Performance Monitoring during Visual Priming

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Abstract

■ Repetitive performance of single-feature (efficient or pop-out) visual search improves RTs and accuracy. This phenomenon, known as priming of pop-out, has been demonstrated in both humans and macaque monkeys. We investigated the relationship between performance monitoring and priming of pop-out. Neuronal activity in the supplementary eye field (SEF) contributes to performance monitoring and to the generation of performance monitoring signals in the EEG. To determine whether priming depends on performance monitoring, we investigated spiking activity in SEF as well as the concurrent EEG of two monkeys performing a priming of pop-out task. We found that SEF spiking did not modulate with priming. Surprisingly, concurrent EEG did covary with priming. Together, these results suggest that performance monitoring contributes to priming of pop-out. However, this performance monitoring seems not mediated by SEF. This dissociation suggests that EEG indices of performance monitoring arise from multiple, functionally distinct neural generators. ■

INTRODUCTION

The recent history of an observer's visual experience has profound impact on the manner in which she perceives, attends to, and acts upon stimuli in the present (Helmholtz, 1867). One salient example of this phenomenon is priming (Tulving & Schacter, 1990). Priming can be observed in visual search when the target and distractors of a pop-out array have unchanging feature assignments (Maljkovic & Nakayama, 1994, 1996). Priming of pop-out has been described for macaque monkeys (Purcell, Weigand, & Schall, 2012; Bichot & Schall, 2002). Investigation into the neural basis of this phenomenon is ongoing with electrophysiology in human participants (Eimer, Kiss, & Cheung, 2010) and neurophysiology in monkeys (Bichot & Schall, 2002). However, the neural mechanism leading to the behavioral consequences remains unknown. Several competing hypotheses explain priming of pop-out. Some investigators identify attention mechanisms (Kristjansson & Asgeirsson, 2019; Kristjansson & Campana, 2010), whereas others emphasize memory mechanisms (Huang & Pashler, 2005; Huang, Holocombe, & Pashler, 2004; Hillstrom, 2000). Given the systematic variation in errors during priming of pop-out, we surmise that performance monitoring also contributes either in conjunction with the other suggested mechanisms or entirely on its own.

Performance monitoring has been operationalized as the realization of errors leading to altered behavior (Sajad, Godlove, & Schall, 2019; Stuphorn, Taylor, & Schall, 2000; Carter et al., 1998; Coles, Scheffers, & Fournier, 1995). Performance monitoring has been shown in both human participants (Gehring, Goss, Coles, Meyer, & Donchin, 1993; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991) and macaque monkeys (Godlove et al., 2011; Emeric, Brown, Boucher, et al., 2007). Neurons in the supplementary eye field (SEF; Emeric, Leslie, Pouget, & Schall, 2010; Stuphorn et al., 2000) and the ACC (Emeric, Brown, Leslie, et al., 2007; Ito, Stuphorn, Brown, & Schall, 2003) signal errors and reward. These neurons reliably modulate their firing rate with respect to the behavioral outcome of a visual task (see Schall, Stuphorn, & Brown, 2002, for a review). These firing rate changes seem to affect behavior on subsequent trials (Sajad et al., 2019).

Interestingly, a prior investigation into whether SEF activity contributes to priming of pop-out suggested that visual processing in SEF is not affected by pop-out search (Purcell et al., 2012). However, that investigation was mainly limited to visual responses, which constitute just a subset of SEF responses. The exact role of SEF for priming of pop-out thus remains unclear, which is further exacerbated by the fact that other studies have shown that SEF can impact behavior beyond visual processing (Emeric et al., 2010; Stuphorn et al., 2000).

Noninvasively, neuronal signatures of performance monitoring can be measured via two ERPs known as the error-related negativity (ERN; Gehring et al., 1993) and error positivity (Pe; Falkenstein et al., 1991). Immediately following an error, the ERP is more negative than when a response was made correctly, and this difference is referred to as the ERN. Following the ERN, the Pe manifests as a greater positive polarization of the ERP when an incorrect response was made. Both the ERN and Pe have been observed in humans and monkeys (Sajad et al.,

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2019; Phillips & Everling, 2014; Godlove et al., 2011; see Woodman, 2012, for a review). Whether or not these two performance monitoring signals modulate during priming of pop-out is unknown.

Previous work has demonstrated that monkeys have homologues of human cognitive ERP components N2pc (Purcell, Schall, & Woodman, 2013; Heitz, Cohen, Woodman, & Schall, 2010; Cohen, Heitz, Schall, & Woodman, 2009; Woodman, Kang, Rossi, & Schall, 2007), contralateral delay activity (Reinhart, Carlisle, Kang, & Woodman, 2012), and performance monitoring components ERN and Pe (Sajad et al., 2019; Godlove et al., 2011). By recording single-unit activity in brain areas demonstrating signals related to cognitive capacities of interest, that activity can be related to the ERP components that manifest at the scalp. This allows investigators to draw conclusions about what brain areas contribute to certain ERP components. Previous work suggests the single-unit activity in SEF contributes to the ERN (Sajad et al., 2019). In this study, we test the hypothesis that both EEG indices and SEF activity related to performance monitoring vary in concert with priming of pop-out. To do so, we simultaneously recorded the activity of single units in SEF and EEG of monkeys performing priming of pop-out visual search.

METHODS

Animal Care and Surgical Procedures

In a series of surgeries, two male macaque monkeys (one *Macaca radiata*, Monkey F, age = 18 years; one *Macaca mulatta*, Monkey Z, age = 7 years) were implanted with a head post and recording chamber concurrent with a craniotomy situated over medial frontal cortex (Purcell et al., 2012). Briefly, surgeries were performed under aseptic conditions with the monkeys under isoflurane anesthesia. Postoperative antibiotics and analgesics were administered. All procedures were in accordance with the National Institutes of Health Guidelines, the American Association for Laboratory Animal Care Guide for the Care and Use of Laboratory Animals, and approved by the Vanderbilt Institutional Animal Care and Use Committee.

