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The unidirectional prosaccade switch-cost: Correct and error antisaccades differentially influence the planning times for subsequent prosaccades

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ABSTRACT

Antisaccades produce longer reaction times (RT) than their prosaccade counterparts and this latency increase has been linked to an oculomotor 'pre-setting' that prevents the evocation of a stimulus-driven prosaccade. Moreover, a consequence of oculomotor pre-setting is a lengthening of the RTs associated with a subsequent prosaccade. The goal of the present study was to determine whether the constituent elements associated with *planning* a correct antisaccade (i.e., response suppression and vector inversion) imparts a residual delay that inhibits the *planning* of a subsequent prosaccade. To that end, participants alternated between pro- and antisaccades in a pseudo-randomized task-switching schedule (e.g., AABBAAB. . .) and responses were cued via a paradigm that was designed to evoke frequent error antisaccades (i.e., a saccade initially, and incorrectly, planned to the target stimulus). Results showed that RTs for correct antisaccades were longer than error antisaccades and that prosaccades preceded by the former, but not the latter, trial-type were associated with a reliable increase in RT (i.e., prosaccade switch-cost). In other words, error antisaccades were associated with a failure to withhold a stimulus-driven prosaccade and did not delay the planning of a subsequent prosaccade. Based on these findings we propose that the prosaccade switch-cost is not related to an explicit awareness of task goals; rather, our results are consistent with the assertion that a consequence of response suppression and vector inversion is a residual inhibition of stimulus-driven oculomotor planning networks.

1. Introduction

Prosaccades are rapid eye movements that are intended to bring a target of interest into central vision. Notably, the spatial coupling between stimulus and response for prosaccades allows for their mediation via direct retinotopically organized motor maps in the superior colliculus (for review see Wurtz & Albano, 1980). It is, however, important to recognize that a stimulus need not reflexively capture one's gaze; rather, a prosaccade can be suppressed in favor of a volitional saccade to another area of interest. Indeed, volitional saccades represent an important area of inquiry because they provide a basis for determining how top-down control influences the oculomotor system's ability to efficiently and effectively execute a response. One paradigm that has been extensively used to examine the issue of top-down oculomotor control is the antisaccade task. Indeed, the most frequently examined antisaccade task involves a variant of the classic saccade paradigm whereby a participant is instructed to saccade mirror-symmetrical (i.e., 180° spatial transformation) to the location of a *single* and *exogenously* presented target. Results have shown that antisaccades produce longer reactions times (RTs) (Everling, Dorris, & Munoz, 1998; Hallett, 1978), increased directional errors (Fischer & Weber, 1992; Forbes & Klein, 1996) and less accurate and more variable endpoints (Hallett, 1978; Heath et al., 2010) than their prosaccade counterparts. Furthermore, electrophysiological and neuroimaging evidence from humans and non-human primates has linked the aforementioned behavioral 'costs' to a two-component process requiring: (1) the inhibition of a stimulus-driven prosaccade (i.e., response suppression), and (2) the *visual* remapping of target properties (i.e., vector inversion) (for review see Munoz & Everling, 2004).

The preparatory phase of the antisaccade task has been related to an increased level of activation within the "classical saccade networks" (i.e., frontal eye field, supplementary eye field, and lateral intraparietal area) (Brown, Vilis, & Everling, 2007; DeSouza, Menon, & Everling, 2003; Ford et al., 2005) as well as an increase in the activation of collicular fixation neurons and a decrease in

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the activation of collicular build-up neurons (Everling et al., 1999). In particular, Everling and colleagues (Brown, Vilis, & Everling, 2007; Everling & DeSouza, 2005; see also Schlag-Rey et al., 1997) proposed that the modulation of oculomotor networks during the preparatory period of the antisaccade is related to a *pre-setting* that inhibits the evocation of a stimulus-driven prosaccade (i.e., the visual grasp reflex: Pierrot-Deseilligny et al., 1995) and provides sufficient time to implement the constituent elements of the antisaccade task (i.e., response suppression and vector inversion).

A corollary prediction drawn from the pre-setting of antisaccades is a lingering inhibition of oculomotor planning mechanisms. Indeed, Barton and Manoach and their co-workers used a *cued-saccade* paradigm to demonstrate the consequence of switching between task-types in blocked (i.e., AABB) and randomized task-switching schedules (Barton et al., 2002; Barton, Goff, & Manoach, 2006; Cherkasova et al., 2002; Manoach et al., 2002; Manoach et al., 2007; see also Barton et al., 2006). In particular, their work provided participants with two continuously visible targets located left and right of a central fixation stimulus prior to response cuing. Notably, following a preview phase one of the targets was cued via a surrounding annulus. A priori participants were instructed to saccade to the cued (i.e., cued-prosaccade) or un-cued (i.e., cued-antisaccade) target. Results showed a reliable 'switch-cost' for prosaccades; that is, a prosaccade preceded by an antisaccade (i.e., prosaccade task-switch trial) elicited longer RTs than prosaccades preceded by their same task counterparts (i.e., prosaccade task-repetition trial). In addition, a 'paradoxical switch-benefit' was associated with antisaccades such that task-switch antisaccades (i.e., an antisaccade completed after a prosaccade) exhibited shorter RTs than their task-repetition counterparts (i.e., the second of two consecutively completed antisaccades). Further, fMRI work by Manoach et al. (2007) showed that the preparatory interval of pro- and antisaccades completed after an antisaccade were associated with reduced activity in bilateral frontal eye fields and the right supplemental eye field. Given these findings, Barton and Manoach and their co-workers proposed that the completion of an antisaccade results in a lingering inhibition of oculomotor networks that delays the planning of *all* subsequent saccades.

