Congruent distractors do not facilitate, but instead intrude on task performance. Related to Figure 1.

Saccades to rewarded targets were marginally faster (~12ms) during congruent trials. This observation could be interpreted in at least two ways. First, it could indicate that two distinct saccade plans, one towards the target and one towards the distractor, were simultaneously active and facilitated each other because their vectors were similar. In this account, congruent distractors intruded on task performance, and thus caused task conflict, because they captured processing resources despite being task-irrelevant. Conversely, in this account, congruent distractors did not cause action conflict because they facilitated target-directed saccades, rather than causing competition at the level of the saccadic response. In a second, alternative account, response time speeding could indicate that congruent distractors increased attention to the target, facilitated target detection, and thereby sped response times. The following observations suggest that the former interpretation is correct: although they facilitated specific saccadic responses, congruent distractors intruded on task performance.

First, and most critically, the monkeys were more likely to make errant saccades on congruent distractor trials. In a facilitated-performance account, congruent distractors would increase the likelihood of saccades towards the rewarded target, not decrease the likelihood of these saccades, as we observe here.

Second, the temporal dynamics of the task were designed to elicit distraction effects, not attentional cueing—the targets and distractors were nearly simultaneously presented. Distractors preceded targets by 50 ms at most, compared to the hundreds of milliseconds employed in a traditional Posner exogenous cueing paradigm [1]. Moreover, in the majority (66%) of trials distractors, were presented very shortly after the target. Although these late distractors were not attentional cues, late, post-target congruent distractors still sped response times ($p < 0.0001$, paired t-test, $t(55) = 18.0$), suggesting that an attentional cueing effect could not by itself explain the effect of congruent distractors on response time.

Third, we observed systematic deviations in saccadic end points that suggest a direct effect of congruent distractors on the saccadic response even during correct, non-errant saccade trials. The oculomotor system is different from other effector systems because responses to multiple stimuli can be combined into a single saccadic response. This is a well-known phenomenon called “averaging saccades” or “center-of-gravity saccades” [2-5]. When sufficiently similar (i.e. saccade vectors within 30° of radial angle [4]), simultaneously activated saccade plans are combined—the resulting saccade targets the space between the stimuli and is faster than saccades in the presence of physically distant targets [4]. If congruent distractors speed target-directed saccades because of facilitation at the level of the saccadic response, then congruent trial saccades should exhibit averaging and saccadic end points should be deviated towards the congruent

distractors. Conversely, if congruent distractors are facilitating target detection, we should observe either improved end point accuracy or at least no systematic bias in saccadic end points on congruent trials. However, we found that saccadic end points were systematically more eccentric during congruent distractor trials, compared to all other trial types (congruent distractor end-points were 0.68° ± 0.60° STD more eccentric than non-congruent end points, within-session paired t-test, $p < 0.0001$, $t(55) = 8.46$, more eccentric on 54/56 individual sessions, within-session range: -0.29° to 2.58°, errant saccade trials excluded). Thus, saccades on congruent trials tended to be averaging saccades that landed between the target and the congruent distractor.

Thus, multiple converging lines of evidence suggest that congruent distractors facilitate saccades directly, rather than speeding response time through enhancing target detection or processing. Congruent distractors caused behavioral changes that suggest that they activated a distinct saccade plan from the target-directed saccade plan, but this saccade plan was facilitative at the level of the action. Congruent distractors nevertheless intruded on task performance through decreasing saccadic accuracy and capturing processing resources despite being irrelevant to the task.

Temporal specificity of pupil-size adjustments and dACC pupil-size signals. Related to Figures 2 and 5.

In order to determine the temporal specificity of the effects of distractors on adjustments in baseline pupil size, we examined the effect of distractor presence on baseline pupil size at various time lags relative to the current trial (time t). Distractor presence affected pupil size up to two trials into the future (effect on t+1: $p < 0.01$, $t(55) = 7.13$, t+2: $p < 0.01$, $t(55) = 7.19$), but zero trials into the past (all $p > 0.05$, Holm-Bonferroni corrected for multiple-comparisons). Though not significant, repeated distractors tended to provoke a larger adjustment in pupil size relative to the distractor-absent baseline than non-repeated distractors ($p = 0.13$, $t(55) = 1.5$).