Experimental Design and Behavior

Monkeys were trained to perform a pop-out visual search task presented on a CRT monitor at 60 Hz where the relevant feature was the color of a stimulus. We used the colors red (Commission International de l'Eclairage chromaticity coordinates $x = 0.648$, $y = 0.331$) and green (Commission International de l'Eclairage chromaticity coordinates $x = 0.321$, $y = 0.598$). These colors were rendered isoluminant at 2.8 cd/m^2 and presented on a uniform gray background. Monkeys began a trial by fixating within a 1-dva diameter space around a central fixation cross for a variable amount of time around 500 msec. Immediately following, a visual search array consisting of eight items at 10-dva eccentricity was presented to the monkey. One item in the array was of a different color than the others (Figure 1), and the monkey was tasked to saccade to that item within 2000 msec and maintain fixation within a 2–4 dva space around the target. Eye movements were monitored continuously at 1 kHz using an infrared corneal reflection system (SR Research). If the monkey successfully shifted gaze to the target and held fixation at the target for 500 msec, the monkey received

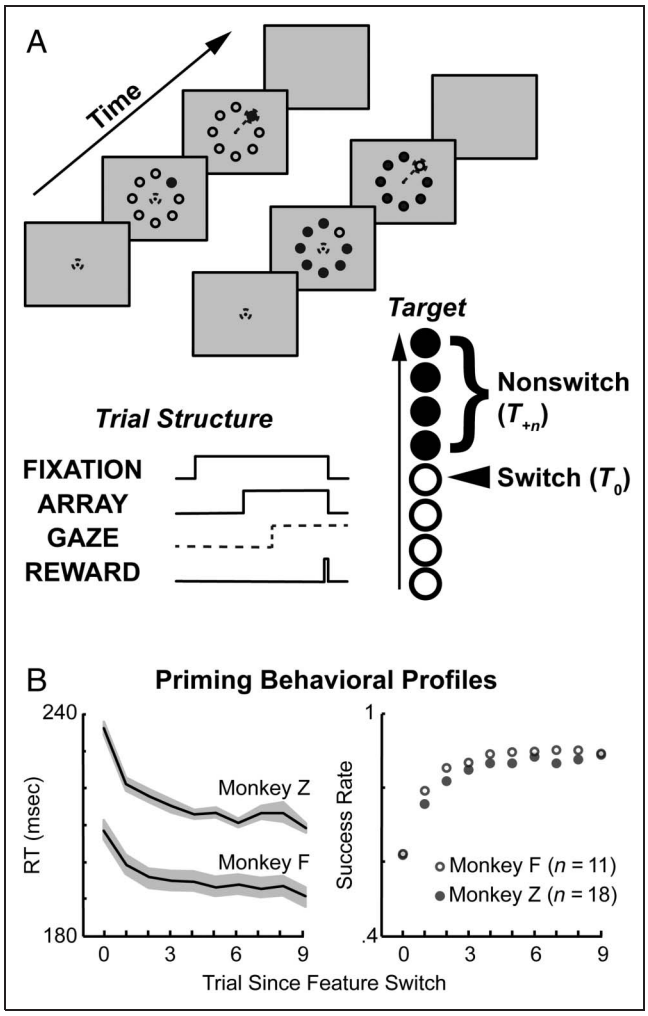

Figure 1. Experimental design and associated task performance. (A) Monkeys ($n = 2$) fixated for a brief duration before an eight-item visual search stimulus array appeared onscreen. Monkeys then were tasked to make a saccade to the pop-out target (e.g., red target among green distractors or vice versa) to receive reward. Monkeys were required to fixate the target for 500 msec before a juice reward was delivered. Every 8–16 trials target and distractor colors were swapped. (B) Left: Profile of correct RTs relative to the color switch for each monkey, averaged across both color combinations. Gray shading represents 95% confidence interval around mean. The trial immediately following the color switch (unprimed trial, 0, T0) shows the slowest RT, followed by facilitation of the response with repetition of target color. Right: Success rates relative to the color switch for each monkey, averaged across both color combinations. Performance improves with repetition of target color.

juice reward. If the monkey made the saccade to a distractor instead, the monkey did not receive juice reward on that trial and experienced a short (1–5 sec) timeout. Trials were presented in blocks of 8–16 trials, sampled from a uniform distribution. The colors of the target and distractors were held constant throughout the block. At the end of the block, the target color and distractor color were swapped.

SEF Localization

Confirmation of recording sites was demonstrated previously (Purcell et al., 2012). Briefly, intracortical microstimulation and histological studies of both monkey brains following completion of the experiment confirmed that all recording locations were within the confines of SEF.

Neurophysiological Procedure

Intracranial neural data were acquired with 40-kHz resolution from the right hemisphere SEF of both monkeys using tungsten microelectrodes (2–4 MΩ, FHC) across 29 sessions (n = 11, Monkey F; n = 18, Monkey Z). All data were referenced to a stainless steel guide tube in contact with the dura mater. To isolate single-unit activity, the neural data were sorted both online and offline using a time–amplitude window discriminator, PCA, and template matching (Plexon). For further processing, spikes were convolved using a kernel resembling the shape of an excitatory postsynaptic potential to derive spike density functions (Westerberg, Cox, Dougherty, & Maier, 2019; Thompson, Hanes, Bichot, & Schall, 1996).

Cranial EEG was acquired following previously reported methods (Purcell et al., 2012; Woodman et al., 2007). Briefly, gold electrodes were implanted at a depth of 1 mm into the skull. Implantation locations approximated human 10–20 locations, scaled to accommodate the smaller size of the monkey skull. EEG signals were recorded at 1 kHz and filtered between 0.2 and 300 Hz. Referencing was performed using a frontal EEG electrode, approximating human Fz. ERPs were extracted relative to events of interest from the average of distal sites O1 and O2 and polarity inverted to represent the polarization at the reference electrode site where the ERPs of interest (the ERN and Pe) are generally largest.

Data Analysis and Statistics

Before analysis, both single-unit and EEG data were baseline-corrected on a trial-by-trial basis by subtracting the mean activity from the 100 msec before search array onset while the animal maintained fixation. Initial analysis identified SEF single units showing error-related activity. We measured the mean activity of each single unit during the postsaccade epoch before reward delivery (e.g., 0–500 msec postsaccade) both on correct trials (i.e., monkey saccaded to the target) and on error trials (i.e., monkey saccaded to a distractor). Error trials where the monkey made a corrective saccade were eliminated from analysis to not contaminate the error response with saccade-related activity. We defined an error-related response as significant using a paired t test with $p < .05$. We found a population of SEF neurons that was significantly facilitated by errors as well as a population that was facilitated by correct responses, hereafter referred to as error-responding and correct-responding neurons, respectively.

To draw conclusions about whether the priming condition affected the error-related activity, we performed standard paired t tests at the session level with equal ns for primed and unprimed conditions and also analyses using Bayes factor-adjusted paired t tests (Rouder, Speckman, Sun, Morey, & Iverson, 2009; Kass & Raftery, 1995). This technique provides evidence for the null hypothesis, in contrast to other statistical methods. By calculating the Bayes factor between conditions over time, we can determine whether or not SEF error-related signals modulate across the visual priming sequence in a statistically rigorous manner that illuminates the validity of both the null and alternative hypotheses. We compared unprimed (switch) trials to primed (nonswitch) trials for analysis of priming effect. Unprimed was operationally defined as the first trial following the change of target color, and primed trials were all subsequent trials. The modifier "unprimed" or "primed" will hereafter refer to the state of the target color on a given trial.