The work of Barton and Manoach and their group provides a direct demonstration that alternating between task-types can influence oculomotor planning times. Notably, however, an important consideration is that the cued-antisaccade paradigm used in their work may not require vector inversion (see also Edelman, Valenzuela, & Barton, 2006). Recall that in their paradigm both pro- and antisaccade target locations were visible prior to, and throughout a response. Thus, their antisaccade task may not require the visual remapping of the target's spatial properties (i.e., vector inversion); rather, the un-cued target may serve as the location for planning a veridical (antisaccade) movement endpoint.[1] As well, other work involving the continued presence of a target during response execution has revealed discrepant pro- and antisaccade RT switch-costs (Reuter et al., 2006; Olk & Jin, 2011). In addressing the aforementioned issues, Weiler and Heath (2012a, 2012b) examined oculomotor task-switching via a classic saccade paradigm wherein participants were instructed to pro- or antisaccade to a single, and briefly (i.e., 50 ms) presented target in blocked (e.g., AABB: Weiler & Heath, 2012a, 2012b) and pseudo-randomized (Weiler & Heath, 2012b) task-switching schedules. Indeed, in such a paradigm both pro- and antisaccade planning and execution occurs without the continued presence of a veridical target, and the antisaccade task requires the obligatory remapping of the target's spatial location in mirror-symmetrical space. In line with Barton and Manoach's group, task-switch prosaccades elicited longer RTs than their task-repetition counterparts. In contrast, task-switch and task-repetition antisaccades exhibited comparable RTs: a finding that differs from the paradoxical switch-benefit observed by Barton and Manoach's group. In support of our results, Chan and DeSouza (2013) recently found that task-switching RT effects in the classic saccade paradigm were restricted to task-switch prosaccades. As such, results from the classic saccade paradigm indicate that the completion of an antisaccade selectively delays the planning of a to-be-completed prosaccade (i.e., the unidirectional prosaccade switch-cost). Based on this result, Weiler and Heath proposed an *oculomotor inhibition hypothesis* wherein the constituent elements of the antisaccade task (i.e., response suppression and vector inversion) imparts a residual inhibition that delays the planning mechanisms supporting stimulus-driven prosaccades. Indeed, the hypothesis contends that both response suppression and vector inversion contribute to the residual inhibition because each process requires the top-down and cognitive control of action (Rossetti et al., 2005). Notably, the hypothesis is drawn from the previously mentioned neuroimaging and electrophysiological evidence showing that an oculomotor pre-setting characterizes antisaccade performance (e.g., Brown, Vilis, & Everling, 2007). As well, the unidirectional nature of the hypothesis is derived from behavioral evidence showing that the active inhibition of a standard or familiar task (e.g., prosaccade) persists inertially following the planning of a non-standard (or unfamiliar) task, whereas no such persistence exists following the planning of a standard task (i.e., task-set inertia: see Allport, Styles, & Hsieh, 1994; see also Wylie & Allport, 2000).

The foundation for the oculomotor inhibition hypothesis is that the *planning* of an antisaccade (including response suppression and vector inversion) delays the *planning* of a subsequent prosaccade. Thus, it is proposed that the unidirectional prosaccade switch-cost should selectively manifest following the completion of a correct (i.e., a response planned and executed mirror-symmetrical to the target) but not an error antisaccade (i.e., a response planned and initially executed to the target and not its mirror-symmetrical location). Indeed, the basis for this assertion is that the pre-setting associated with the planning of a correct antisaccade produces a level of residual inhibition that delays the planning of a to-be-completed prosaccade. In contrast, an error antisaccade entails a reduced, or incomplete, level of pre-setting and therefore results in the evocation of a prosaccade; that is, the participant fails to suppress a stimulus-driven response (see Everling, Dorris, & Munoz, 1998). As a consequence, it is predicted that the planning for a subsequent prosaccade would not be subjected to a residual level of oculomotor inhibition.

The present investigation used the classic saccade paradigm to examine the proposal that the unidirectional prosaccade switch-cost manifests following a correct, but not an error, antisaccade. Of course, in accomplishing our objective we recognized that it was important to design a task-switching schedule and target presentation paradigm that elicited a sufficient corpus of error antisaccades. Thus, we sought to induce frequent error antisaccades by employing a pseudo-randomized pro- and antisaccade task-switching schedule wherein target stimuli were presented in a gap paradigm (i.e., fixation cross removed prior to target onset) paired with a task-irrelevant tone. Notably, increased antisaccade errors have been shown to occur when performed in an unpredictable as opposed to blocked presentation schedule (Olk & Kingstone, 2003), and saccade countermanding errors have been shown to increase under gap and task-irrelevant tone paradigms (for no-gap vs. gap paradigm see Fig. 1 of Munoz & Everling, 2004; for task-irrelevant tone paradigm see Colonius & Arndt, 2001; Corneil & Munoz, 1996). In terms of research predictions, if the oculomotor

[1] An anonymous reviewer indicated that antisaccades performed in a cued saccade-paradigm may require vector inversion because participants are responding to the position of the annulus and not the "markers" that serve as the location for the saccade endpoint. Notably, evaluation of this issue awaits a directed study contrasting task-switch costs in cued- and classic-saccade paradigms.

