlled processes, whereas prosaccade performance is driven by relatively automatic processes (Hallet, 1978; Hallet & Adams, 1980; see Everling & Fischer, 1998 for a review). Thus, if automatic and controlled processes can be seen as falling on a continuum (e.g., Cohen, Dunbar, & McClelland, 1990), it should be possible to make processes necessary for antisaccade performance relatively automatic. That is, with enough training performance on the antisaccade should resemble performance on prosaccade in terms of both accuracy and RT leading to a reduction and possible elimination of the antisaccade cost. Prior work has suggested that extensive antisaccade training can lead to increases in accuracy and decreases in RT (Dyckman & McDowell, 2005). Furthermore, if the antisaccade cost is due to the fact that, at least, two processes are controlled, then with sufficient practice we should see changes in both processes. Specifically, with practice we should see both a shift in and a reduction in the tail of the antisaccade distribution such that the distribution is similar to the distribution for prosaccades. To examine this, we had participants participate in four days of practice on the antisaccade in which participants completed a total of 3500 antisaccade trials.

### 8. Method

#### 8.1. Participants and design

Participants were 22 undergraduate students recruited from the subject-pool at the University of Georgia. Participants were between the ages of 18 and 35 and received course credit for their participation. All participants came to the laboratory on Monday and completed 500 trials of antisaccade. On the next three days (Tuesday, Wednesday, and Thursday) all participants returned and completed 1000 antisaccade trials on each day.

#### 8.2. Saccade Task

The same antisaccade task as Experiments 1 and 2 was used. The only difference was that now participants received 3500 real trials. Overall, there were 14 blocks of 250 trials each.

### 9. Results

Analyses of practice/training on the antisaccade as a function of training block suggested that performance dramatically improved across the 14 training blocks. Specifically, as shown in Fig. 5, proportion correct on the antisaccade improved over the training blocks from 68% correct on the first block of training to 92% on the last block of training. This effect was statistically significant,  $F(13, 273) = 45.52$ ,  $MSE = .01$ ,  $p < .01$ , partial  $\eta^2 = .68$ . It should be noted that performance across blocks within a given day tended to remain fairly static, but performance across days tended to increase.

Next, we examined correct RT as a function of training block. Similar to the prior experiments, RTs that fell below 200 ms or 3 SDs below the individual's mean or above 2000 ms or 3 SD above the individual's mean were excluded from all RT analyses. As shown in Fig. 6, correct antisaccade RTs became significantly faster as a function of training block,  $F(13, 273) = 31.67$ ,  $MSE = 3290$ ,  $p < .01$ , partial  $\eta^2 = .60$ . Correct antisaccade RTs dropped by over 230 ms going from 703 ms on the first training block to 469 ms on the last training block. These results suggest that training on 3500 trials on the antisaccade task resulted in dramatic improvements in task performance (see also Dyckman & McDowell, 2005).

Next in order to examine the drop in RT as a function of training in more detail, we fit an ex-Gaussian to each individual's RT distributions for the first six blocks of trials. We only examined the first six blocks of trials given that is where the drop in RT seemed to asymptote. First, examining  $\mu$ , the results suggested that  $\mu$  decreased substantially as a function of training block,  $F(5, 105) = 13.53$ ,  $MSE = 2674$ ,  $p < .01$ , partial  $\eta^2 = .39$ . Specifically, as shown in Fig. 7a,  $\mu$  dropped from 427 ms to 325 ms in the first six blocks of training. As shown in Fig. 7b and c similar drops were found for both  $\sigma$  and  $\tau$ . Specifically, there was a significant decrease in  $\sigma$ ,  $F(5, 105) = 7.50$ ,  $MSE = 492$ ,  $p < .01$ , partial  $\eta^2 = .26$ . There was also a significant decrease in  $\tau$ ,  $F(5, 105) = 6.62$ ,  $MSE = 4730$ ,  $p < .01$ , partial  $\eta^2 = .24$ , such that  $\tau$  dropped from 271 ms to 166 ms. Thus, training served to drastically reduce mean RT and this reduction was seen in all three parameters of the ex-Gaussian distribution such that the overall distribution shifted over to lower values, the distribution became tighter, and the tail substantially decreased.

