Electrophysiological correlates of default-mode processing in macaque posterior cingulate cortex

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During the course of daily activity, our level of engagement with the world varies on a moment-to-moment basis. Although these fluctuations in vigilance have critical consequences for our thoughts and actions, almost nothing is known about the neuronal substrates governing such dynamic variations in task engagement. We investigated the hypothesis that the posterior cingulate cortex (CGp), a region linked to default-mode processing by hemodynamic and metabolic measures, controls such variations. We recorded the activity of single neurons in CGp in 2 macaque monkeys performing simple tasks in which their behavior varied from vigilant to inattentive. We found that firing rates were reliably suppressed during task performance and returned to a higher resting baseline between trials. Importantly, higher firing rates predicted errors and slow behavioral responses, and were also observed during cued rest periods when monkeys were temporarily liberated from exteroceptive vigilance. These patterns of activity were not observed in the lateral intraparietal area, an area linked to the frontoparietal attention network. Our findings provide physiological confirmation that CGp mediates exteroceptive vigilance and are consistent with the idea that CGp is part of the "default network" of brain areas associated with control of task engagement.

default network | lateral intraparietal cortex | working memory | task engagement | attention

The neural mechanisms supporting our engagement with the outside world remain poorly understood. Many studies have implicated the activation of a dorsal frontoparietal network of brain regions in selective attention and the consequent benefits in reaction time and accuracy in task performance (1–3). Recent studies suggest that a complementary network of brain areas, known as the default network, is deactivated during elevated task engagement (4–7). The default network comprises several brain regions that show a high metabolic and hemodynamic activity at rest that is suppressed during goal-directed tasks (3–5, 7–11). A subset of these regions, including the posterior cingulate cortex and ventromedial prefrontal cortex, shows resting metabolic and hemodynamic activity that is significantly higher than the global mean (5–6, 9).

Deactivation of the default network has been implicated in attention, arousal, and task engagement. Increased hemodynamic response in the default network predicts occasional lapses in attention (12), failures to encode memories (13), and failures to perceive near-threshold somatosensory stimuli (14). Variations in the activity of the default network have been linked to self-directed cognition (4, 15), episodic memory retrieval (13), environmental monitoring (16), and motivated behavior (11). These data suggest that the default network may track moment-to-moment variations in the balance of exteroceptive vigilance and interoceptive cognition.

Despite these observations, the precise contribution of the default network, and the posterior cingulate cortex (CGp) specifically, to cognition remains hotly debated for several reasons. First, the relatively sluggish hemodynamic response underlying the blood-oxygen-level-dependent (BOLD) signal obscures information about moment-to-moment variations in neuronal activity within the default network (6, 17). Spiking activity varies over tens of milliseconds, and it remains unclear how slow hemodynamic changes in the default network map onto faster spiking events. Second, there is currently no evidence that single-unit responses in any brain area behave analogously to the BOLD signal in the default network, despite the fact that it is highly unlikely that the changes associated with the default mode are limited to hemodynamics. Thus, understanding the neuronal correlates of the default state can provide an important foundation for understanding its contribution to behavior and cognition.

We addressed these questions by recording the firing rates of single neurons and multiunits, as well as local field potentials (LFPs), in CGp, a canonical default network area, during performance of 2 attention-demanding tasks as well as at rest. For comparison, we also recorded the same measures of neuronal activity in the lateral intraparietal area (LIP), an area within the dorsal frontoparietal attention network lying outside of the default network (3), during the same tasks. We predicted that if CGp and, by extension, the default network, actively contributes to the balance of exteroceptive vigilance and interoceptive cognition, that the actual spiking of individual neurons, as well as multiunits, in this area would vary inversely with task engagement and mental effort. Conversely, we predicted that neuronal activity in LIP, a canonical frontoparietal attention area, would not do so.

We found that firing rates of CGp neurons were elevated at rest and suppressed during task performance, and that spontaneous firing rates predicted behavioral indices of task engagement on a trial-by-trial basis—with higher firing rates associated with poorer performance. Finally, cued rest periods in which monkeys were temporarily liberated from exteroceptive vigilance evoked the highest activity. Importantly, LFP in the gamma band, which has been closely linked to synaptic activity and, by extension, the BOLD fMRI signal (17, 18), was also suppressed by active task performance. These patterns of activity were not observed in LIP. Our data therefore provide previously undescribed physiological evidence to support the hypothesis that the firing rates of single neurons in CGp and, by extension, the default network, reflect the degree of task engagement and, moreover, that these relationships are not restricted to humans.