RESULTS

Behavioral Evidence for Priming of Pop-out

Two monkeys performed an eight-item, color pop-out visual search task (Figure 1A). Monkeys fixated a central cross for a variable amount of time (~500 msec) before the presentation of the search array. On any given trial, one item in the array was a different color from the others (either red or green with the distractors being a homogenous set of the other color). Monkeys were required to make a saccade to the singleton target to receive a juice reward once they had fixated the target for 500 msec. If they made an incorrect saccade (i.e., to any other location than the singleton target), they did not receive juice and experienced a short timeout. Trials were clustered into blocks so that monkeys searched for the same color singleton among the same distractors for a randomly chosen 8–16 consecutive trials.

Both animals' task performance was consistent with previously reported behavioral data from studies in humans and macaques (Bichot & Schall, 2002; Maljkovic & Nakayama, 1994, 1996) and replicated work of an earlier study of the same behavioral data (Purcell et al., 2012). Specifically, the RTs of the animals consistently decreased, whereas response accuracy increased with repeated trials as long as search conditions remained

consistent (Figure 1B). Following a swap of target and distractor color assignments, performance briefly deteriorated before improving again.

Priming Modulates ERP Indices of Performance Monitoring

We first determined whether the ERN immediately after the saccade and subsequent Pe manifest during the pop-out visual search task (Figure 2) regardless of priming condition in each monkey. Figure 2A shows the ERP around the time of response for Monkey Z (Figure 2A, left) and Monkey F (Figure 2A, right). Figure 2B quantifies the polarization during the ERN, immediately following the response (0–200 msec postresponse) and the Pe (200–400 msec postresponse). Both monkeys showed a significant ERN (Monkey Z, $M_{error} = -1.50$, $M_{correct} = 0.46$, $SD = 3.2$, paired t test $t(17) = 2.46$, $p = .025$; Monkey F, $M_{error} = -2.60$, $M_{correct} = -0.45$, $SD = 2.80$, $t(10) = 2.86$, $p = .014$) and Pe (Monkey Z, $M_{error} = -3.80$, $M_{correct} = -8.10$, $SD = 3.20$, $t(17) = -5.62$, $p = 3.80\text{E}{-05}$; Monkey F, $M_{error} = -8.70$, $M_{correct} = -10.70$, $SD = 3.00$, $t(10) = -2.44$, $p = .029$). Turning our attention to the effect of priming condition (Figure 3), we found a significant ERN, measured as the mean difference in polarization during the 200 msec following the saccade between correct and error responses on primed (nonswitch) trials through a one-sample t test ($M = 1.40$, $SD = 3.00$), $t(28) = -2.53$, $p = .01$. Interestingly, an ERN was not detected on unprimed (switch) trials using the same measure ($M = 0.343$, $SD = 5.10$), $t(28) = 0.364$, $p = .718$. In contrast, the Pe was significant in both unprimed ($M = 2.7$, $SD = 5.3$), $t(29) = 2.77$, $p = .009$, and primed conditions ($M = 4.2$, $SD = 4.1$), $t(28) = 5.49$, $p = 7.29 \times 10^{-6}$. These results show that the ERN does not manifest immediately following a search array feature switch but emerges later in the priming sequence, whereas the Pe occurs regardless of position in priming sequence.

We next sought to determine whether there was a systematic change in the magnitude of these indices as a function of priming condition. We found a significant difference between the magnitude of the ERN between the unprimed ($M = 0.343$, $SD = 5.10$) and primed ($M = 1.40$, $SD = 3.00$) conditions, $t(28) = 2.14$, $p = .02$. Also, the Pe was marginally but not significantly smaller in the unprimed ($M = 2.70$, $SD = 5.30$) relative to primed ($M = 4.20$, $SD = 4.10$) trials, $t(28) = -1.48$, $p = .07$.

To investigate further the variation of ERN and Pe polarization between the different priming and performance conditions, we plotted the mean polarization for primed and unprimed correct and error responses (Figure 3C). Although there was no difference between unprimed correct and error trials through the subtractive analyses detailed above, the polarization of these two conditions was more similar to that of the primed error than to the primed correct. To characterize the ERN and Pe polarization across the four trial types, we performed

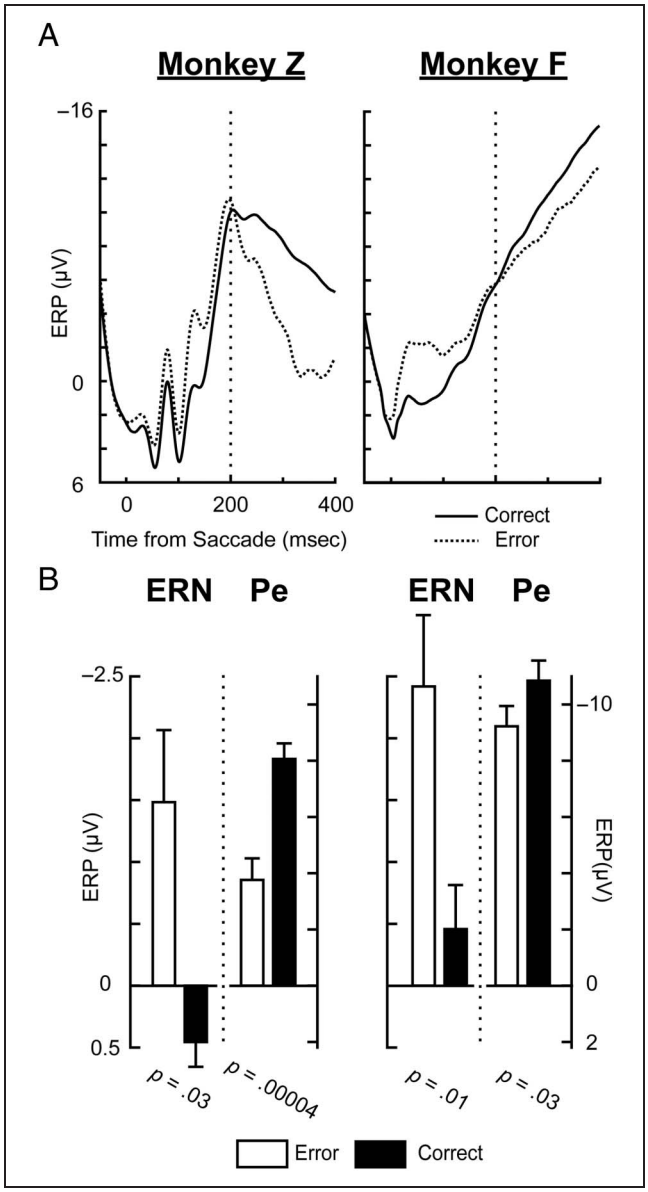

Figure 2. Monkeys show ERN and Pe following response in visual search. (A) Individual monkey ERPs around the time of saccadic response averaged across sessions (left, Monkey Z, $n = 18$; right, Monkey F, $n = 11$) for correct (solid) and error (dotted) conditions. Dashed vertical line at 200 msec denotes cutoff point for ERN (0–200 msec postsaccade) and Pe (200–400 msec postsaccade) for subsequent analysis. (B) Mean values for correct (black) and error (white) conditions during the ERN and Pe epochs for each monkey with error bars (2 *SEM*). Both monkeys show a significant ERN and Pe with p values noted below bar graphs.