Similar to the prior experiments we also examined the distributions via vincentizing. Shown in Fig. 8 are the resulting vincentile plots for the antisaccade blocks 1–6 and the resulting fits of the ex-Gaussian. Consistent with the prior analyses, with training the antisaccade distributions shifted to lower values and the tail was substantially reduced. Furthermore, like the prior experiments, the data was well fit by the ex-Gaussian.

For our final set of analyses we wanted to determine if these changes in antisaccade performance (both accuracy and RT) as a function of training would bring performance to the same levels as prosaccade performance. To examine this, we compared performance on the last block of training to prosaccade performance from

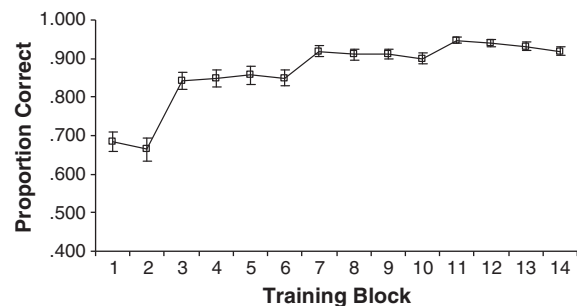
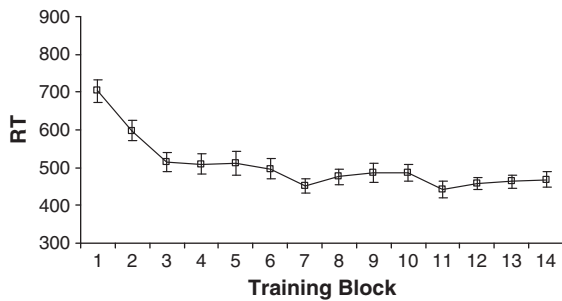
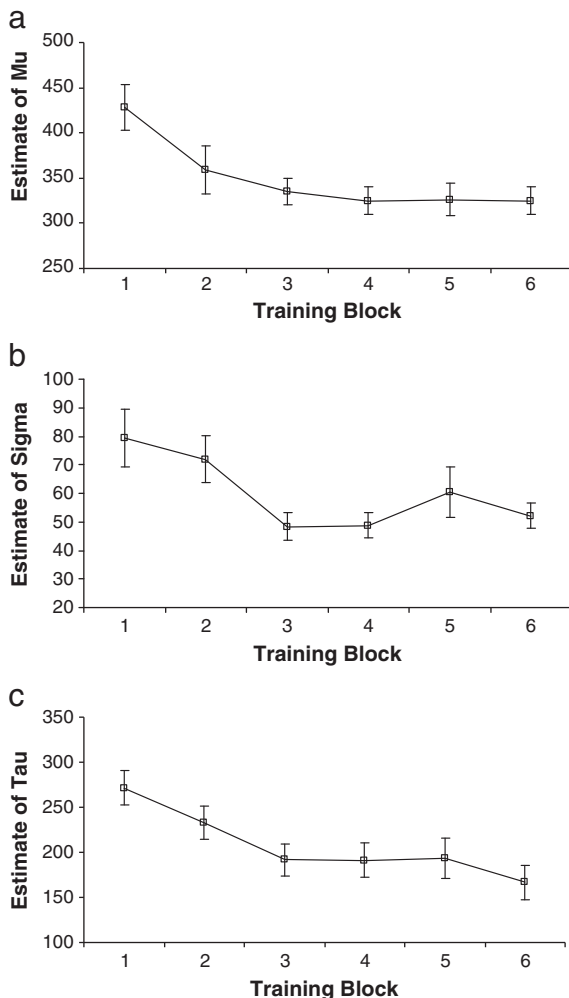


Fig. 5. Antisaccade proportion correct as a function of training block for Experiment 3. Error bars represent one standard error of the mean.