Results

Single CGp Neurons Show High Baseline Activity That Is Suppressed During Task Performance. Task-related deactivations in hemodynamic and metabolic activity are a hallmark of the default network in humans (4). Therefore, we first asked whether task-related suppression occurs in the activity of single CGp neurons of 2 monkeys performing 2 tasks (Fig. 1*A*). In the attentive task, monkeys actively maintained fixation on a central point while

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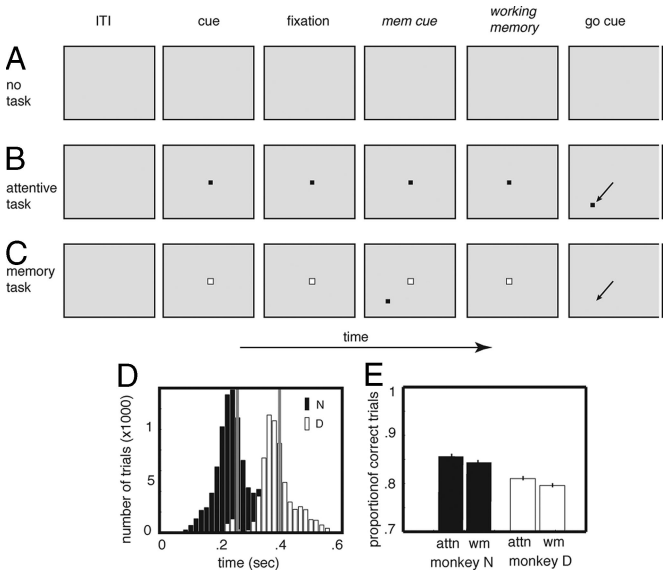

Fig. 1. Tasks and behavioral performance. (*A–C*) Schematic of tasks. On each task trial, a central cue was illuminated, which the monkey fixated. After a delay, the cue changed color, and the monkey maintained fixation for 4 s (monkey N) or 3 s (monkey D). In the working memory task (*C*), an eccentric cue appeared during the delay and remained illuminated for 1 second. Following the fixation period, the cue was extinguished, and in the attentive task (*B*), an eccentric target appeared at 1 of 36 locations from a grid surrounding the central cue. Monkeys received a reward for shifting gaze to eccentric target or the remembered location. In the no-task condition (*A*), no targets appeared, and no reward was given. On timeout trials, the central cue appeared, but it did not change color, no peripheral target appeared, no behavioral response was required, and no reward was given. Gray lines indicate means of distributions. (*D*) Plot of reaction times for monkey N (black) and monkey D (white). Bars indicate median reaction time. (*E*) Correct trial performance of monkey N (black) and monkey D (white) on all recording days. attn indicates attention task; wm, working memory. Bars indicate one standard error.

waiting for the appearance of a peripheral target at an unpredictable location after a fixed delay (4 s in monkey N; 3 s in monkey D). Monkeys were rewarded for an immediate gaze shift (<500 ms) to the target. In the working memory task, the peripheral target appeared for 1 second after a 2-second delay (1 second, monkey D), and then it disappeared while fixation was maintained for another second. Performance on both of these tasks was not perfect (<90% for both monkeys), suggesting that both tasks were attentionally demanding. Because previous neuroimaging studies of default processing in humans have used fixation as a passive task, it is worth noting that in contrast to these studies, monkeys actively attended so as to detect the appearance of the target at an unpredictable location and execute a gaze shift within a limited time window. The monkeys' poorer performance on the working memory task ($P < 0.01$ for both monkeys individually, Student's t test on percent of correct trials per day) indicated that it was more difficult than the attentive task.

We also examined activity in a no-task condition, in which the monitor remained blank, no fixation was required, and no reward was given. The duration of the no-task condition was identical to that of the 2 active tasks (4 s in monkey N and 3 s in monkey D). The no-task condition provided a control with timing as close as possible to the tasks. The no-task condition, the attentive task, and the working memory task were randomly interleaved on a trial-by-trial basis. We compared responses in these conditions to responses in the intertrial interval (ITI), defined for analysis as a 1-second epoch beginning 2 s before the trial.

We recorded single-unit activity (SUA) from 127 isolated neurons in CGp (83 in monkey N and 44 in monkey D). We found that the firing rates of single CGp neurons were reliably suppressed during the attentive task compared with the ITI (Fig. 2 *A* and *B*). In this and most other analyses, the task epoch began 500 ms after fixation was acquired and extended until 500 ms before the saccade (3 s in monkey N; 2 s in monkey D). In our example neuron, firing rates in the 2 tasks were 7.1% (0.83 spikes per second) lower than those in the ITI ($P < 0.005$, Student's t test on average modulations for each neuron individually; Fig. 2*A*). This reduction in firing was 6.8% of baseline ($P < 0.005$, Student's t test). In the population, task-related suppression was 6.3% (0.73 spikes per second) for the attentive task and 7.9% (0.92 spikes per second) for the working memory task (Fig. 2*B*). To derive an unbiased estimate of neuronal activity in CGp, we did not exclude any neurons from this analysis. Significant suppression was observed in 35% of neurons (44 of 127) for the 2 tasks ($P < 0.05$), whereas significant enhancement was observed in 9.4% of neurons (12 of 127; $P < 0.05$). This distribution of modulations in the sample of neurons was significantly biased toward neurons with suppressive effects (χ^2 test, $P < 0.001$). Significant task-related suppression was also observed in both monkeys individually (32 of 83 cells in monkey N, and 12 of 44 cells in monkey D; $P < 0.05$, χ^2 test).