However, it remains plausible that the activity of dACC neurons does not predict adjustments in pupil size on the next trial, but rather lacks temporal specificity. To address this issue, we repeated the GLM in main text equation 3 to determine the number of cells that significantly signaled pupil adjustment on trials at various lags before or after the current trial. We reasoned that if neuronal activity in dACC merely scales with local volatility in pupil size, firing rate on trial t should be just as predictive of pupil adjustment on the current (t) and previous (t-1) trials as it is for adjustments on the next trial (t+1). We examined 9 possible lags, from pupil adjustment 3 trials in the past (t-3) to pupil adjustment 5 trials into the future (t+5). We observed a strong temporal effect, with most neurons predicting adjustments in pupil size either on the next trial or trial t+2 (t+1 = 40 neurons, t+2 = 41 neurons). No more than a chance fraction of neurons predicted adjustments

in pupil size at any other lag (< 5 cells). Due to the sluggishness of pupil dynamics (particularly dilation), it remains unclear whether the temporal origin of these changes was 1) tonic changes in some underlying process that carried forward into trials t+1 and t+2, or 2) a phasic response of some underlying process on trial t that had effects on pupil size that were only observed later in time. Regardless, these findings strongly suggest that dACC activity specifically predicts future adjustments in pupil size, consistent with a causal role in regulating control states via arousal.

Spatial tuning does not explain putative conflict signals. Related to Figure 4.

In a previous study in the rhesus macaque [19], it was suggested that putative neuronal "conflict" signals merely reflect co-activation of distinct pools of neurons tuned to saccade direction on high-conflict trials. Although the authors of that study did not find evidence of action conflict signals in dACC, spatial selectivity for saccade direction has previously been reported for neurons in macaque dACC [6] and it thus remains possible that such spatial tuning could have contributed to the modest differences we observed in responses of dACC neurons to congruent and incongruent distractors.

Eighteen neurons showed a significant preference for either congruent or incongruent distractors, but the sign of this preference was mixed across the population. A simple account of co-activated spatially tuned pools of neurons could not explain this result because it would predict an increase in firing rates for incongruent trials and a decrease in firing rates for congruent trials [19]. Most importantly, only 2 neurons showed spatial tuning for saccade direction (as indexed by significant differences in responses to leftward and rightward saccades, in the absence of distractors; Wilcoxon rank sum test). This number was not significantly larger than would be expected by chance, and neither of these neurons preferred congruent or incongruent distractors. Thus, spatial selectivity for saccade target location cannot explain the preference of some neurons for incongruent or congruent stimuli in our study.

dACC firing rate during fixation predicts error likelihood. Related to Figure 3.

In addition to differing responses to distractors between the task and ITI, we observed a baseline shift in firing rate before distractor onset (main text, figure 3B). Therefore, we asked whether single neuron activity during fixation predicted any aspects of task performance, in particular, we looked at error likelihood.

We used generalized linear models to predict the log odds of error commission on the basis of firing rate in the first 300 ms of fixation. This model was run independently on each cell:

$$\log\left(\frac{p(error)}{1-p(error)}\right) = \beta_0 + \beta_1(FR)$$

A second generalized linear model (below) was fit to the population profile in figure S1B. To examine the population response, firing rates were divided into 8 quantile bins within each cell and the bin numbers were used as the firing rate regressor. The number of bins was chosen to minimize the number of empty bins within each cell. The mean error frequency within each cell and bin was normalized to the mean error frequency in that recording session.

$$errors = \beta_0 + \beta_1(FR) + \beta_2(FR^2)$$

Where “errors” is the normalized error frequency within that firing rate bin for that cell. We used AIC and BIC model comparison to determine that the quadratic model (AIC: 473.2; BIC: 482.4) better explained the data, compared to a linear model (AIC: 486.2; BIC: 490.8) and a model with an added cubed term (AIC: 475.2; BIC: 489.0). The linear model had an Akaike weight of less than 0.01, and a BIC model weight of less than 0.02, compared to the model with a squared term, indicating that omitting a term that allowed for a U-shaped relationship resulted in substantial information loss.