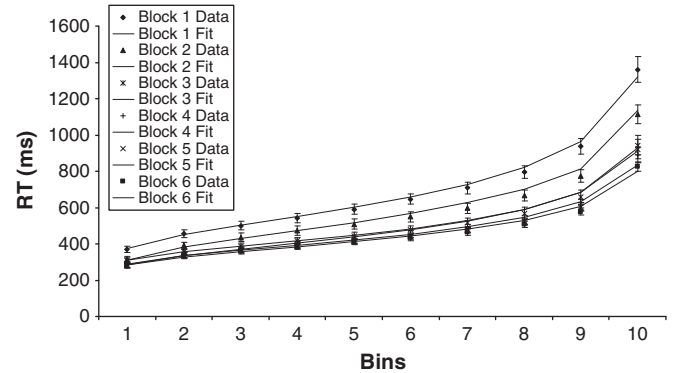


**Fig. 6.** Correct antisaccade response time as a function of training block for Experiment 3. Error bars represent one standard error of the mean.

Experiment 1. This was done because for both, participants completed 250 trials of either prosaccade or antisaccade. If training served to make antisaccades relatively automatic, then performance on prosaccade and antisaccade trials should be roughly equal. First, examining overall accuracy, the results suggested that there was no difference in accuracy for prosaccades and the last block of antisaccades,  $t(45) = .09$ ,  $p > .93$ , with accuracy at 92% correct in both cases. Examining correct RT, the results suggested that antisaccades on the last block of training were actually faster than prosaccades,  $t(45) = 2.22$ ,  $p < .05$ , with a mean antisaccade RT of



**Fig. 7.** (a) Estimate of  $\mu$  ( $\mu$ ) as a function of training block for Experiment 3. (b) Estimate of  $\sigma$  ( $\sigma$ ) as a function of training block for Experiment 3. (c) Estimate of  $\tau$  ( $\tau$ ) as a function of training block for Experiment 3. Error bars represent one standard error of the mean.



**Fig. 8.** Vincetile plots of Blocks 1–6 antisaccade distributions for Experiment 3. Data points and standard errors represent participants' mean vincentiles. Best fitting ex-Gaussian vincentiles are represented by lines.

468 ms and a mean prosaccade RT of 545 ms. Comparing ex-Gaussian estimates for the prosaccade trials and block six of the antisaccade trials, suggested that two saccade tasks had equivalent estimates of  $\mu$ ,  $t(45) = .15$ ,  $p > .88$ , with the estimate of  $\mu$  for prosaccades equaling 322 ms and the estimate of  $\mu$  for antisaccades equaling 325 ms. Similarly, the two saccade tasks had equal estimates of  $\sigma$ ,  $t(45) = .95$ ,  $p > .34$ , with the estimate of  $\sigma$  for prosaccades equaling 52 ms and the estimate of  $\sigma$  for antisaccades equaling 65 ms. For  $\tau$ , the results suggested that antisaccade estimates of  $\tau$  were actually slightly lower than prosaccade estimates of  $\tau$ ,  $t(45) = 1.80$ ,  $p < .08$ , with the estimate of  $\tau$  for prosaccades equaling 223 ms and the estimate of  $\tau$  for antisaccades equaling 166 ms. Similar to the prior experiments,  $\mu$  and  $\tau$  (for both prosaccade and antisaccade trials together) did not significantly correlate,  $r(47) = .04$ ,  $p > .75$ . Thus, training on antisaccade trials served to bring antisaccade performance in line with prosaccade performance such that antisaccades were just as accurate as prosaccades and antisaccades were actually slightly faster than prosaccades.