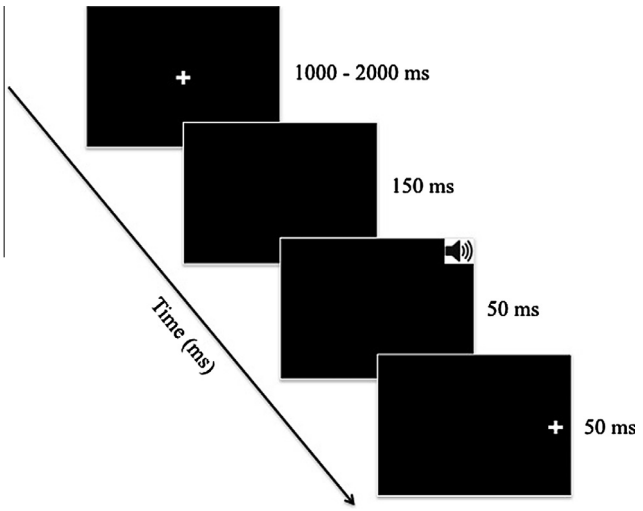

Fig. 1. Timeline of visual and auditory events. A green (i.e., prosaccade) or a red (i.e., antisaccade) fixation cross was presented for a randomized foreperiod (1000–2000 ms). Following the foreperiod, the fixation cross was extinguished (so-called gap paradigm) and 150 ms into the gap interval a 50 ms task-irrelevant tone was presented. In combination with tone offset one of four target stimuli was presented. The onset of a target served as the imperative to complete the instructed task (i.e., pro- or antisaccade).

inhibition hypothesis is correct then the unidirectional prosaccade switch-cost should be observed following the completion of correct, but not error, antisaccades. Certainly, such a finding would suggest that the planning processes associated with an antisaccade (i.e., response suppression and vector inversion) contribute to the residual inhibition of the planning processes supporting stimulus-driven prosaccades. In turn, if the switch-cost is independent of the nature of the preceding antisaccade trial (i.e., correct vs. error) then results would suggest that the residual inhibition may relate to factors such as explicit awareness of task goals (Day & Lyon, 2000). In other words, results would indicate that response suppression and vector inversion are not directly tied to the unidirectional prosaccade switch-cost. As a final element, we note that the unidirectional prosaccade switch-cost is predicted to selectively manifest during response planning. Thus, task-switch and task-repetition prosaccades are predicted to elicit comparable movement times and endpoint accuracy.

2. Methods

2.1. Participants

Twenty-six individuals (sixteen female and ten male: age range 18–25 years) from The University of Western Ontario community volunteered for this experiment. All participants declared being right-hand dominant and had normal or corrected-to-normal vision. Participants signed consent forms approved by the Office of Research Ethics, The University of Western Ontario, and this work was conducted according to the Declaration of Helsinki.

2.2. Apparatus and procedure

Participants were seated at a table (775 mm in height) with their head placed in a head-chin rest for the duration of the experiment. Visual stimuli were presented on a 30-in. LCD monitor (60 Hz, 8 ms response rate, 1280 × 960 pixels, Dell 3007WFP, Round Rock, TX, USA) placed 550 mm from the participant and centered on their midline. The gaze location of participants' left eye was obtained using a video-based and chin-mounted eye-tracking system (Eye-Trac 6: Applied Science Laboratories, Bedford, MA, USA) sampling at 360 Hz. Prior to data collection a nine-point calibration of participant's viewing space was performed and confirmed via an immediate follow-up calibration. Two additional monitors that were visible only to the experimenter provided: (1) real-time point of gaze information, (2) visual depiction of trial-to-trial saccade kinematics (i.e., displacement, velocity), and (3) information on the accuracy of the eye tracking system (i.e., to allow for drift correction or re-calibration when necessary). All computer events were controlled via MATLAB (ver 7.8.0, The MathWorks, Inc., Natick, MA, USA) and the Psychophysics Toolbox extension (ver 3.0; see Brainard, 1997). The lights in the experimental suite were extinguished during data collection.