model comparisons among a set of three linear models. Model 1 treats ERP polarization as the sum of a motor signal (manifest in the correct primed trials), a surprise signal (manifest in unprimed trials), and a trial-specific error signal (manifest in unprimed and primed error trials):

$$\text{ERP} = M + S + E_u + E_p \quad (1)$$

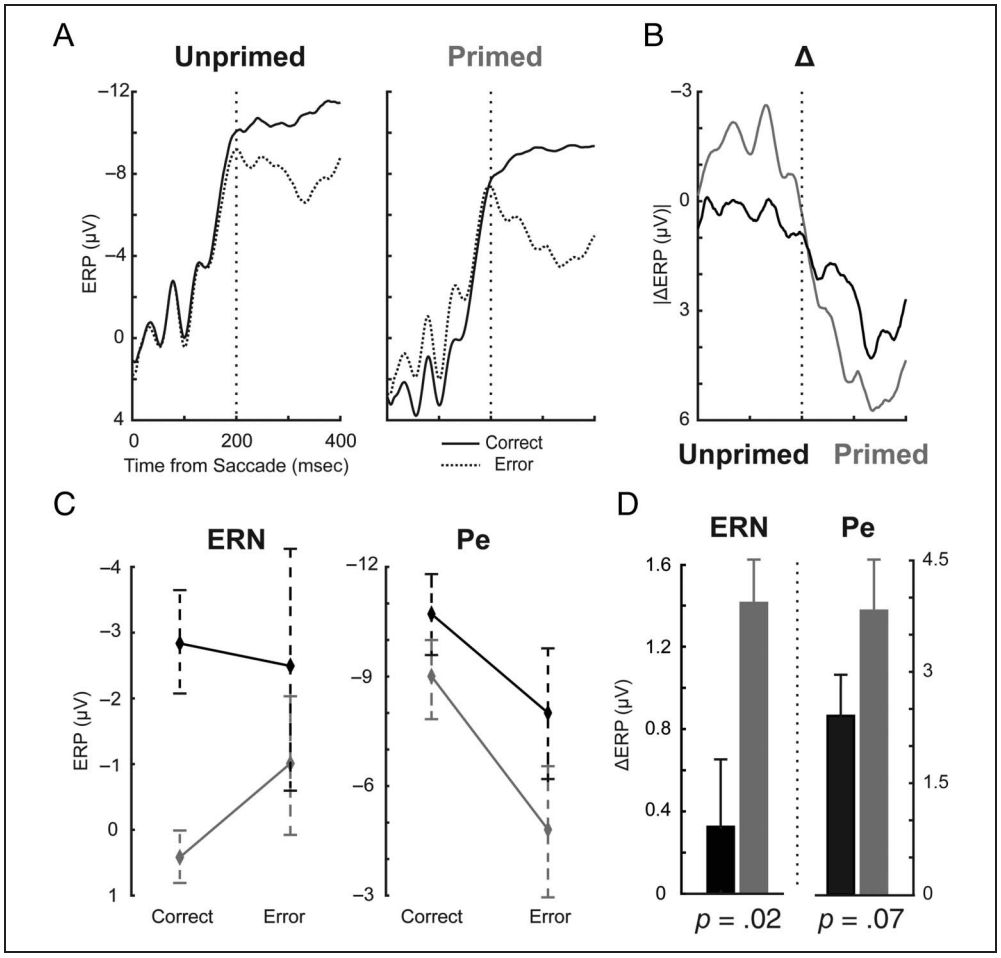

Figure 3. Priming enhances ERN and Pe. (A) Mean ERP averaged across 29 sessions (n = 29) and both monkeys (n = 2). Solid lines denote the ERP following a correct saccade to the target and dotted lines denote the ERP following a saccade to a distractor. Trials are parsed by whether it was the first trial in a sequence (left; unprimed) or later in the sequence (right; primed). Vertical, dotted line at 200 msec denotes the cutoff between the ERN and the Pe. (B) Difference between ERPs following error and correct responses for unprimed (black) and primed (gray) trials. (C) Comparison of mean polarizations for ERN (left) and Pe (right) across priming conditions (black, unprimed; gray, primed). Dashed lines denote 2 *SEM*. (D). Mean ERP differences across sessions collapsed across designated epoch. Bar height denotes mean, and line denotes 2 *SEM*.

where ERP is the estimated mean polarization of the ERN or the Pe, M is the motor signal, S is the surprise signal, and E_u and E_p are unique error signals for the unprimed and primed conditions, respectively. Model 2 treats the ERP as the sum of the same motor and surprise signals but only a single error signal regardless of priming condition:

$$\text{ERP} = M + S + E_{up} \quad (2)$$

where E_{up} is an error signal common to both priming conditions. Finally, Model 3 treats the ERP as the sum of the same motor signal, no surprise signal, and a single error signal common to all trials:

$$\text{ERP} = M + E \quad (3)$$

where E is an error term spanning unprimed correct, unprimed error, and primed error conditions.

For the ERN, Model 3 best captured the experimental observations. The F statistic for Model 3 was more than double that of Model 1 (F_{M3} = 9.63, p_{M3} = .0024; F_{M1} = 4.54, p_{M1} = .0048) and had a lower Akaike information criterion (AIC; AIC_{M3} = 639.18, AIC_{M1} = 639.27). Although the AIC was lower for Model 2 (AIC_{M2} = 638.91) than for Model 3, the addition of the error term to Model 2 was insignificant (Model 2: $E_{up} = -0.54$, T = −0.77, p = .44).

For the Pe epoch, Model 2 best captured the experimental observations. Model 2 had the largest F statistic computed against a constant model (F_{M2} = 8.86, p_{M2} = .00027; F_{M1} = 6.06, p_{M1} = .00073; F_{M3} = 0.885, p_{M3} = .35) and the lowest AIC (AIC_{M2} = 722.19, AIC_{M1} = 723.62, AIC_{M3} = 736.19). All estimated coefficients of Model 2 were significant (Model 2: $M = -8.63$, $T = -9.91$, $p = 4.93 \times 10^{-17}$; $S = -2.45$, $T = -2.44$, p = .016; E_{up} = 3.45, T = 3.43, p = .00083).