## 10. Discussion

The results from Experiment 3 suggested that giving participants extensive training on antisaccade trials served to drastically increase performance in terms of increasing accuracy and decreasing RT. This decrease in RT was reflected in changes in all three parameter estimates from the ex-Gaussian, suggesting that training served to not only shift the distribution downward, but also drastically shorten the tail and decrease the overall amount of variability. Furthermore, these changes in both the shift and the tail as a function of training block were equivalent. That is, the drop in  $\mu$  paralleled the drop in  $\tau$  such that both decreased at the same rate (i.e., the training block by parameter estimate interaction was not significant,  $F < 1$ ). Finally, comparing these increases in performance to standard performance on prosaccade trials suggested that by the end of training, antisaccades were just as accurate as prosaccades and were actually slightly faster. Note the finding that antisaccade accuracy increased to such a high level seems to argue against potential strategic explanations of the data. For instance, it would seem possible that over trials participants adopt a strategy of simply focusing on one side of the screen and then identifying the target on that side or rapidly directing attention to the opposite side. However, if participants were simply focusing on one side of the screen we would expect that accuracy on that side would be very high, but accuracy on the other side would be much lower. An examination of each individual's accuracy for both right and left responses suggested equivalent performance across all blocks of trials. Thus, it seems very unlikely that participants adopted such a strategy. Overall, these results are consistent with the notion that extensive training on a controlled task



allows for automaticity to build, thus making processes that were once largely controlled in nature, now relatively automatic. Thus, like similar findings for the Stroop task (e.g., [Cohen et al., 1990](#); [MacLeod, 1998](#)), extensive training on the antisaccade allowed for antisaccades to be performed in a relatively automatic fashion. Furthermore, this seems to occur for both the inhibition of the prosaccade and generation of antisaccade as well as goal maintenance processes that are thought to be attention control processes necessary for successful antisaccade performance. That is, as argued throughout, antisaccades likely require a mixture of control processes and practice allows for automaticity to build for each of these control processes in parallel.

## 11. General discussion

In three experiments we examined performance differences between prosaccade and antisaccade trials. In Experiment 1 we replicated prior work demonstrating that prosaccades are faster and more accurate than antisaccades. An examination of the full RT distributions suggested that the RT difference between prosaccades and antisaccades was due to both a shifting in the antisaccade distribution and a lengthening of the tail of the antisaccade distribution compared to the prosaccade distribution. Experiment 2 replicated and extended these findings by demonstrating that accuracy and RT for antisaccades varied as a function of foreperiod duration. Specifically, performance was poorest in terms of both accuracy and RT at the shortest foreperiod duration for antisaccades. There was no effect of foreperiod duration on prosaccades. Furthermore, an examination of the full RT distributions for the shortest and longest foreperiod durations on antisaccade trials suggested that the effect of foreperiod duration on RT was localized to the tail of the distribution. Thus, suggesting that the shift and the lengthening of the tail of the antisaccade distribution reflect two, possibly independent, processes. Experiment 3 demonstrated that it was possible to drastically increase performance (in terms of both accuracy and RT) on antisaccade trials after extensive training. The decrease in RT as a function of training was shown to be due to both a shift downward in the overall antisaccade distribution as well as shortening of the tail of the antisaccade distribution. Furthermore, extensive training on antisaccade trials was shown to bring antisaccade performance to the same levels as prosaccade performance in terms of equivalent accuracy and overall similar RT distributions.

Overall these results can be interpreted in terms of a two factor theory of attention control ([Engle & Kane, 2004](#); [Kane & Engle, 2003](#)) which suggests that both competition resolution and goal maintenance are needed for accurate performance on a variety of attention control tasks like Stroop and antisaccade. In particular, the current results suggest that the shift in the antisaccade distribution relative to the prosaccade distribution is due to the fact that each antisaccade trial requires some form of competition resolution in the form of inhibition of a pre-potent prosaccade and generation of a correct antisaccade. This competition resolution adds roughly a constant amount of time to antisaccades making them slower on average than prosaccades. In addition to constant competition resolution, antisaccades also require goal maintenance processes to actively maintain the task goal (e.g., [Godijn & Kramer, 2007](#); [Kane et al., 2001](#); [Nieuwenhuis et al., 2004](#); [Roberts et al., 1994](#); [Unsworth et al., 2004](#)). If the task goal is properly maintained, then a relatively fast correct antisaccade is generated. Transient failures of goal maintenance (i.e., goal-neglect) lead either to errors, or to very slow correct RTs as indexed by a lengthening of the tail of the antisaccade distribution. This factor requires preparatory attention control to activate and maintain the task goal prior to actual stimulus presentation. When there is adequate time to prepare for the upcoming trial, accuracy is increased and correct antisaccade RTs are relatively fast. If there is little time to prepare for the upcoming trial, either an error is made or a longer than normal RT is made.