Visual stimuli were presented against a high contrast black background and included a green and a red fixation cross (1.0°) centered horizontally on the monitor and at the eye level of the participant. Yellow crosses (1.0°) located 10.8° (proximal) and 13.8° (distal) left and right of the fixation cross served as targets. Each trial commenced with the presentation of either the green or the red fixation cross and participants were instructed to direct their gaze to its location. The color of the fixation cross signified the nature of the upcoming response: the green fixation cross instructed participants to saccade to the veridical target location (i.e., prosaccade), whereas the red fixation cross denoted a saccade to the target's mirror-symmetrical location (i.e., antisaccade). After a stable fixation was maintained (±1.5° for 420 ms), a randomized foreperiod (1000–2000 ms) was introduced during which time the fixation cross remained visible. Recall that the goal of this study was to compare the latencies of prosaccades preceded by a correct antisaccade to prosaccades preceded by an error antisaccade (i.e., a response initially directed to the location of the veridical target instead of the instructed mirror-symmetrical location). As such, we employed a gap paradigm in conjunction with a task-irrelevant tone to increase the frequency of antisaccade directional errors. Specifically, and as shown in Fig. 1, the fixation cross was extinguished at the end of the randomized foreperiod and a tone (64 dB, 1900 Hz) presented for a duration of 50 ms was provided 150 ms following fixation cross extinction. Concurrent with tone offset, one of the target stimuli was briefly presented (50 ms) and participants were specifically instructed to complete their response "as quickly and as accurately possible" in response to target onset. Thus, the overall duration of the gap period was 200 ms. Notably, we did not provide participants with any instruction related to the tone. Rather, inclusion of the tone was based on evidence showing that close temporal proximity between a task-irrelevant tone and a task-relevant visual cue shortens saccade RTs and increases the probability of failing to withhold a stimulus-driven saccade (Colonius & Arndt, 2001; see also Corneil & Munoz, 1996).

Participants completed four trial blocks (108 trials/block) involving pro- and antisaccade trial sequences. Trial blocks were completed across two sessions (2 blocks/session) separated by 24 h, and the different sessions were used because our previous work has shown that participants experience a degree of eye strain/fatigue following 250 trials (Weiler et al., 2011). Each block entailed alternating pro- and antisaccade trials across each of the visual space (i.e., left, right) and target eccentricity (i.e., proximal, distal) combinations in a pseudo-randomized task-switching schedule (e.g., AABBAAB...) that could not be predicted by participants. In particular, each block contained 29 sequences of which 21 were AABB sequences and 8 were AAB sequences. The pseudo-randomization of the sequences was structured such that AAB sequences could not occur consecutively. Thus, responses were classified into task-switch (i.e., prosaccade preceded by an antisaccade, or vice versa) and task-repetition (i.e., pro- or antisaccade preceded by the same task) pro- and antisaccade trials. The unpredictable task-switching schedule precluded an equal number of pro- and

antisaccades trials and resulted in participants completing 32 more trials of the former task. As well, the unpredictable task-switching schedule was employed as an additional means to increase the probability of error antisaccades (cf. Olk & Kingstone, 2003). Each block commenced with a prosaccade and because such a response was neither a task-switch nor a task-repetition trial it was excluded from subsequent analyses.

2.3. Data analyses and dependent variables

Displacement data were filtered offline using a dual-pass Butterworth filter employing a low-pass cut-off frequency of 15 Hz. Filtered displacement data were used to compute instantaneous velocities via a five-point central finite difference algorithm. Acceleration data were similarly obtained from the velocity profiles. Saccade onset was determined on the basis of velocity and acceleration values that exceeded 30°/s and 8000°/s^2, respectively. Saccade offset was marked when velocity fell below 30°/s for 15 consecutive frames (i.e., 42 ms). The primary dependent variable was reaction time (RT: time from target presentation to saccade onset). Where appropriate we also report results for the percentage of directional errors as well as movement time (MT: time from movement onset to movement offset), saccade amplitude and associated variability in the primary (i.e., horizontal) movement direction. Only significant effects and interactions are reported below.

3. Results

3.1. Correct antisaccades produce a prosaccade RT switch-cost

Previous work has shown that prosaccade RTs elicit a unidirectional switch-cost (Weiler & Heath, 2012a, 2012b). Here we sought to replicate the aforementioned finding and thus submitted RT data to 2 (task: pro-, antisaccade) by 2 (task transition: task-switch, task-repetition) repeated measures ANOVA.[2] Error pro- and antisaccade trials were excluded from this analysis, as were task-switch pro- and antisaccades that were preceded by an antisaccade error. Table 1 presents the number of trials removed for the aforementioned criteria and we further note that 5.5% of trials were removed due to: (1) Signal loss (e.g., blinking), (2) A RT greater than two standard deviations above the mean group performance (i.e., RT > 660 ms), and (3) An anticipatory response (i.e., RT < 85 ms) (Wenban-Smith & Findlay, 1991). Results yielded main effects for task $F(1,25) = 98.08$, $p < 0.001$, task transition, $F(1,25) = 26.31$, $p < 0.001$, and their interaction, $F(1,25) = 13.24$, $p < 0.01$. As shown in Fig. 2, task-switch prosaccades (224 ms, $SD = 46$) produced longer RTs than their task-repetition counterparts (206 ms, $SD = 44$) ($t(25) = 7.55$, $p < 0.001$), whereas RTs for task-switch (276 ms, $SD = 44$) and task-repetition (272 ms, $SD = 42$) antisaccades did not differ ($t(25) < 1$, p = n.s.).

In addition to RT, we used the ANOVA model specified above to examine the percentage of directional errors, saccade MT, amplitude and amplitude variability. Results for all variables yielded main effects for task, $Fs(1,25) = 32.67$, 15.40, 8.94, and 39.69, respectively for the percentage of directional errors, MT, amplitude and amplitude variability, $ps < 0.01$. Prosaccades produced fewer directional errors (3.0%, $SD = 3$), shorter MTs (62 ms, $SD = 8$) with greater (12.9°, $SD = 1.0$) and less variable amplitudes (2.4°, $SD = 0.4$) than their antisaccade counterparts (directional errors:

Table 1
Participant-by-participant mean reaction times (ms) and standard deviations for the prosaccade categories used here: (1) prosaccade task-switch trials preceded by a correct antisaccade (PrS-C), (2) prosaccade task-switch trials preceded by an error antisaccade (PrS-E), and (3) prosaccade task-repetition trials (PrR). In addition, a frequency (*f*) column presents the participant-specific frequency of trials in each of the categories.