A modest population of individual dACC neurons predicted error likelihood during fixation, in advance of any information about the upcoming trial. Out of the whole population of recorded cells, 19 cells (20%). had significant predictive turning for error likelihood, assuming a simple linear relationship. The sign of the effects was mixed: approximately equivalent numbers of cells had positive and negative relationships between firing rate and error likelihood (positive: 11 cells, beta weights between 0.03 and 0.19; negative: 8 cells, beta weights between -0.05 and -0.12; all $p < 0.05$; example responses from two neurons are in figure S1A). Across the whole population of recorded neurons, the relationship between dACC firing rate and error likelihood was u-shaped (figure S1B).

Across the population, intermediate firing rates during fixation predicted reduced likelihood of error commission on that trial, relative to the within-session mean (post-hoc Wilcoxon zero median test, $p < 0.002$, $z(93) = -3.10$). This result suggests that anticipatory shifts in dACC activity can predict successful maintenance of task performance despite the presence of distraction.

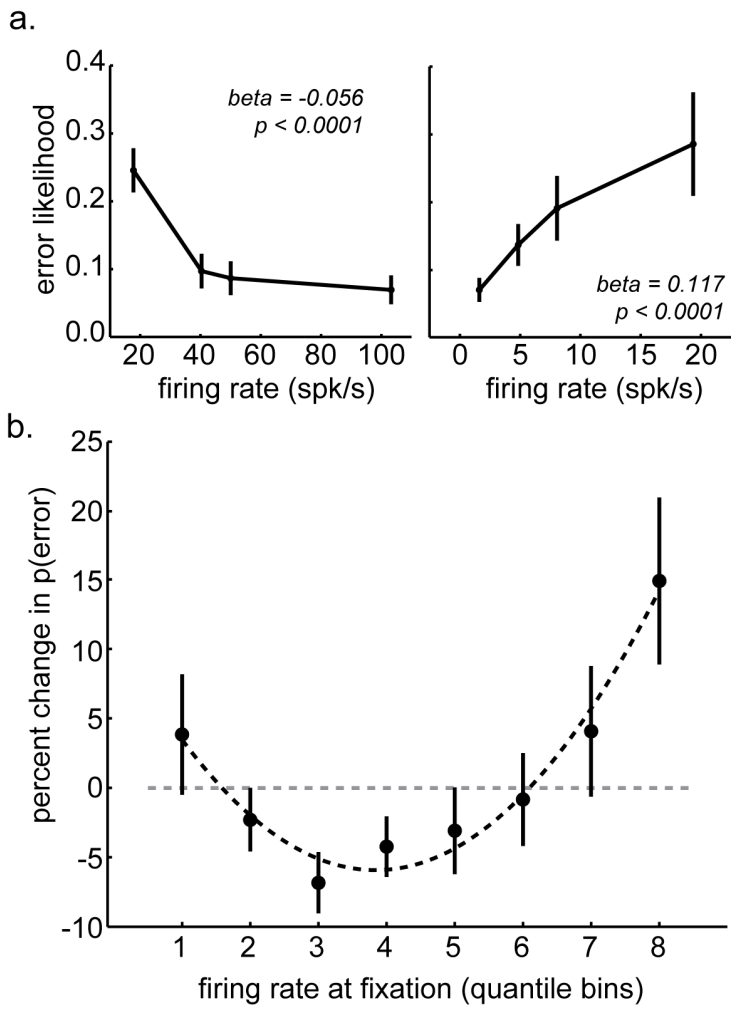

Figure S1, related to Figure 3: Firing rate during fixation predicts error likelihood. A) Individual neurons had largely linear relationships with error likelihood. Two representative cells are illustrated here. Approximately equal numbers of cells were decreasing (example in first panel) and increasing (example in second panel). Firing rate was divided into 4 quantile bins within each example neuron, the center of the bin is aligned to the mean firing rate within that bin. B) Across the population of recorded cells, dACC activity had a u-shaped relationship with error likelihood, normalized to the within-session probability of error commission (see Methods).

Previous-trial distractor type alone does not predict executive control on subsequent trials. Related to Figure 2.

In humans, executive control is typically enhanced following task conflict [10,11], an effect thought to be mediated by dACC activity. Therefore, in addition to examining the relationship between task distractors and task-facilitating

adjustments in pupil size, we also examined the effect of distractor type on response time and error likelihood.