We also compared activity in the 2 tasks to that in the no-task condition. Here, activity was no different (0.7%, 0.09 spikes per second) from during the ITI ($P = 0.45$, Student's t test on average modulations for each neuron individually). Neuronal responses during the attentive task were 6.4% lower than during the no-task condition (reduction was 0.74 spikes per second; $P < 0.003$, Student's t test). Neuronal responses in the working memory task were 8.0% lower than during the no-task condition (0.94 spikes per second; $P < 0.001$, Student's t test). This reduction in firing rate seems unlikely to reflect a reduction in visual scanning, because firing rates of CGp neurons are not modulated by free viewing in the absence of specific task goals (see *SI Results*). These results demonstrate that performing an attentionally demanding task reduces neuronal activity in CGp.

Tonic Suppression of Neuronal Activity Alternates with Phasic Excitation in CGp. Across the population, a significant phasic response to the cue during the working memory task (4% enhancement relative to previous 1-second epoch, 0.4 spikes per second; $P = 0.026$, Student's t test on modulations for individual neurons) was observed in 44.8% of neurons ($P < 0.05$, Student's t test on single-trial responses; Fig. 2*A*). During the memory epoch, neuronal activity was suppressed relative to the attentive task during the corresponding time period (0.28 spikes per second; $P < 0.02$, Student's t test) in 38% of neurons ($P < 0.05$, Student's t test on single-trial responses). This overall suppression of CGp activity during active maintenance of a remembered saccade target distinguishes CGp from other areas, including lateral prefrontal cortex (19), LIP (20), and mediodorsal nucleus (21), which exhibit enhanced activity during working memory delays.

CGp neurons exhibited distinct multisecond tonic and subsecond phasic patterns of activity during task performance. Tonic activity—the main focus of the present paper—was reliably suppressed during the attentive task. Phasic enhancements were associated with important trial events, including fixation, target presentation, saccade onset, and reward delivery (22–24). Although we observed some directional selectivity in these responses, these signals have been reported previously (23), so they are not further studied here. The phasic enhancement at the beginning of the trial shows that neuronal activity in CGp is not simply and immediately extinguished by task engagement (cf. 25).

If the tonic and phasic response domains are independent, they may have different functions, and they may reflect distinct inputs; conversely, if they are related, they may reflect similar or interacting influences. To investigate these possibilities, we compared the average size of the tonic suppression during the 2 tasks to the size of the phasic enhancement observed during both the initial saccade

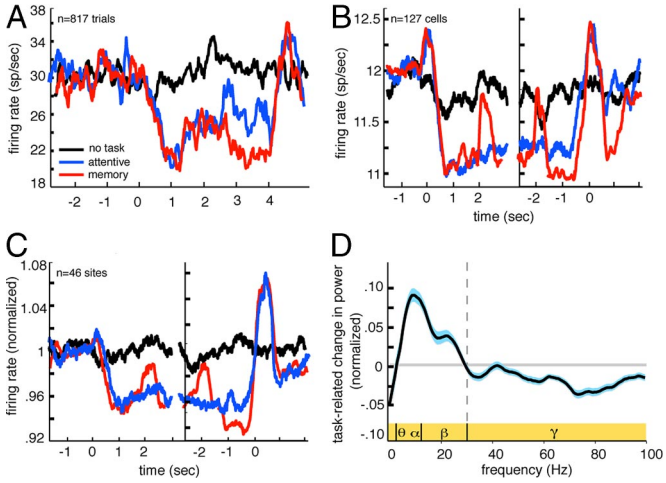

Fig. 2. Attentive vigilance suppresses activity of CGp neurons. (*A*) Peristimulus time histograms (PSTHs) show average firing rates of a single CGp neuron during attentive task (blue line), working memory task (red line), and no-task condition (black line). Responses are aligned to cue fixation. Firing rate was suppressed during the tasks, although responses were phasically enhanced at the beginning and end of trials. sp/sec indicates spikes per second. (*B*) PSTHs showing average firing rates of all CGp neurons in the population (n = 127). Late portion of neural response is aligned to acquisition of target. Conventions as in *A*. (*C*) PSTHs showing average MUA at all CGp sites in the population (n = 43). Conventions as in *B*. (*D*) Differential power spectra of LFPs for attentive task minus control condition. Vertical axis indicates proportional difference in power between the 2 tasks for all neurons (normalized). Power in the gamma band was suppressed relative to ITI, whereas power in lower-frequency bands was enhanced.