Participant	*f*	PrS-C	*f*	PrS-E	*f*	PrR
1	78	254 (79)	11	288 (101)	100	242 (84)
2	84	188 (47)	18	176 (30)	110	188 (52)
3	79	189 (75)	26	190 (73)	107	187 (66)
4	97	185 (49)	2	188 (2)	103	171 (41)
5	97	235 (84)	10	191 (78)	107	198 (64)
6	91	242 (77)	8	214 (112)	105	220 (68)
7	84	359 (75)	3	321 (50)	90	354 (68)
8	78	190 (77)	24	173 (57)	106	174 (46)
9	93	296 (66)	5	207 (39)	103	258 (56)
10	85	168 (56)	20	169 (78)	106	167 (50)
11	93	203 (85)	6	261 (133)	104	187 (70)
12	95	232 (72)	7	236 (56)	104	215 (64)
13	104	194 (71)	4	140 (20)	113	178 (53)
14	67	248 (96)	14	222 (88)	102	197 (79)
15	97	208 (65)	13	179 (39)	108	192 (50)
16	75	310 (79)	15	301 (84)	87	289 (70)
17	63	155 (56)	38	150 (56)	104	151 (51)
18	93	193 (62)	7	154 (45)	102	180 (47)
19	102	222 (58)	5	203 (69)	107	205 (48)
20	76	186 (77)	23	172 (63)	98	172 (59)
21	93	261 (86)	11	207 (58)	106	241 (85)
22	75	249 (56)	13	264 (49)	103	240 (42)
23	83	234 (87)	18	189 (76)	103	201 (74)
24	83	216 (73)	14	217 (71)	106	193 (50)
25	88	195 (67)	19	180 (56)	114	178 (46)
26	94	201 (67)	16	181 (59)	110	177 (55)
Grand mean	2246	224 (46)	350	207 (47)	2708	206 (44)

Note: Participant-specific standard deviations are reported in combination with the grand mean.

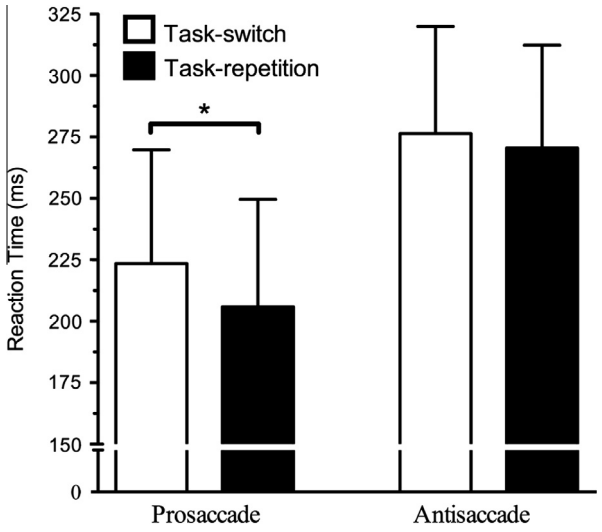

Fig. 2. Mean reaction time (ms) for task-switch and task-repetition pro- and antisaccade trials. As noted in the Results, the data presented here involve only correct pro- and antisaccades and only prosaccade task-switch trials preceded by a correct antisaccade. Error bars represent one between-participant standard deviation and the asterisk denotes the location of the reliable between-condition difference.

[2] Task-switch and task-repetition pro- and antisaccades completed in the direction opposite a previous response have been shown to elicit RTs that are shorter than when completed in the same direction as the previous response (Barton, Goff, & Manoach, 2006a; see also Olk & Jin, 2011). Thus, our initial ANOVA model included response direction (direction-repeat, direction-switch) as a factor. Inclusion of this factor produced a task transition by response direction interaction, $F(1,25) = 45.78$, $p < 0.001$. Task-switch trials associated with a direction-repeat produced shorter RTs than their task-repetition counterparts ($p < 0.05$). In contrast, task-switch and task-repetition trials involving a direction-switch did not differ (p = n.s.). Importantly, response direction did not interact with task or produce a higher-order interaction involving task and task transition. As such, response direction was included as a collapsed factor in the final ANOVA model.

12.8%, *SD* = 7.5; MT: 66 ms, *SD* = 12; amplitude = 11.9°, *SD* = 1.9; amplitude variability: 3.1°, *SD* = 0.7). As well, task-repetition trials yielded fewer directional errors (6.0%, *SD* = 8.0) than their task-switch (9.1%, *SD* = 8.4) counterparts, $F(1,25) = 16.52$, $p < 0.001$.