Performance Monitoring SEF Neurons

Although EEG was measured extracranially, we also sampled the spiking of single units in the SEF of the medial frontal cortex during task performance. We sampled 91 single units from two monkeys across 29 sessions. To determine which units showed performance monitoring-related activity, we calculated the mean difference in firing rate from the 100 msec preceding the saccade until 500 msec following the saccade. We then compared the resulting response magnitude between trials where the animal saccaded to the correct item location (the color singleton) to when the animal saccaded to a distractor item in the

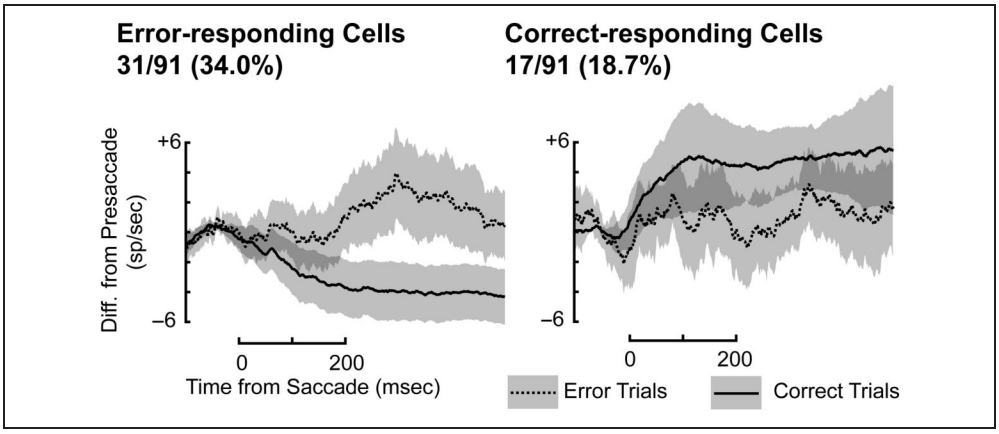

Figure 4. Neural populations showing performance monitoring activity in SEF. Across 30 sessions (n = 12, Monkey F; n = 18, Monkey Z), 91 single units were isolated (n = 29, Monkey F; n = 62, Monkey Z). Among these, we found two populations with performance monitoring activity that were either significantly enhanced or suppressed with failure to perform the task. These units are denoted as "error-responding" and "correct-responding," respectively. We baseline-corrected all data to the presaccadic period so that we would only capture differences related to performance. Each line represents the mean across each population with the gray shading denoting the 95% confidence interval around each mean.

array. To eliminate a confound of eye movement-related activity during the interval of performance monitoring activity, we eliminated trials when a secondary (corrective) saccade was made <500 msec following the initial response.

Of 91 SEF single units, 48 (52.7%) showed a significantly different mean response between correct and error trials (paired t test, $p < .05$). We subdivided these performance monitoring neurons into two subpopulations based on whether each single unit responded significantly more following a correct response or an error response. We refer to these neurons as correct-responding and error-responding neurons, respectively. Of the 48 single units modulating in relation to performance monitoring, 31 (31.0% of the total population) were more active after error trials (error-responding population), and 17 (18.7%

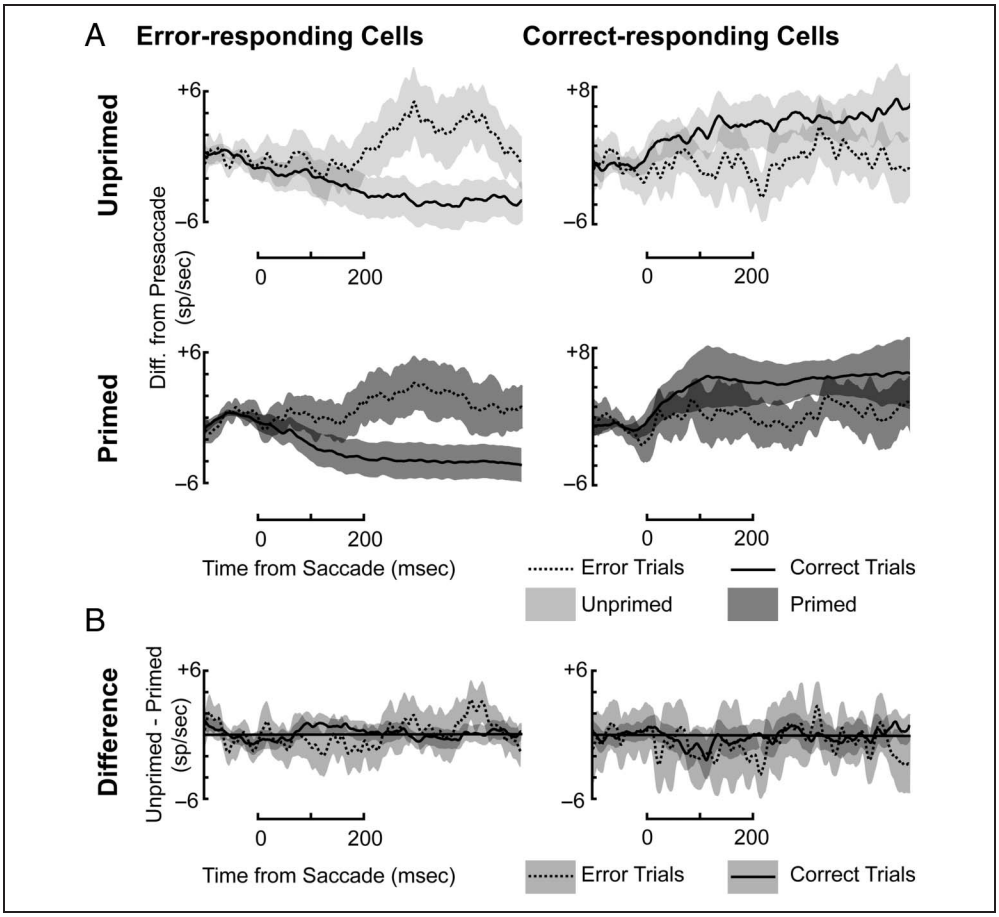

Figure 5. Population response by priming condition. (A) Mean response across all monkeys (n = 2) and sessions (n = 30) for each SEF cell population (left, error-responding, n = 31; right, correct-responding, n = 17) separated by priming condition (top, unprimed; bottom, primed) comparing correct (solid) and error (dotted) conditions. Each line represents the mean across each population with the gray shading denoting the 95% confidence interval around each mean. (B) Difference plots between unprimed and primed conditions (unprimed–primed) for the error (dotted) and correct (solid) conditions, individually, for each SEF population (left, error-responding; right, correct-responding). A difference profile was computed for each session and then averaged to obtain the trace shown. Again, each line represents the mean across each population, with the gray shading denoting the 95% confidence interval around each mean.