that began the trial and the saccade that resulted in reward. As in other analyses, we defined the tonic epoch as a 3-second epoch (2 s for monkey D) beginning 500 ms after the fixation spot was acquired, and the phasic epoch as a 500-ms epoch beginning 250 ms before the end of the saccade and ending 250 ms after the end of the saccade. We repeated these analyses using a 200-ms postsaccadic epoch beginning 200 ms after movement offset (cf. 23, 24). We observed no correlations between tonic and phasic responses in all neurons (perisaccadic epoch: $r = 0.072$, $P > 0.5$; postsaccadic epoch: $r = -0.003$, $P > 0.5$) or in the subset of neurons exhibiting a significant suppression effect associated with active fixation (perisaccadic epoch: $r = 0.061$, $P > 0.5$, correlation test; postsaccadic epoch: $r = -0.02$, $P > 0.5$). Consistent with these observations, we found that those neurons exhibiting a significant tonic suppression (44 of 127 neurons) were no more likely than chance to exhibit a phasic enhancement in either epoch (perisaccadic epoch: 57 of 127 neurons; correlation test, $P > 0.5$; postsaccadic epoch: 54 of 127). We also found that the size of the neuronal responses in these epochs was not correlated on a trial-by-trial basis for single neurons (average correlation of 0.02 for the perisaccadic epoch and 0.02 for the postsaccadic epoch; $P > 0.5$, correlation test). Indeed, a significant correlation was observed in ≈5% of neurons [7 (5.5%) of 127 for perisaccadic epoch, and 3 (3.1%) of 127 neurons for postsaccadic epoch; $P < 0.05$, correlation test in both cases], which is not significantly different from chance (χ^2 test, $P > 0.05$). Collectively, the results of these analyses suggest that the tonic and phasic responses of CGp neurons are independent. We acknowledge that these data are not definitive, and future studies will be needed to fully characterize the relationship between tonic and phasic domains of the neuronal response in CGp.

Multiunit Activity (MUA) and LFPs in CGp Also Show Suppression During Task Performance. A few studies have shown close correspondence between BOLD and SUA, MUA, and high-frequency LFPs (gamma band) in the sensory cortex (18, 26–29). However, these measures are sometimes uncorrelated in other brain areas (6, 30), making it difficult to infer neuronal activity within any particular area directly from BOLD signals. We thus investigated the relationship between SUA, MUA, and LFP. At a subset of our recording sites (n = 43), we recorded MUA and LFPs.

MUA was suppressed by 5.1% ($P = 0.001$, Student's t test) during the tasks and by 1.29% during the no-task condition ($P = 0.047$, Student's t test). During the tasks, MUA was weaker than in the no-task condition ($P = 0.01$, Student's t test on modulations for individual neurons, Fig. 2*C*). Moreover, LFP power in the gamma band (30–100 Hz) was suppressed by 2.4% ($P < 0.005$, Student's t test) in the attentive task compared with no task. [We excluded data from the working memory task from this analysis because working memory may evoke LFP changes independent of task difficulty (Fig. S1).]

We also observed a general enhancement of LFP power at lower frequencies (4.2% enhancement; $P < 0.005$, Student's t test). Given the likely suppression of BOLD activity in this task based on prior observations, our data suggest that neuronal activity in CGp closely mirrors BOLD signals in the same area (18, 26–29). These observations are consistent with the hypothesis that the BOLD signal is tightly coupled with neuronal activity in CGp and demonstrate the generality of default processing across multiple measures of neuronal activity.