First, we looked at whether previous-trial distractor type predicted response slowing, an index of increased executive control, by looking at response time on distractor-absent trials. Response time on distractor absent trials was not affected by distractor type on the previous trial (mean RT following a no-distractor trial = 0.1295; following a neutral distractor = 0.1291; following an incongruent or congruent distractor = 0.1301; $p > 0.45$ in all paired pair-wise t-tests).

Next, we looked at whether previous-trial distractor type predicted error likelihood on distractor-present trials. Again, there was no significant relationship (mean percent errors following a no-distractor trial = 0.1131; following a neutral distractor = 0.1138; following an incongruent or congruent distractor = 0.1098; $p > 0.52$ in all paired pair-wise t-tests). Thus, while distractor type predicted adjustments in pupil size that were associated with reduced distraction, distractor type did not directly predict changes in distractor state itself.

The pupil light response to distractors does not explain the change in baseline pupil size on subsequent trials. Related to Figure 2.

Because distractors were bright images, it remains possible that variation in pupil size due to a pupil light response (PLR) was the cause of differences in baseline pupil size on subsequent trials, rather than control processes or changes in autonomic arousal. This is an unlikely explanation because that the images were briefly flashed (for 67 ms) and baseline pupil size was measured several seconds later, after an intervening saccade, reward or error outcome, ITI, and fixation onset (mean latency = 4.0 s). However, we also addressed this possibility analytically by examining the correlations between the PLR and baseline pupil size on the next trial. We found very modest correlations between the two measures that were largely independent of the actual presence of a distractor, suggesting that the PLR was not the reason for the changes in the pupil we observed in the present experiment. Moreover, we found that controlling for any effect of a light response in our firing rate models did not change the results reported in the main text.

We measured PLRs to real distractors or sham distractors on each trial. We used the method in [29] and calculated PLR as the minimum pupil size in the 400-600 ms following distractor onset or a sham-distractor timestamp, and normalized this measure by dividing by pupil size in the 100 ms before distractor onset. Smaller numbers indicated a larger PLR but a transiently smaller pupil. We asked whether PLR predicted the magnitude of change in baseline pupil size from one trial to the next—the pupil measure most likely to be confounded by the PLR in our data. In the presence of distractors, PLRs did have a small, positive

relationship with pupil change—smaller magnitude PLR (larger physical pupils), predicted larger pupil size on the next trial, relative to pupil size on the current trial (Pearsons's linear correlation: mean R = 0.05, range = -0.17 to 0.19, $p < 0.05$ in 19/56 sessions; Spearman's rank correlation: mean rho = 0.07, range = -0.10 to 0.27, $p < 0.05$ in 22/56 sessions). However, we also observed similar correlations in the pupil "response" to sham distractors (Pearsons's linear correlation: mean R = 0.04, range = -0.22 to 0.32, $p < 0.05$ in 10/56 sessions; Spearman's rank correlation: mean rho = 0.08, range = -0.14 to 0.30, $p < 0.05$ in 10/56 sessions). These within-session correlations are plotted in figure S2A. Thus, any predictive power that pupil light "response" had for the change in pupil size on the next trial did not depend on the actual presence of a distractor, but was instead due to autocorrelations in pupil size, local pupil dynamics, or other processes beyond the scope of the current paper.

The pupil light response to distractors does not explain the relationship between dACC activity and changes in pupil size. Related to Figure 5.

Next, we directly asked whether pupil light responses explained the relationship between baseline pupil size or changes in pupil size and dACC neuron firing rates. We compared the results of our GLM analysis (main text equation 3) with and without an additional term to account for PLR. This analysis allowed us to determine whether the relationships between pupil size and dACC firing rate actually relied on an effect of PLRs on firing rate.

We observed similar numbers of significant cells for each term in the two models (compare 23 cells significant for distractor presence with PLR in the model, vs 24 cells in the original analysis; 49 neurons significant for baseline pupil size vs 52 neurons in the original analysis; 34 neurons significant for changes in baseline pupil size vs. 31 neurons in the original analysis). Critically, beta weights were statistically indistinguishable between the two models across cells (all $p > 0.5$, paired t-test; specific beta weights for the change in baseline pupil size term: $p > 0.8$, figure S2B). Thus, distractor-evoked PLRs could not explain the present observations.