Table 1. Performance Monitoring Across Priming Conditions and SEF Populations

Priming Condition	*Population*	*M (Correct)*	*M (Error)*	*SD*	*df*	*t*	*p*
Unprimed	Error-responding	−3.15	1.18	5.18	30	4.66	6.14E−05
Unprimed	Correct-responding	4.29	0.40	4.16	16	−3.85	.0014
Primed	Error-responding	−3.30	1.10	3.32	30	7.37	3.24E−08
Primed	Correct-responding	4.50	1.21	4.00	16	−3.39	.0037

Means were taken from the 500 msec following the saccade. *M* and *SD* measures are in sp/sec.

total population) were more active after correct trials (correct-responding). The mean spiking response for each of these populations is shown in Figure 4.

Priming Does Not Modulate SEF Performance Monitoring

After identifying the neural populations contributing to performance monitoring during the pop-out task, we investigated their role during priming. Previous work suggested that SEF contributes directly to the generation of the ERN (Sajad et al., 2019). Given that the ERN modulated significantly as a function of priming, we hypothesized that SEF performance monitoring neurons would modulate likewise. To test this hypothesis, we performed two analyses. First, we determined if there was an absence of performance monitoring signals in the neural populations of SEF in unprimed trials, immediately following a switch, like the absence seen in the ERN. Then, we compared spiking responses of the error-responding and correct-responding populations between trials immediately following the color switch between target and distractors and the trials following the switch (i.e., unprimed and primed trials), respectively.

Figure 5 details the performance monitoring activity of SEF populations as a function of priming condition. Qualitatively, there appears to be a difference between correct and error responses for both the error-responding (Figure 5A, left) and correct-responding populations (Figure 5A, right) in both the unprimed (Figure 5A, top) and primed conditions (Figure 5A, bottom). Quantitative analysis was done by performing paired *t* tests ($p < .05$) on the mean activity during the 500 msec following the saccade between the correct and error conditions for both neural populations and priming conditions. We found a significant difference for all four combinations of priming condition and population. Table 1 summarizes the results of this statistical analysis, which reveal a disparity between performance monitoring signals measured in the EEG and in SEF neural discharges. Furthermore, it may suggest a lack of sensitivity of SEF populations to priming conditions.

We next determined whether there was a significant change in the responses from unprimed to primed conditions at the population level. Figure 5B shows the difference between unprimed and primed responses for correct and error conditions for both populations. Qualitatively, there appears to be no difference in the response of either population by outcome between unprimed and primed trials. Quantitative analysis via paired *t* tests ($p < .05$) confirms this suspicion (Table 2). This suggests that, at the population level, SEF performance monitoring populations do not modulate as a function of priming condition regardless of trial outcome.

It is conceivable that there are subpopulations within the error-responding or correct-responding populations that could be sensitive to priming conditions. With the lack of modulation at the population level, we examined the data at a finer level, unit-by-unit, to determine whether any single units in fact modulate with priming of pop-out. Activity of an error-responding unit during primed and unprimed trials is shown in Figure 6A. Qualitatively, this unit shows no differentiation between primed and unprimed trials. Given this observation, we adopted a statistical test that can endorse invariance, because standard frequentist statistics such as Student's *t* test cannot provide evidence for a null hypothesis. Hence, we implemented a Bayesian approach (Rouder et al., 2009; Kass & Raftery, 1995) to

Table 2. SEF Population Sensitivity to Priming Conditions by Outcome and Population

Trial Outcome	*Population*	*M (Unprimed)*	*M (Primed)*	*SD*	*df*	*t*	*p*
Error	Error-responding	1.18	1.10	3.78	30	0.12	.91
Error	Correct-responding	0.40	1.21	2.21	16	−1.51	.15
Correct	Error-responding	−3.15	−3.30	1.25	30	0.66	.51
Correct	Correct-responding	4.29	4.50	1.62	16	−0.55	.59

Means were taken from the 500 msec following the saccade. *M* and *SD* measures are in sp/sec.

test the level of evidence for differences in spiking responses between the primed and unprimed trials (alternative hypothesis) or no significant effect on neural activation (null hypothesis).

Figure 6B illustrates the results of this analysis for the example unit. A Bayesian-adjusted p value was computed across the performance monitoring epoch between the primed and unprimed conditions. Values below 1 represent evidence in support of the null hypothesis, whereas values above 1 (which were not present in the analysis of this unit) represent evidence in support of the alternative hypothesis. Significance levels were taken from previously determined thresholds (Kass & Raferty, 1995). This example unit showed strong evidence across time

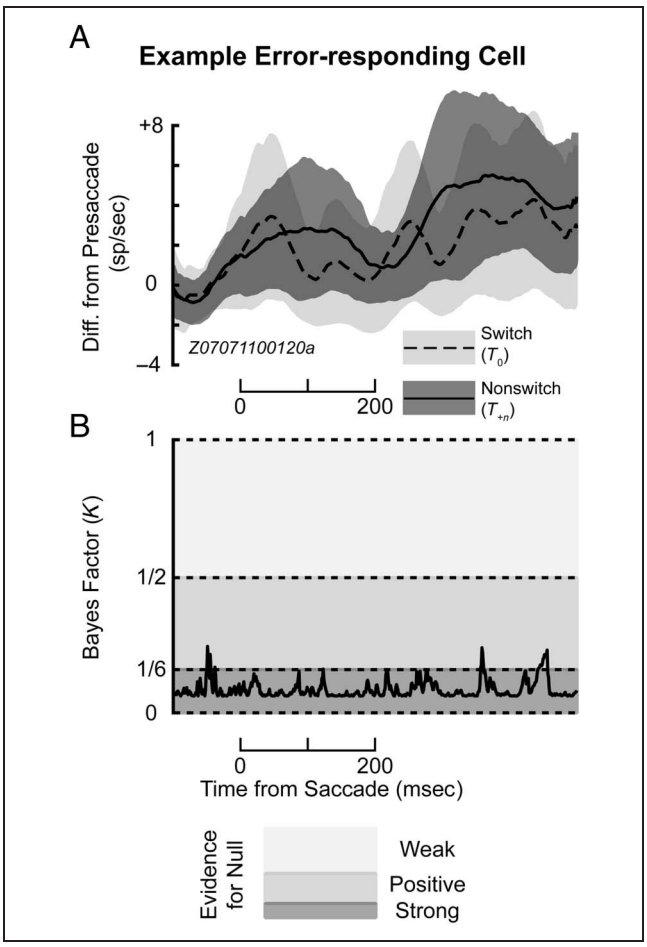

Figure 6. Example single unit showing priming invariance. (A) Comparison of spiking differences when an error was made on a nonprimed trial (dashed line) to an error during a primed trial (solid line). Means across trials are denoted by the lines and are smoothed with a 50-msec moving average window (used for visualization only). Corresponding gray shades denote the 95% confidence interval around the mean. (B) Bayes factor (K) converted t statistics (see Methods for details) as a function of time, comparing the primed versus unprimed conditions for the same single unit. Note that these data are not smoothed. Most of the trace exists below the level of strong evidence for the null hypothesis, suggesting that the likelihood for a systematic response difference between these conditions is low.

for lack of a response difference between primed and unprimed trials.