Neuronal Activity in Area LIP Is Not Suppressed by Task Performance. We recorded neuronal activity from 54 neurons and MUA from 28 sites in area LIP in the same 2 monkeys (Fig. 3). Neurons in CGp and LIP were not recorded simultaneously. We hypothesized that the activity would not be suppressed during task performance (9, 31). We found that the activity of LIP neurons during the task epoch was enhanced by 4.1% (0.97 spikes per second; $P < 0.005$, Student's t test on modulations for individual neurons; Fig. 3*B*) during the 2 tasks. This enhancement was not a consequence of working memory, because it was observed in the attentive task, which had no working memory component (activity was enhanced by 4.02% in the attentive task, 0.93 spikes per second; $P < 0.0075$, Student's t test). We found no evidence of task-related suppression of LIP neurons during the memory epoch (enhancement <1%, 0.1 spikes per second; $P = 0.92$, Student's t test) of the working memory task. Enhancement was observed in both monkeys individually (1.45 spikes per second in monkey N, and 0.66 spikes per second in monkey D; $P < 0.05$ in both cases). Responses in 20% of LIP neurons (11 of 54) were enhanced, whereas responses in 7.4% (4 of 54) were suppressed. Neuronal activity in LIP during the no-task condition was enhanced (3.26%, 0.75 spikes per second; $P = 0.0137$). These results demonstrate that responses of LIP neurons are qualitatively different from those in CGp (Fig. S2).

Neuronal Activity in CGp Predicts Task Engagement. We next probed the relationship between neuronal activity and 2 measures of task engagement: reaction time and errors. For the attentive task, median reaction times for the 2 monkeys (N and D) were 0.29 s (95% were between 0.22 and 0.41 s) and 0.39 s (95% were between 0.309 and 0.55 s). For the working memory task, median reaction times for the 2 monkeys (N and D) were 0.36 s (95% were between 0.27 and 0.49 s) and 0.43 s (95% were between 0.34 and 0.5 s). Reaction times thus indicate that the working memory task was more difficult than the attentive task. To investigate the relationship between firing rate and reaction time, we separately examined firing rates on fast and slow-reaction time trials. We defined fast-reaction time trials as those in which reaction times were faster than the median reaction time, and slow-reaction time trials as those in which reaction times were slower than the median reaction time.

We found that firing rates on faster trials were 5.8% (0.68 spikes per second; $P = 0.001$, Student's t test on modulations for individual neurons; Fig. 4*A*) lower than firing rates on slower trials during the task epoch (Fig. 4*A*). We found a significant positive correlation

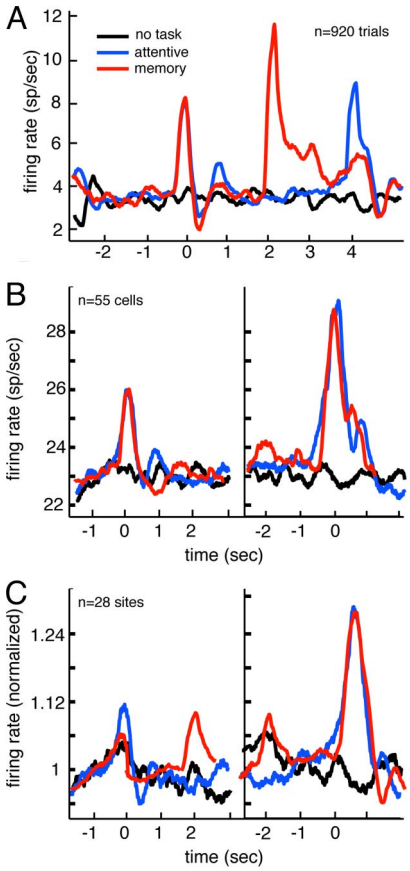

Fig. 3. Attentive vigilance enhances activity of LIP neurons. (*A*) PSTHs showing average firing rates of a single LIP neuron during attentive task (blue line), working memory task (red line), and no-task condition (black line). Responses are aligned to cue fixation. Firing rate of neuron was not suppressed relative to the ITI, and neuron showed tonic enhancements aligned to trial beginning and end. (*B*) PSTHs showing average firing rates of all LIP neurons in the population ($n = 54$). Responses were slightly enhanced relative to baseline. Late part of response is aligned to acquisition of target. (*C*) PSTHs showing average MUA of all LIP sites in the population ($n = 28$). Conventions as in *B*. sp/sec indicates spikes per second.

between the firing rates of CGp neurons and reaction time in a substantial number of cells [38 (30%) of 127; $P < 0.05$, bootstrap correlation test; Fig. 4*B*]. We found significant negative correlations in 11% of cells. We observed a significant correlation between firing rate and reaction time for the 2 monkeys individually (firing rate was 6.7% greater on slower trials for monkey N and 4.9% greater on slower trials for monkey D; $P < 0.01$ in both cases, Student's t test on modulations for individual neurons). For more data on individual monkeys, please see Fig. S3 and Tables S1–S5. It is notable that the difference in firing rate associated with reaction times began before the beginning of the trial (5.2%, 0.608 spikes per second; $P < 0.005$). In other words, the level of task engagement, as indexed by reaction time, is predictable based on spontaneous activity recorded in CGp before the trial even begins. This observation suggests that neuronal correlates of reaction time observed in CGp reflect behavioral state variables, such as arousal, attentiveness, or task engagement, that arise long before task onset.