3.2. Error antisaccades do not produce a prosaccade switch-cost

The above analysis demonstrated that planning times for prosaccades are lengthened when they are preceded by a correct antisaccade: a result we have previously attributed to a residual inhibition of stimulus-driven oculomotor networks (i.e., the oculomotor inhibition hypothesis). In the following analysis, we sought to determine whether the aforementioned switch-cost is dependent or independent of the nature of the preceding antisaccade trial. As such, we included the same prosaccade task-switch (i.e., PrS-C) and task-repetition (i.e., PrR) trials as used in the above analysis (i.e., those completed without a preceding error antisaccade), and also included prosaccade task-switch trials preceded by an error antisaccade (i.e., PrS-E). Table 1 presents the frequency of trials in each category and it should be noted that PrR trials were not dichotomized due to the paucity of errors on the preceding trial (i.e., less than 1% of trials). Subsequently, RTs for the different prosaccade categories were submitted to a one-way ANOVA and results yielded a significant effect, $F(2,50) = 8.94$, $p < 0.001$: PrS-C trials (224 ms, *SD* = 46) produced longer RTs than either PrS-E trials (207 ms, *SD* = 47) ($t(25) = 2.91$, $p < 0.01$) or PrR trials (206 ms, *SD* = 44) trials ($t(25) = 7.55$, $p < 0.001$). In turn, RTs for PrS-E and PrR trials did not differ ($t(25) = 0.15$, p = n.s.) (Fig. 3).

As noted in Table 1, there were more PrS-C trials than PrS-E trials. Thus, it is possible that the between-category difference in RTs may reflect a between-category difference in the dispersion of RT values within- and between-participants. To address this issue, we contrasted the within-participant standard deviations of RTs across prosaccade task-switch trials completed following error (i.e., PrS-E) and correct (i.e., PrS-C) antisaccades. As shown in Table 1, the results of this analysis demonstrated that RT variability for PrS-E (63 ms) and PrS-C (71 ms) trials did not differ ($t(25) = 1.70$, p = n.s.).

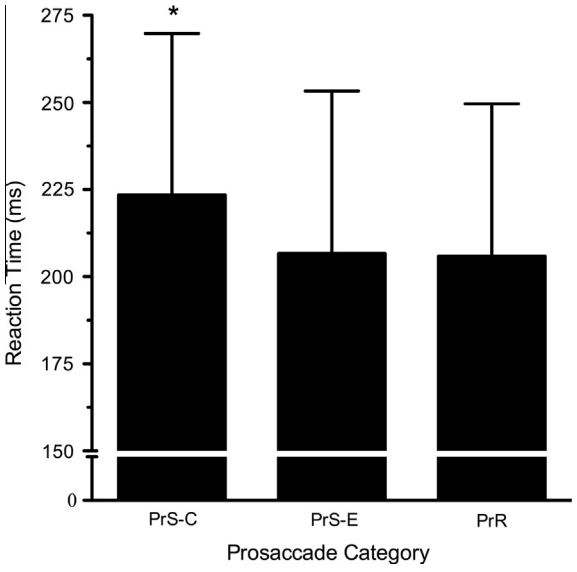

Fig. 3. Mean reaction time (ms) for the prosaccade categories used here: (1) prosaccade task-switch trials preceded by a correct antisaccade (PrS-C), (2) prosaccade task-switch trials preceded by an error antisaccade (PrS-E), and (3) prosaccade task-repetition trials (PrR). Error bars represent one between-participant standard deviation and the asterisk denotes the location of the reliable between-condition difference. Participant-by-participant data for this figure are shown in Table 1.

3.3. Error antisaccade trials and prosaccade task-repetition trials exhibit comparable reaction times

In the following analyses we sought to examine the behavioral properties of correct and error antisaccade trials. First, results showed that RTs for error antisaccades (204 ms, *SD* = 56) were shorter than their correct counterparts (273 ms, *SD* = 42; $t(25) = 8.39$, $p < 0.001$). Second, RTs in the former condition did not reliably differ from PrR trials (206 ms, *SD* = 44) ($t(25) = 0.35$, p = n.s.). In other words, error antisaccades were associated with planning times commensurate with stimulus-driven prosaccades.

3.4. Correct and error antisaccades produce comparable movement endpoints

Fig. 4 provides an exemplar trajectory for a PrR trial as well as trajectories for correct and error antisaccades. The exemplar for the error antisaccade is particularly salient because 96% of the trials in this category involved a rapid (137 ms, *SD* = 37) trajectory reversal resulting in a saccade endpoint in mirror-symmetrical space. Moreover, the endpoints for correct and error antisaccades did not differ, $F(1,25) = 1.25$, p = n.s.

4. Discussion

Our first analysis contrasted the RTs for correct pro- and antisaccade task-switch and task-repetition trials. The results of this analysis yielded two salient findings. First, antisaccade RTs were longer than prosaccades, and this difference was independent of task-switch and task-repetition trials. Of course, such a finding is consistent with an extensive literature and is taken to evince that the top-down demands of suppressing a stimulus-driven prosaccade (i.e., response suppression) and inverting a target's coordinates in mirror-symmetrical space (i.e., vector inversion) are measureable processes (for review see Munoz & Everling, 2004). Second, RTs for task-switch prosaccades were longer than their task-repetition counterparts, whereas antisaccade RTs were comparable across task-switch and task-repetition trials. Moreover, MT, saccade amplitude and amplitude variability did not differ across prosaccade task-switch and task-repetition trials; that is, task-switching effects were restricted to response planning. As such, the present results are in line with work showing that the