Figure 7 summarizes the results of this analysis for error-responding (Figure 7, left) and correct-responding (Figure 7, right) units when monkeys produced correct responses (Figure 7, bottom) or errors (Figure 7, top). Across both populations and trial outcomes, the vast majority of SEF units showed strong evidence for the null hypothesis with vanishingly few units showing evidence for the alternative hypothesis. This finding suggests that SEF neurons do not signal errors differently as a function of priming. This result marks an interesting dissociation from the EEG data and suggests that, although SEF may contribute to the generation of the ERN generally (e.g., Sajad et al., 2019), the priming-associated changes in the ERN and Pe are not due to changes in the neural activity in SEF.

DISCUSSION

We show that, although monkeys' RTs and task performance improved with repetition of feature identity in visual search, the activity of their SEF neurons did not modulate in step. Conversely, the ERN index of performance monitoring measured through EEG does significantly modulate with the Pe index seemingly changing, but not to a statistically significant degree. Together, these findings suggest that performance monitoring mechanisms operate during priming of pop-out visual search. However, the well-known neural machinery for performance monitoring in area SEF is not involved.

Representation of Errors in the ERN and Pe during Pop-out Visual Search

Through planned comparisons between unprimed and primed conditions based on subtractive analysis of correct and error trials, we found that the ERN was manifest only during primed trials; the polarization after correct and error responses in the unprimed condition were not different. However, upon further investigation it appears that the absence of an ERN in the unprimed subtraction comparison was not due to a lack of negative polarization, but rather a negative polarization across both correct and error responses. Through model comparisons, we found that the negative polarization associated with the unprimed correct, unprimed error, and primed error conditions were indistinguishable. This implies that the polarization on these three types of trials arise from a common source rather than being distinct signals. This suggests a more general role for the ERN in representing information, perhaps better described as a surprise signal. That is, a surprise signal during the ERN epoch can occur on error trials when feature assignments are unchanging (primed error), on incorrect trials when feature assignments change (unprimed error), and on correct

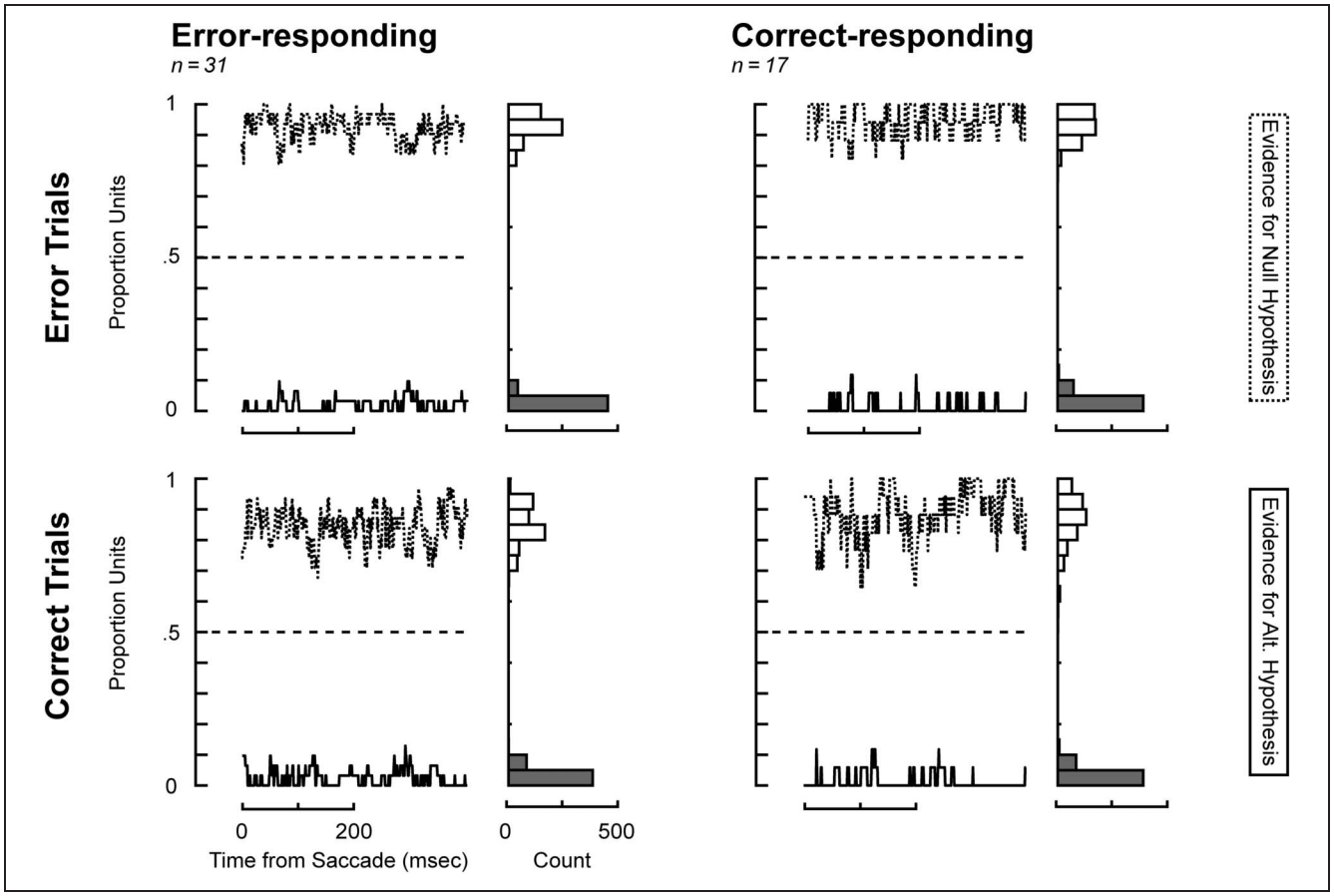

Figure 7. Population analysis of SEF performance monitoring units. Proportion of error-responding single units (left column; $n = 31$) and correct-responding units ($n = 17$) that show strong evidence for the null hypothesis (dotted line; no difference for priming) and for the alternative hypothesis (solid line; significant difference for priming) as a function of time. Comparisons are broken down by trial performance, with error trials in top row and correct trials in bottom row. Histograms to the right of each plot represent the total number of time points where the corresponding proportion of single units showed evidence for either the null (white) or alternative (gray) hypothesis. Across neural populations and performance conditions, the vast majority of single units did not significantly modulate their firing between primed and unprimed trials.

trials when feature assignments were changed (unprimed correct). This interpretation agrees with previous descriptions of the ERN as a surprise signal (Alexander & Brown, 2011); however, it is important to note this is a single task and generalization of this observation may not be appropriate.