Monkeys made errors by prematurely looking away from the fixation point on about 15% of trials (15.9% of trials for monkey N and 18.3% of trials for monkey D; Fig. 1*C*). On error trials, average neuronal activity within CGp (excluding activity within the last half of a second before the error occurred) was 10.3% greater than on correct trials (1.2 spikes per second; $P < 0.001$, Student's t test; Fig. 4 *C* and *D*). Before the beginning of the trial, firing rates on error trials were 3.5% greater than on correct trials (0.41 spikes per second; $P < 0.01$, Student's t test on modulations for individual neurons).

CGp Neuronal Activity Is Enhanced by Cued Task Disengagement. Finally, we assessed whether we could enhance activity within CGp through a behavioral manipulation (Fig. 4 *E* and *F*). We hypothesized that providing a reliable signal that no trial would occur for a specific period would reduce task vigilance, thereby enhancing activity in CGp (16). We examined neuronal responses in CGp during signaled breaks ("timeout condition": 23 CGp neurons in monkey N and 7 in monkey D) in which a small central cue indicated that the next trial would not begin for 4 s (3 s in monkey D). Tonic firing rates were significantly greater in the timeout than during the no-task condition (5.9%, 0.64 spikes per second; $P = 0.012$, Student's t test on modulations for individual neurons). These effects were significant for each monkey individually ($P < 0.05$ in both cases, Student's t test). Eye movements did not differ during the timeout and no-task conditions (mean saccade frequency and amplitude did not differ significantly; $P = 0.7$, Student's t test). Collectively, these results indicate that activity within CGp is not simply "on" or "off," but instead varies along a continuum of exteroceptive vigilance (Fig. 5 *A* and *B*). In contrast, neuronal responses in LIP did not track levels of task engagement (Fig. 5*C*).

Discussion

The CGp, along with adjacent precuneus, consumes more glucose than any other cortical region in humans (4, 32). Despite its metabolic demands, the function of this large brain region has long remained mysterious (33). Here, we show that firing rates of CGp neurons track levels of task engagement, thus endorsing the idea that CGp mediates cognitive processes that compete directly with externally directed cognition, including efficient performance of laboratory tasks. These results are somewhat unique in that they implicate CGp in governing a subject's engagement in a variety of tasks rather than proposing that CGp plays a particular role in a single cognitive function, such as working memory or attention (see also ref. 34). Although we believe that CGp does indeed perform such functions (23, 24), we suspect that these 2 roles are somewhat independent.

Specifically, we conjecture that degrees of task engagement are reflected in slow, long-lasting changes in neural activity (as shown in this report), whereas specific cognitive functions, such as action and perception, are reflected in short, phasic changes in activity (as shown in our previous reports, including refs. 22–24). Indeed, we find here that the activity of CGp neurons is phasically enhanced during and after important task events, such as the beginning and end of the trial, but is tonically suppressed during periods of attentive fixation. Our data suggest, but do not prove definitively, that the tonic and phasic response domains are independent, both within and across individual neurons. Given these findings, we infer that the size of the phasic modulations observed in this and previous studies does not reflect liberation from exteroceptive vigilance, although relatively slow and long-lasting postreward enhancements may reflect a return to the higher firing rate default state (22–24). We also hypothesize that downstream neurons can successfully filter the low- and high-frequency components of these signals. The present results suggest that metabolic and hemodynamic responses associated with default processing correspond more closely with tonic than with phasic modulations in neuronal activity. In any case, these data demonstrate the complementary contribution that high-temporal resolution physiological recordings can make to neuroimaging studies of default-mode processing.

Two important caveats accompany the present data. First, rewards were presented in the attentional and working memory tasks but were not presented in the ITI condition, the no-task condition, and the timeout condition. Thus, firing rate modulations between, but not within, these 2 classes of task may reflect, in part, the