Finally, because both competition resolution and goal maintenance are thought to be controlled processes, it is possible to make them relatively more automatic with enough training/practice. In this case, both processes seem to transition from relatively controlled processes to relatively automatic processes at the same rate. Overall, the current results suggest that correct antisaccades are likely due to a mixture of processes. Furthermore, the current results suggest that the cognitive operations used on antisaccade trials are conceptually similar to those used on other attention control tasks like the Stroop. In both cases, differences between trials results in changes in both  $\mu$  and  $\tau$  ([Heathcote et al., 1991](#); [Spieler et al., 1996](#)). Thus, antisaccade and Stroop tasks likely rely on both goal maintenance and competition resolution mechanisms ([Kane & Engle, 2003](#)). These findings are slightly different from other work which suggests that attention control mechanism in tasks like flankers and global/local primarily results in a difference in  $\mu$  (e.g., [Mewhort et al., 1992](#); [Spieler et al., 2000](#)). These differences could potentially come down to differences in task requirements where some tasks require the restraint of attention (Stroop and antisaccade) away from potent distractors whereas other tasks require the constraint of attention (flankers and global/local) to targets ([Poole & Kane, 2009](#)). Furthermore, these differences could also potentially come down to differences in the samples used. Specifically, we (e.g., [Unsworth et al., submitted for publication](#)) have recently found that the flanker cost was associated with not only a shift in the overall distribution, but also a lengthening of the tail but only in low working memory capacity individuals. In high working memory capacity individuals the flanker cost was associated with a shift only similar to [Spieler et al. \(2000\)](#). Thus, it may be possible to find differences in RT distributional characteristics as a function of the ability range of the sample used (see also [Tse, Balota, Duchek, Yap, & McCabe, 2010](#)). More work is needed to directly compare RT distributions in these tasks to determine potential similarities and differences.

It should be noted that throughout we have suggested that both inhibition of an incorrect prosaccade and generation of a correct antisaccade both fall under the general heading of competition resolution even though these two processes are also likely distinct. That is, prior work has suggested that when inhibition is theoretically equated between prosaccades and antisaccades the RT difference between prosaccades and antisaccades was markedly reduced ([Olk & Kingstone, 2003](#)). Furthermore, other work has suggested that endogenously generated prosaccades and standard antisaccade produce similar mean RTs (e.g., [Stuyven, Van der Goten, Vandierendonck, Claeys, & Crevits, 2000](#); [Unsworth et al., 2004](#)), thus suggesting that the generation of a correct saccade (rather than inhibition per se) partially accounts for the RT difference. Clearly, more work is needed to better determine whether the shift in the antisaccade distribution is due to the need to inhibit an incorrect prosaccade, generate a correct antisaccade, or both.

It should also be noted that we are not suggesting that  $\mu$  and  $\tau$  equal competition resolution and goal maintenance, respectively. Rather, we are suggesting that fitting an ex-Gaussian function to RT distributions is a parsimonious way of characterizing RT distributions and examining how experimental manipulations influence certain aspects of the distribution. As noted by [Spieler et al. \(2000\)](#) there is not a one-to-one mapping between the parameters of the ex-Gaussian and underlying cognitive processes. Clearly estimates of  $\tau$  from different tasks (i.e., free recall and antisaccade) do not estimate the same cognitive processes. Thus, in the current study we have simply relied on the ex-Gaussian as means of examining RT distributions and RT differences between prosaccade and antisaccade tasks in hopes of elucidating the underlying processes of interest. The ex-Gaussian was chosen because a number of prior studies have used the ex-Gaussian to examine attention control in other tasks such as the Stroop, flankers, and global/local tasks and we wanted to specifically make contact with that prior work (see also [Balota et al., 2008](#); [Yap et al.,](#)

2008). Clearly other functions such as the Wald, ex-Wald, and Weibull could have been used to examine the shape of the RT distributions and to determine differences between prosaccade and antisaccade trials. Indeed a quick examination of our data suggested that both the Wald and Weibull provide reasonable fits (Cousineau, Brown, & Heathcote, 2004) to the data similar to those obtained with the ex-Gaussian. Future work is needed to determine which of these functions might be the most appropriate for examining RT distributions in various cognitive tasks.