Although a single "error" term could explain the polarization during the ERN period, the Pe was a different story. To properly describe the dynamics of the Pe in priming of pop-out, it was necessary to incorporate both the surprise associated with the unprimed trials and the differences between correct and error trials. This difference may suggest a different neural source for the Pe than the ERN that may represent the additional information, a hypothesis that will be discussed below.

Dissociation of SEF Spiking Activity and Performance Monitoring-related ERP Signatures

Recent work from our group has shown that changes in error processing in SEF translates into changes of the ERN (Sajad et al., 2019). Here, we show that, although SEF activity may be involved in the generation of the ERN in a general way or during the stop signal task, the modulation of the ERN by priming of pop-out is not due to differential spiking activity in SEF. One possibility is that SEF's role in generating the ERN might be task dependent as we measured the ERN during visual search whereas the previous study used a countermanding task (Sajad et al., 2019). It is also important to note that the previous findings relating SEF activity to the generation of the ERN was specific to neurons in the supragranular layers of SEF. For technical reasons, we were unable to identify the laminar position of single units in our data set. It thus is conceivable that we undersampled neurons from the supragranular layers and therefore missed certain SEF modulation during priming.

Provided that we did sample equally across layers, the dissociation between SEF spiking and the ERN observed in our study is interesting, as it would suggest that SEF is not the only area contributing to the ERN. One likely candidate is the ACC. ACC of the macaque monkey is positioned directly below SEF and is physically oriented in

such a way that its response-generated dipoles could contribute to frontally maximal ERP components such as the ERN (Nunez & Srinivasan, 2006). ACC also plays an important role in performance monitoring, as seen in both neurophysiological studies in monkeys (Emeric, Brown, Leslie, et al., 2007; Nakamura, Roesch, & Olson, 2005; Ito et al., 2003) and neuroimaging studies in humans (Oliveira, McDonald, & Goodman, 2007; Barch, Braver, Sabb, & Noll, 2000; Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999; Carter et al., 1998). Indeed, there is evidence from both inverse modeling (Vocat, Pourtois, & Vuilleumier, 2004; Herrmann, Römmler, Ehlis, Heidrich, & Fallgatter, 2004) and lesions (Stemmer, Segalowitz, Witzke, & Schönle, 2004) that ACC contributes to the ERN. In any case, the dissociation between SEF activity and performance monitoring ERPs suggests that these ERPs are due to an ECD that is summed across different brain areas, as researchers have long hypothesized (Nunez & Srinivasan, 2006; Luck, 2005) but have only empirically demonstrated recently (Reinhart et al., 2012; Cohen et al., 2009).

The hypothesis that the contribution of brain areas to ERP components is task dependent could be tested experimentally. Monkeys could be trained to perform multiple tasks (i.e., switching between priming of pop-out and countermanding) while intracranial neural activity and EEG are recorded. Using this paradigm, one can test the hypothesis that the same SEF neurons that contribute to the ERN during countermanding do not contribute to the modulation associated with priming of pop-out.

Significance of Differentiation in Dynamics Between the ERN and Pe During Priming

We found that priming pop-out of visual search affected performance monitoring-related ERPs following the indication of the target choice by saccadic eye movement. Both the ERN and Pe were found during task performance. However, the dynamics of these components in the priming context were not parallel. Specifically, the ERN was present for primed trials only, whereas the Pe manifested both in unprimed and primed trials. The ERN thus was significantly affected by (in fact, dependent on) priming in this task, whereas the difference in the Pe was smaller and did not reach significance. The most parsimonious explanation for this dissociation between signals might be that these EEG components are evoked by different neural circuits. Indeed, source localization of the ERN and Pe suggests that the primary contributors to the ERN and Pe are the ACC and posterior cingulate cortex, respectively (Vocat et al., 2004). Therefore, it seems plausible to assume that the error signals in these two areas are distinct, which in turn leads to differences in the respective ERPs they primarily contribute to. Given that SEF does not contribute to the ERN modulation evoked in this task, one could further speculate that the neural representation of errors under priming conditions is different for ACC and posterior cingulate cortex, respectively.

Another theoretical perspective on our ERN findings is that they support the view that the ERN is related to a general learning mechanism. That is, researchers have proposed that the ERN may index a mechanism that tracks error likelihood learning (Alexander & Brown, 2011; Brown & Braver, 2005) or reinforcement learning (Holroyd & Coles, 2002). From these perspectives, it is not surprising that an effect dependent on prior experience and learning is tracked by the amplitude of the ERN. Indeed, previous work has suggested that priming and other types of learning are due to the same underlying memory processes (Logan, 1990), and our findings appear consistent with this interpretation.

Neural Mechanisms for Priming in Pop-out Visual Search

We find that priming of pop-out does have neuronal correlates as reflected in EEG measures of performance monitoring. However, these are not the only ERP components that have been shown to modulate with priming of pop-out. In particular, human participants also show a speeded N2pc with priming (Eimer et al., 2010). The N2pc is an index of covert spatial attention (Woodman & Luck, 1999; Eimer, 1996; Luck & Hillyard, 1994). This result, among other neurophysiological evidence (Bichot & Schall, 2002), has led investigators to speculate that priming of pop-out is mediated through priming of attentional selection (Kristjansson & Asgeirsson, 2019; Kristjansson & Campana, 2010). It then seems possible that the differences in performance monitoring following priming are secondary to an attentional priming effect. FEF, an area implicated in attentional selection (Armstrong, Chang, & Moore, 2009; Monosov, Trageser, & Thompson, 2008; Thompson, Biscoe, & Sato, 2005; Juan, Shorter-Jacobi, & Schall, 2004; Kodaka, Mikami, & Kubota, 1997), shares extensive connections with not only SEF but also ACC (Schall, Morel, King, & Bullier, 1995; Schall, Morel, & Kaas, 1993). One hypothesis derived from the above is that changes in perceptual or attentional processing, in FEF or visual cortex, lead to changes in error processing in ACC. Indeed, measures of attentional selection, such as target selection time, have been shown to be facilitated in FEF with priming of pop-out (Bichot & Schall, 2002). In the same vein, visual area V4 has been shown to be necessary for behavioral changes in color-based priming of pop-out (Walsh, Le Mare, Blaimire, & Cowey, 2000). Although attentional priming seems likely to be involved in priming of pop-out, there are also competing models. Specifically, some investigators have speculated that the priming effect results from retrieval from episodic memory instead (Huang & Pashler, 2005; Huang et al., 2004; Hillstrom, 2000). Further investigation is needed to determine the exact mechanism of priming in pop-out visual search or whether multiple mechanisms are at play.

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