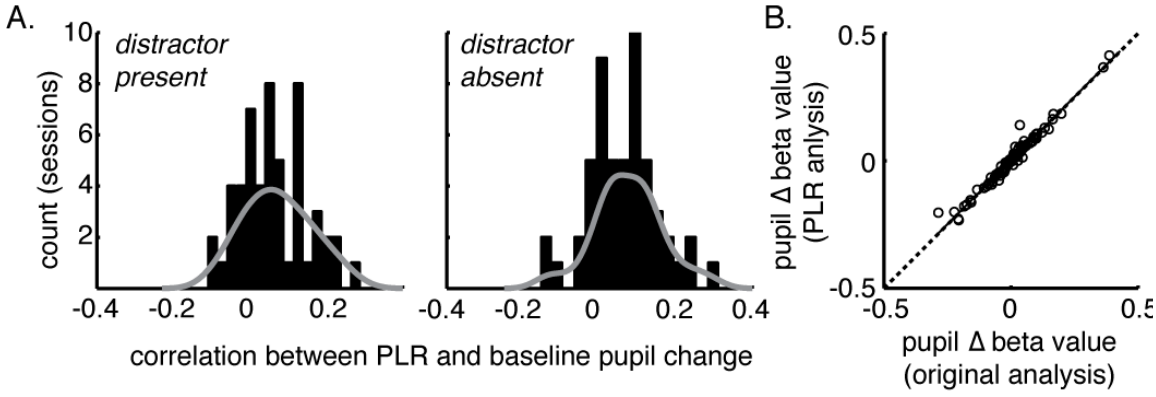

Figure S2, related to figures 2 and 5: The pupil light response (PLR) to distractors does not explain changes in baseline pupil size or associated dACC signals. A) The magnitude of correlations between PLRs and changes on pupil size on the next trial were both small (mean Spearman's rho = 0.07, Pearson's R = 0.05) and independent of the veridical presence of a distractor (illustrated: Spearman's rank correlation coefficients for each session). A nonparametric density estimate is overlaid in gray. B) The sign and magnitude of beta weights describing the relationship between dACC firing rate and changes in baseline pupil size were unchanged when we added a term to account for PLR magnitude to this analysis, indicating that PLR magnitude was not a better predictor of dACC firing rate than were adjustments in baseline pupil size.

Supplemental Experimental Procedures:

Surgical Procedures. All procedures were approved by the Institutional Animal Care and Use Committee at Duke University. Two male rhesus macaques participated in this experiment (H, Y). In order to allow eye position monitoring and electrophysiological recording, the monkeys were surgically-prepared with small head restraint prostheses, as described previously [68]. A recording chamber (Christ Instruments) was stereotaxically placed over the cingulate sulcus and its location was verified with MRI. Appropriate analgesics and antibiotics were delivered after all procedures. The recording chamber was maintained with regular sterile saline washes and sealed with a sterile cap between recording sessions. The monkeys were acclimated to the laboratory, to head restraint, and then trained to perform the task for liquid rewards.

Electrophysiological Recording. We recorded from single neurons in the dorsal bank, ventral bank, and fundus of dACC in two monkeys during task performance. Single electrodes (Frederick Haer) were lowered with a hydraulic microdrive (Kopf) into grid sites identified with MRI as located over the cingulate sulcus. Neuroimaging was performed at the Center for Advanced Magnetic Development at Duke University Medical Center, on a 3T Siemens Medical Systems Trio MR Imaging Instrument using 1 mm slices. In each session, we lowered the electrode until the waveform of single (1–3) neuron(s) was isolated at the MRI-determined depth. Individual action potentials were identified by standard criteria and isolated on a Plexon system. Isolations were confirmed both online and post-hoc through principal component analysis. Neurons were selected for recording on the basis of the quality of isolation only.