Another potential concern with the current results is that we relied on button press responses to examine differences between prosaccade and antisaccade tasks rather than examining actual oculomotor responses as measured with an eyetracker. Clearly, it would have been optimal to rely on an eyetracker and directly record eye movements, but we feel that our overall results obtained with button press responses should provide the same information as those obtained with an eye-tracker. To directly examine this we reanalyzed data from Unsworth et al. (2004) that used eyetracking to examine prosaccade and antisaccade tasks. In this study participants performed prosaccade and antisaccade trials with oculomotor responses only (i.e., there were no button press responses and no target identification task). Similar to the current experiments we fit each individual's raw RT distribution using QMLE (Brown & Heathcote, 2003) and all fits successfully converged within 250 iterations. Consistent with the current results there were differences between prosaccade and antisaccade trials on all three ex-Gaussian parameters (all  $t$ 's > 2.22, all  $p$ 's < .05). Furthermore, it should be noted that the results from Unsworth et al. are for correct trials only and do not include trials where a reflexive saccade was made prior to a corrective antisaccade. The similarity in results across button-press and oculomotor response tasks suggest that the current results are not unduly biased by the requirement to press a button or by misclassifying initially reflexive saccades as correct antisaccades. Thus, the overall conclusions from the current study seem to hold when examining RT distributions based on button press responses as well as oculomotor responses. Of course future work should examine this issue in more detail to ensure that these distributions are similar across a variety of experimental manipulations suggesting that both competition resolution and goal maintenance are important determinants of antisaccade performance.

The notion that antisaccade performance is driven by both competition resolution and goal maintenance is also supported by a number of recent neuroimaging studies. In particular, a number of studies have found that several areas are more active for antisaccades than prosaccades during the preparatory interval (e.g., Brown, Vilis, & Everling, 2007; Curtis & D'Esposito, 2003; Ford, Goltz, Brown, & Everling, 2005). Furthermore, level of preparatory activation (especially in the dorsolateral prefrontal cortex and anterior cingulate cortex) is directly related to whether or not a correct or incorrect antisaccade is generated (e.g., Brown et al., 2007; Curtis & D'Esposito, 2003; Ford et al., 2005). If the goal is not properly maintained then an erroneous prosaccade will be executed. Other recent work has found that several areas are active during the response generation period that are classically associated with motor control over saccades including frontal eye field, supplementary eye field, and intraparietal sulcus. Importantly, a recent study by Brown et al. (2007) found that dorsolateral prefrontal cortex and anterior cingulate activity was greater in antisaccades than prosaccades during the preparatory interval but not during actual response generation. This is consistent with the notion that these areas are important for goal maintenance processes, but that other areas are important for the generation of a correct response.

The current results and interpretation suggest a promising avenue of future research in which attention control is examined in the antisaccade task via RT distribution analyses. In particular, it should be possible to examine how different manipulations affect either the

shift in the distributions or affect a change in the tail of the distribution based on whether competition resolution or goal maintenance is theoretically being affected. Furthermore, future work should examine the extent to which inhibition of a prosaccade and the generation of a correct antisaccade reflect different processes that combine to produce an overall shift in the distribution. Finally, these distributional analyses should be combined with neuroimaging analyses to see if activation in certain control areas or deactivation in certain default network areas are related to a shift in the overall distribution or to a lengthening of the tail. Based on the preceding review, we would suggest that such activation would be related to a lengthening of the tail but not related to the shift in the distribution. Analyses of the full distribution of responses rather than just mean RT should help elucidate the processes necessary for accurate performance in goal driven situations.

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