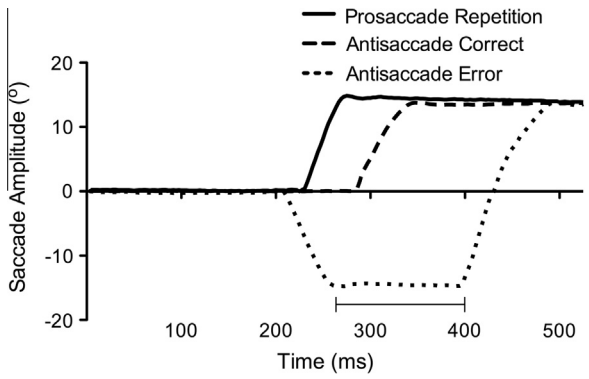

Fig. 4. Exemplar trajectories for a prosaccade task-repetition trial as well as a correct and an error antisaccade trial. The error antisaccade trial exhibits an initial planning error followed by a short-latency trajectory reversal enabling a mirror-symmetrical endpoint. In line with previous work (i.e., Evdokimidis, Tsekou, & Smyrnis, 2006), the capped horizontal line associated with the error antisaccade represents the measured latency for the trajectory reversal. Last, the figure demonstrates that the error antisaccade and task-repetition prosaccade trials produced comparable RTs that were shorter than the correct antisaccade trial.

classic saccade paradigm elicits a unidirectional prosaccade switch-cost (Chan & DeSouza, 2013; Weiler & Heath, 2012a, 2012b), and is taken as indirect support for the oculomotor inhibition hypothesis' assertion that response suppression and vector inversion engender a residual delay in the planning mechanisms supporting stimulus-driven prosaccades.

The foundation of the oculomotor inhibition hypothesis is that the *planning* processes associated with an antisaccade impart a residual inhibition on the *planning* processes mediating a to-be-completed prosaccade. That is, the hypothesis asserts that the top-down and executive demands of response suppression and vector inversion engender a persistent inhibition of the oculomotor networks that mediate the planning of stimulus-driven prosaccades. In support of this view, Schall and colleagues have shown that stimulus-driven prosaccades completed after a successful stop-signal saccade are associated with a change in the time when movement-related neurons in the superior colliculus and frontal eye fields first begins to accumulate (Pouget et al., 2011). Moreover, the source of the aforementioned delay has been linked to an extended 'holding period' by which top-down driven signals from frontal brain regions (i.e., dorsolateral prefrontal cortex, supplementary eye field, and anterior cingulate cortex) modulate the activity of collicular fixation and movement neurons (Lo et al., 2009; see also Stuphorn & Schall, 2006). As well, the top-down and cognitive demands of vector inversion have been tied to the delay in antisaccade planning times (Heath et al., 2011). Thus, a direct and testable prediction from the oculomotor inhibition hypothesis is that a lengthening of prosaccade RT should be observed following correct, but not error, antisaccades; after all, only the former trial-type entails response suppression and vector inversion. In order to test that prediction, prosaccades were categorized as: (1) task-switch trials preceded by a correct antisaccade (PrS-C), (2) task-switch trials preceded by an error antisaccade (PrS-E), and (3) task-repetition trials (PrR). Results showed that PrS-C trials yielded longer RTs than PrS-E or PrR trials, and that the latter two trial-types did not differ. Importantly, that PrS-C (but not PrS-E) trials exhibited a selective lengthening of RT relative to PrR trials indicates that the appropriate planning of an antisaccade (including response suppression and vector inversion) is necessary to delay the planning of a subsequent prosaccade. Indeed, that erroneous antisaccade planning (i.e., PrS-E trials) resulted in a null prosaccade switch-cost provides direct support for the oculomotor inhibition hypothesis.

There are three issues to address in terms of supporting the above-mentioned interpretation. The first relates to whether the task- and stimulus-presentation paradigm used here produced a sufficient number of error antisaccades by which to dichotomize prosaccade task-switch trials. Table 1 shows that 13.5% of prosaccade task-switch trials were preceded by an error antisaccade (i.e., trials categorized as PrS-E) and that participants produced on average 13 trials in this category. Moreover, we observed that RT variability did not vary as a function of PrS-E and PrS-C trials. Thus, the present paradigm provides a suitable frequency and extant RT stability by which to examine the planning times of PrS-E trials. The second issue relates to the planning properties of correct and error antisaccades. More specifically, did the RTs for the trials preceding PrS-C and PrS-E responses differ? Correct antisaccades produced longer RTs than their error counterparts, and RTs for error antisaccades did not differ from PrR trials. Certainly, the longer RTs of correct antisaccades are indicative of the temporal demands of response suppression and vector inversion (Munoz & Everling, 2004), whereas the shorter RTs of error antisaccades suggests a failure to suppress a stimulus-driven prosaccade (cf. Everling, Dorris, & Munoz, 1998; Mokler & Fischer, 1999).[3] As such, results show that the *planning* of a correct antisaccade is necessary to delay the *planning* of a subsequent prosaccade. In turn, the finding that error antisaccades produce a null switch-cost suggests that such actions were planned as stimulus-driven prosaccades (i.e., the action was planned without response suppression or vector inversion) and as a consequence did not impart a prosaccade switch-cost. The third issue relates to whether the outcome (i.e., endpoint), and not the planning processes, associated with correct and error antisaccades influenced the unidirectional prosaccade switch-cost. Indeed, it could be the case that only correct antisaccades produced a switch-cost because such actions were selectively associated with the obligatory decoupling between-stimulus and response; that is, the attainment of an endpoint in mirror-symmetrical space. In addressing this issue, we note that the majority of the error antisaccades (i.e., 96%) involved a short-latency trajectory correction (see Mokler & Fischer, 1999) resulting in an endpoint position commensurate to correctly planned antisaccades. That error antisaccades were associated with a trajectory correction indicates that the prosaccade switch-cost cannot be attributed to a difference between the outcome of correct and error antisaccades, or a difference in visual attentive processes supporting the decoupling of stimulus and response (Kowler & Blaser, 1995). Further, the equivalent endpoints indicate that the switch-cost is not accounted for by between trial-type differences in the maintenance of high-level task goals (Day & Lyon, 2000); rather, evidence shows that antisaccade planning selectively delays the planning of a subsequent prosaccade.