Behavioral Techniques. The animals were maintained on controlled access to fluids to motivate them to perform the task. Matlab (Psychtoolbox-3) was used to display stimuli and record eye data. Task stimuli were colored targets presented against a dark background on a 51 cm wide LCD monitor (60 Hz refresh rate, 1920 x 1080 resolution), located 60 cm in front of the monkey. Eye position and pupil size was monitored at 1000 Hz via an infrared eye tracking system (SR Research; Eyelink). The manufacturer's standard method for calculating pupil area was used. Trials in which blinks or any other occulsions of the pupil were detected during fixation were aborted.

Monkeys performed a simple, visually-guided saccade task, in which they first centrally fixated a 1° target (+/- 6° of error) for 450-650ms and then shifted gaze to an eccentric target (1° square, 14° offset) appearing on either the left or right of fixation. Fixation on the eccentric target (+/-6° of error) for 150ms-450ms resulted in a juice reward, which was constant for each monkey within sessions and ranged from 0.15mL to 0.35mL per trial. Inter-trial intervals (ITI) ranged from 1750 to 2500 ms.

On 75% of trials, a distractor image was briefly flashed (for 67 milliseconds) at one of three locations. Across all three distractor locations, the leading edge of distractors was 15° from fixation (compare target center at 14°), which ensured that the distractor never physically occluded any part of the 1° target. Distractor-to-target SOAs ranged from 50 ms before target onset to 100 ms after. The variable SOA ensured that the monkeys could not predict distractor timing and allowed sufficient temporal jitter to dissociate the distractor response from other signals such as target onset or saccades.

The monkeys had a 2 second window to fixate the target. Errors were due to early fixation breaks (before distractor onset), breaks of fixation after distractor onset, or failure to hold target fixation after entering the fixation window. The task was quite difficult for the monkeys, in order to allow a sufficient number of errors to analyze. The mean error rate across sessions was 12.6% (monkey H: 12.8% +/- 5.3% standard deviation; monkey Y: 12.3% +/- 4.0% standard deviation). Error trials were excluded from all analyses except for the analyses of error responses.

ITI distractors were presented during the middle of the ITI (875-1250 ms after ITI onset). The frequency of ITI distractors changed every 50 trials for the majority of recording sessions, but in some sessions was fixed at 10%. ITI distractors were presented in the same three locations as task distractors, 15° from the center of the screen.

Data analysis: Data was analyzed with custom software in MATLAB and R. Peristimulus time histograms (PSTHs) were constructed by aligning spike time rasters (1 ms resolution) to specific trial events and averaging firing rates across multiple trials within each 1 ms bin. When no task event was present for alignment (as in distractor absent trials aligned to distractor onset), sham event time stamps were generated by random sampling (with replacement) from the time stamps of actual event occurrences on event present trials. Multiple sham time stamps were generated for every event-absent trial in order to match sample sizes for event-present and event-absent conditions. For display, PSTHs were smoothed with a Gaussian filter (2 ms standard deviation). In all models where activity was collapsed across multiple cells, firing rates were rescaled between 0 and 1 within each cell and cell identity was coded as a dummy variable to account for variation in the mean response.

To identify distractor and error sensitive neurons, we used a bootstrapping method [7] on binned (1 ms bins) but otherwise unsmoothed data. Shuffled data sets were constructed by randomly resampling the observed firing rate data (with replacement) into the existing trial labels, thus simulating a distribution under the null hypothesis. Shuffled datasets were thus matched in size to the original datasets. 1000 shuffled datasets were generated for each cell. Sliding Wilcoxon rank-sum tests were then performed on both the shuffled and original data (100

ms bins, 10 ms steps). Within-cell significance thresholds were set to the number of continuous significant bins observed in less than 5% of shuffled datasets.

The structural equation model (illustrated graphically in figure 6) was based on a standard moderated-mediation approach [8], and fit using freely in freely available statistical software (lavaan: [9]). The model fit to the population of 18 neurons that had significant encoding of both distractors and adjustments in future pupil size. In order to account for variation between neurons, cell identity was dummy-coded and included in the error terms of the model. For completeness, the model also included the effect of current-trial baseline pupil size on firing rate and pupil adjustment respectively, as well as disturbance terms for all measured variables. These additional terms are omitted from the equation and graphical depiction of the model for clarity. The model was fit via maximum likelihood procedures with robust standard errors.

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