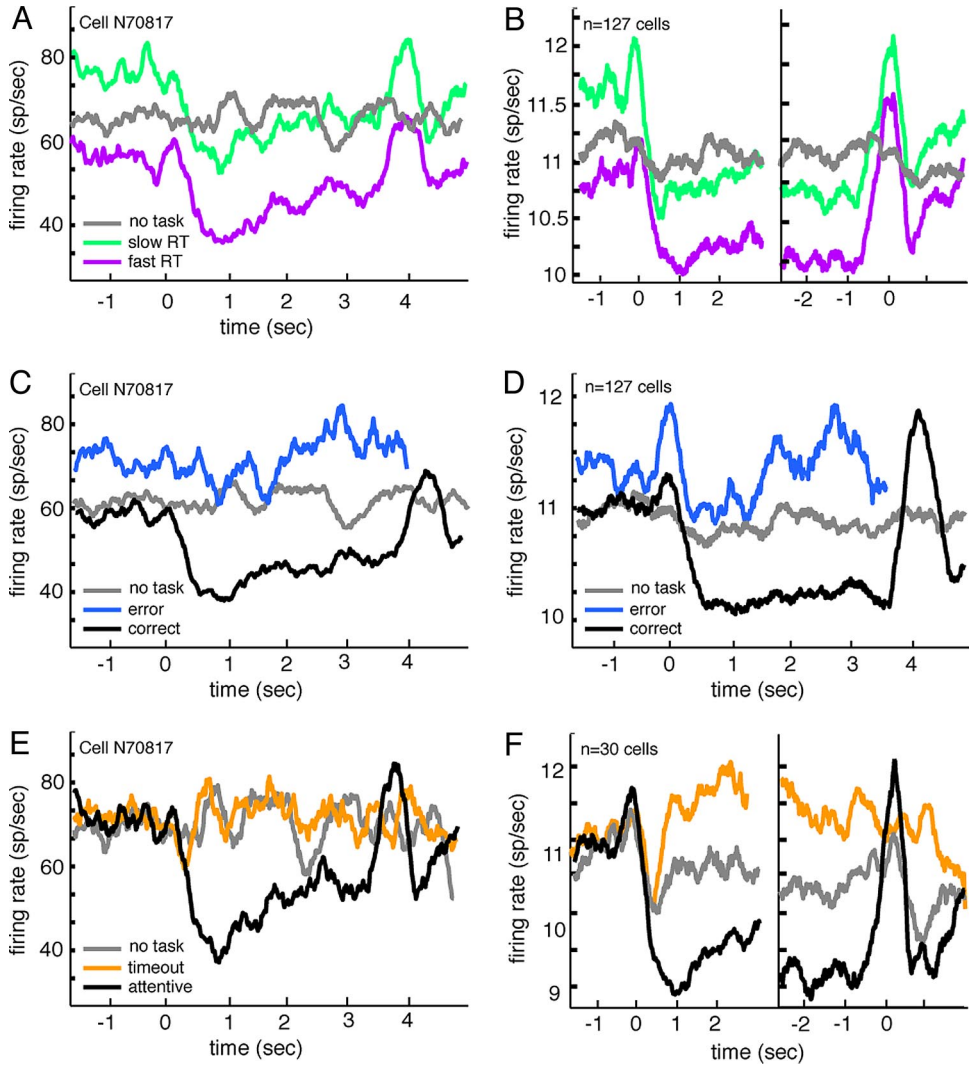

Fig. 4. Neuronal activity in CGp tracks level of task engagement. (*A* and *B*). PSTHs showing average firing rates of a single CGp neuron (*A*) and the CGp population (*B*) on slow (reaction time > median; green line) and fast (reaction time < median; purple line) trials. Gray line indicates firing rates in the no-task condition. Single-neuron and population responses were lower during trial and before trial onset, when subsequent reaction times were faster. Early part of response is aligned to acquisition of fixation. Late part of response is aligned to acquisition of target (*B* only). (*C* and *D*) PSTHs showing average firing rates of a single CGp neuron (*C*) and the neuronal population (*D*) on error (blue line) and correct (black line) trials of attentive task, and during the no-task condition (gray line). To eliminate any potential perisaccadic or phasic error signals, responses on error trials are truncated 500 ms before error. The difference in neuronal activity emerged before trial onset. (*E* and *F*) PSTHs showing average firing rates of a single CGp neuron (*E*) and all CGp neurons (*F*) in which timeout condition was tested ($n = 30$ cells). When cue indicated that the next trial would not begin for 3–4 s (timeout; orange line), neuronal activity in CGp was significantly enhanced relative to tasks (black line) and the no-task condition (gray line). sp/sec indicates spikes per second.

expectation of reward. (In fact, such an expectation may be one factor that distinguishes interoceptive from exteroceptive processing.) By similar logic, eye movements were controlled in the attentive and working memory tasks but were not controlled in the ITI condition, the no-task condition, and the timeout condition. Thus, eye movements may contribute to firing rate differences between conditions. However, we observed no modulation in firing during free viewing when responses were aligned to saccades (*SI Results*), suggesting that perisaccadic firing does not strongly contribute to neuronal responses in CGp. Moreover, this concern does not apply to the task engagement effects (Fig. 4), in which eye movements were matched in all comparisons.

