1 EEG signals index a global signature of arousal embedded in neuronal 2 population recordings

3

4 Richard Johnston^{1,2,3}, Adam C. Snyder^{4,5,6}, Rachel S. Schibler⁷, and Matthew A. Smith^{1,2,3}

5

6 ¹Department of Biomedical Engineering, Carnegie Mellon University, Pittsburgh, USA;

7 ²Neuroscience Institute, Carnegie Mellon University, Pittsburgh, USA; ³Center for the Neural Basis

8 of Cognition, Carnegie Mellon University, Pittsburgh, USA; ⁴Department of Brain and Cognitive

9 Sciences, University of Rochester, Rochester, USA; ⁵Department of Neuroscience, University of

10 Rochester, Rochester, USA; ⁶Center for Visual Science, University of Rochester, Rochester, USA; ⁷

11 Harvey Mudd College, California, USA.

12

13 Corresponding author:

14 Matthew A. Smith, PhD

15 Carnegie Mellon University

16 Department of Biomedical Engineering and Neuroscience Institute

17 Mellon Institute, Room 115

18 Pittsburgh

19 Pa, 15213

20 Email: mattsmith@cmu.edu

21

22 Acknowledgements:

23 M.A.S. was supported by NIH Grants R01 EY022928, R01 MH118929, R01 EB026953, and P30

24 EY008098; NSF Grant NCS 1734901; a career development grant and an unrestricted award from

25 Research to Prevent Blindness; and the Eye and Ear Foundation of Pittsburgh. A.C.S. was supported

26 by NIH grant K99 EY025768. R.S.S. was supported by NIH Grant R90 DA023426. The authors

27 would like to thank Ms. Samantha Schmitt for assistance with surgery and data collection.

28

29

30

31 **Abstract**

32 Electroencephalography (EEG) has long been used to index brain states, from early studies
 33 describing activity during visual stimulation to modern work employing complex perceptual tasks.
 34 These studies shed light on brain-wide signals but lacked explanatory power at the single neuron
 35 level. Similarly, single neuron studies can suffer from inability to measure brain-wide signals. Here,
 36 we combined these techniques while monkeys performed a change detection task and discovered a
 37 link between EEG and a signal embedded in spiking responses. This ‘slow drift’ was associated with
 38 arousal: decreases in pre-stimulus α power/increases in P1 amplitude were accompanied by :1)
 39 increases in pupil size, false alarm rate and saccade velocity; and 2) decreases in microsaccade rate
 40 and reaction time. These results show that brain-wide EEG signals can be used to index modes of
 41 activity acquired from direct neural recordings, that in turn reflect global changes in brain state that
 42 influence perception and behavior.

44 **Introduction**

45 For decades, researchers have investigated how the spiking responses of single cortical
 46 neurons relate to performance on decision-making [1], attention [2] and working memory [3] tasks.
 47 Interactions between pairs of neurons have also been studied extensively since technological
 48 advances in neural recording systems (e.g. microelectrode arrays and two-photon imaging) made it
 49 possible to monitor the activity of neural populations simultaneously [4–6]. At the same time, it is
 50 becoming increasingly apparent that major insight about the neurobiological basis of cognition can
 51 be gained if one goes beyond studying single and pairwise statistics [7–14]. Furthermore, it has been
 52 suggested that low-dimensional neural activity patterns can be used to index global brain states, which
 53 influence performance on cognitive tasks. For example, Stringer et al. [15] applied principal
 54 component analysis (PCA) to data recorded from more than 10,000 neurons in the mouse and found
 55 that fluctuations in the first principal component were associated with a host of arousal-related
 56 variables including whisking, pupil size, and running speed. Musall et al. [16] found that uninstructed

57 movements, which themselves may occur at varying frequency based on arousal, were related to
 58 brain-wide activity in the mouse. In our own work in macaque monkeys, we have reported a brain
 59 wide ‘slow drift’ of neural activity [17] which is correlated with a distinctive pattern of eye metrics
 60 that is strongly indicative of changes in arousal [18]. However, it is unknown if they are associated
 61 with other arousal-related variables that can be measured in a rapid, accurate and non-invasive
 62 manner.