The major findings from this study were that correct antisaccades produced longer RTs than error antisaccades and that prosaccades following the former trial-type were selectively associated with a switch-cost. We believe that such results support the oculomotor inhibition hypothesis and are in agreement with work showing that the planning of a correct antisaccade is mediated by an oculomotor pre-setting that withholds the evocation of a stimulus-driven prosaccade. More specifically, neuroimaging evidence has shown that fronto-parietal regions common to pro- and antisaccades exhibit increased activity during the preparatory period for the latter task (Brown, Vilis, & Everling, 2007; Connolly et al., 2002; Ford et al., 2005). The increased activation has been linked to the increased top-down demands of the antisaccade task and is also thought to reduce the chance that a stimulus-driven prosaccade will be executed (see Brown, Vilis, & Everling, 2007). As well, single-cell recordings in the superior colliculus have shown that the preparatory period of the antisaccade task is linked to greater activation of fixation neurons and a decreased activation of build-up neurons in order to lock the eyes onto the fixation until the constituent elements of the antisaccade task have been computed (i.e., response suppression and vector inversion) (Everling et al., 1999; Everling, Dorris, & Munoz, 1998). Furthermore, Pouget et al. (2011) has shown that the lengthening of RT following a stop-signal saccade is tied to the same stochastic accumulator mechanisms as those characterizing speed/accuracy adjustments (Fitts, 1954). In other words, the top-down demands and sensorimotor consequences of a previous trial (i.e., pre-setting to withhold a stimulus-driven prosaccade) influences the time in which the neural activity associated with the planning of a subsequent prosaccade first begins to accumulate.

A final issue that we address is how the oculomotor inhibition hypothesis differs from Allport, Styles, and Hsieh's (1994) task-set inertia theory. Indeed, Allport et al. reported that switching

[3] Some work (Everling, Dorris, & Munoz, 1998) has shown that error antisaccades produce RTs in the range of express prosaccades (~100 ms; see Fischer & Ramsperger, 1984). In contrast, other studies including the present results have shown that error antisaccade produce RTs that are comparable to their non-express prosaccade counterparts (Evdokimidis, Tsekou, & Smyrnis, 2006; Mokler & Fischer, 1999; Tatler & Hutton, 2007). Although the basis for the aforementioned discrepancy is beyond the scope of this paper, it is important to highlight that the literature has consistently demonstrated that error antisaccades produce RTs that are shorter than their directionally correct counterparts.

from the unfamiliar color-naming (i.e., non-standard task) to the familiar word-naming (i.e., standard task) variant of the Stroop task elicited a reliable increase in word-naming RTs, whereas the converse switch did not. Based on these findings, Allport et al. proposed that a non-standard task requires the active inhibition of a standard task and thereby produces a persistent inhibition that delays the planning of a subsequent standard task. In turn, it was concluded that a standard task does not entail active inhibition and therefore does not interfere with the planning of a subsequent non-standard task (see also Wylie & Allport, 2000). Notably, however, the cognitively motivated nature of task-set inertia theory does not provide a framework (or prediction) for dissociable switch-costs for standard tasks preceded by correct or error non-standard tasks. Thus, and although task-set inertia provides a valid basis for our findings showing a switch-cost for prosaccades (i.e., standard task) preceded by a correct antisaccade (i.e., non-standard task), it does not provide an account for the null switch-cost for prosaccades preceded by an error antisaccade. Moreover, we believe that the proposed oculomotor inhibition hypothesis offers a more parsimonious account because it provides a direct and mechanistic framework for understanding the *planning* processes influencing the expression of the unidirectional prosaccade switch-cost.

5. Conclusions

The unidirectional prosaccade switch-cost manifests as a function of the planning, and not outcome, consequences of a preceding antisaccade. More directly, we propose that the constituent elements related to the planning of a correct antisaccade (i.e., response suppression and vector inversion) inhibit the efficiency and effectiveness of the planning mechanisms supporting stimulus-driven prosaccades. Notably, our future work is aimed at determining the degree to which each of the constituent elements of the antisaccade task contribute to the inhibition of stimulus-driven oculomotor networks.

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