Our findings support, and build upon, studies showing that hemodynamic activity in CGp is increased during lapses in attention and failures to perceive and encode environmental stimuli (12–14). Moreover, these results show that default effects correspond to spiking activity of CGp neurons, and not just synaptic responses reflecting inputs to CGp (cf. 17, 18). By showing that such effects correspond to the activity of single neurons, and by showing the rapid changes in firing rates associated with task performance, our results significantly advance functional understanding of CGp and, by extension, the default network. More fundamentally, these data confirm the idea that metabolic and hemodynamic changes associated with default processing reflect underlying neurophysiological events and confirm that the default network is homologous in humans and monkeys (8).

If function is indeed conserved between humans and monkeys, then the defining functional property of the default network cannot be a uniquely human cognitive process. Thus, either cognitive functions, such as self-awareness, introspection, or theory of mind—which have been attributed to the default network—are not uniquely human (4), or the default network plays a more fundamental role in basic cognitive processes that are usually suppressed during focused task performance. Such processes may include

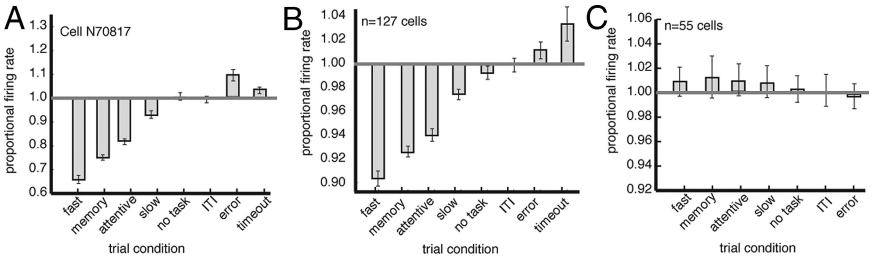

Fig. 5. Neuronal activity in CGp covaries with exteroceptive vigilance. Bar graphs showing average normalized firing rate of a single example CGp neuron (*A*) and the studied population of CGp neurons (*B*) in each of the conditions described above. (*C*) In LIP, responses do not track measures of task engagement.

retrieval of information from memory, monitoring the internal milieu, and global receptiveness to the gamut of information available in the local environment. Nonetheless, subsequent studies with tasks designed to parametrically manipulate these processes will be needed to address these questions (4, 15). Our findings suggest the clear utility of future studies designed to identify the precise contributions of single units, both within and outside of the default network, to cognitive processing.

Materials and Methods

Surgical and Behavioral Procedures. Standard surgical and behavioral procedures were used (see *SI Results* for details). All procedures were approved by the Duke University Institutional Animal Care and Use Committee and were designed and conducted in compliance with the Public Health Service's Guide for the Care and Use of Animals. Eye positions were sampled at 1,000 Hz by an infrared eye-monitoring camera system (SR Research). Visual stimuli were small, colored squares on a computer monitor placed directly in front of the animal and centered on his eyes. A standard solenoid valve controlled the duration of juice delivery. Reward volume was 0.2 mL in all cases.

In the attentive and working memory tasks, the trial began with the appearance of a small, yellow, fixation square. The square then changed color to indicate the nature of the task (red for memory, green for attentive, and remaining yellow for timeout). The square remained on for 4 s (monkey N) or 3 s (monkey D) and was then extinguished, signaling a saccade. In the working memory task, an eccentric cue appeared after 2 s of fixation (monkey N) or 1 second (monkey D). The cue remained illuminated for 1 second and then disappeared. In the attentive task, an eccentric cue appeared at the end of the delay, and the monkey was rewarded for shifting its gaze to it as quickly as possible. In the working memory task, no eccentric cue appeared, and the monkey had to shift its gaze to the remembered location. A fluid reward was given following successful completion of either task. In the timeout condition, no other stimuli appeared, and no reward was given. ITIs were fixed at 3 s in all cases.

Microelectrode Recording Techniques. Single electrodes (Frederick Haer Co.) were lowered under microdrive guidance (Kopf) until the waveforms of 1–4 single neuron(s) were isolated. Individual action potentials were identified by their unique waveforms and isolated on a Plexon system (Plexon Inc.). Neurons were selected for recording on the basis of the quality of isolation only. In all cases, neurons were considered isolated only if their waveforms were distinct from those of other neurons and background hash. Ultrasound images taken in the sagittal plane confirmed that the CGp recordings were made in areas 23 and 31 in the cingulate gyrus and ventral bank of the cingulate sulcus. This method has been used to confirm the positioning of CGp in several earlier studies in our lab (23, 24).

MUA was defined as nondiscriminable waveforms that crossed an arbitrary threshold. As such, absolute firing rates for MUA have no meaning, but temporal dynamics do. SUA was collected at every site, but MUA was not collected at every site. LFPs were collected by using the Plexon recording system from the same electrode. Data from all traces were analyzed; no posthoc selection of units occurred.

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