63 Spontaneous (i.e., pre-stimulus) oscillations in the α frequency band ($\sim 8 - 12\text{Hz}$) are
 64 associated with global changes in brain state. For example, research in humans using EEG and
 65 magnetoencephalography (MEG) has shown that the likelihood of detecting a near-threshold visual
 66 stimulus increases when pre-stimulus oscillations in the α band decrease [19–26]. The principles of
 67 signal detection theory [27] dictate that changes in behavior on detection tasks can arise due to
 68 changes in sensitivity or response criterion [28–30]. To that end, recent work has shown that a
 69 decrease in pre-stimulus α power is associated with increased hit rate and false alarm rate [31,32].
 70 These results point to link between pre-stimulus α power and response criterion, a key component of
 71 signal detection theory, which is modulated, at least in part, by subcortical structures that control
 72 arousal levels [31,33]. In addition, pre-stimulus α power is known to be correlated with eye metrics
 73 including pupil size [34,35] and reaction time [35–38] such that decreased pre-stimulus α power is
 74 accompanied by increased pupil size and decreased reaction time. These findings raise the possibility
 75 that other EEG signals such as the P1 component of the visually evoked potential (VEP) might also
 76 be associated with arousal.

77 Several studies have established a link between early components of the VEP and changes in
 78 global brain state. In contrast to pre-stimulus α oscillations, P1 amplitude is significantly larger on
 79 trials in which a weak visual stimulus is detected in comparison to when it is not detected
 80 [19,25,39,40]. Furthermore, studies that have explored the effects of spatial attention on early
 81 components of the VEP have shown that P1 amplitude is negatively correlated with reaction time
 82 [41]. In light of these results, and the well-known involvement of oscillatory brain activity in

stimulus-evoked responses [42], one might expect a negative relationship between pre-stimulus α oscillations and early VEP components. There is some evidence to suggest that this is the case. For example, Iemi et al. [43] found that the amplitude of the C1 and N150 increased when pre-stimulus power in the α frequency band decreased. These results suggest that pre-stimulus α power and early components of the VEP (e.g. P1 amplitude) can be used to index global changes in arousal. However, it is currently unclear how to link these EEG signals to underlying patterns of neural activity in the cortex.

In this study, we explored if pre-stimulus oscillations in the α frequency band and P1 amplitude are associated with slow drift in visual cortex. We simultaneously recorded EEG from the scalp and spiking activity from populations of neurons in V4 of two monkeys while they performed an orientation-change detection task (Figure 1A). Results showed that neural slow drift was associated with a pattern that is indicative of changes in the subjects' arousal levels over time. For example, decreases in pre-stimulus power specifically in the α band (i.e., not other frequencies) and increases in P1 amplitude were accompanied by 1) increases in pupil size, false alarm rate and saccade velocity; and 2) decreases in microsaccade rate and reaction time. These findings are important because they reveal that spontaneous/evoked components of the EEG signal recorded non-invasively on the scalp index low-dimensional patterns of neural activity acquired from microelectrode array recordings in the brain. They support previous research [17,18] showing that slow drift is associated with gradual changes in arousal over time, and provide a strong link between global measurements made across recording modalities and species.

Results

To determine if spontaneous and evoked components of the EEG signal can provide insight into the internal brain state associated with slow drift, we trained two macaque monkeys to perform an orientation change-detection task in which pairs of stimuli were repeatedly presented (Figure 1A). Spiking responses of populations of neurons in visual cortex (V4) were recorded using 100-channel

“Utah” arrays as well as EEG on the scalp (Figure 1B). On each trial, we calculated: 1) the mean amplitude of oscillations in different frequency bands during each pre-stimulus period (300-500ms) using a fast Fourier transform (FFT); and 2) the VEP during each 400ms stimulus period (see Methods). Previous research has shown that pre-stimulus α power and stimulus-evoked P1 amplitude across a wide range of frontal, midline and occipital electrode sites are associated with improved performance on detection tasks [19,22,31]. Hence, the signals from all 8 EEG electrodes (referenced online to the head post, see Methods) were averaged prior to computing the FFTs and VEPs. We focused primarily on pre-stimulus power in the α frequency band and P1 amplitude as they capture spontaneous (i.e., pre-stimulus) and evoked aspects of the EEG signal and have been linked to global changes in brain state. However, we also measured pre-stimulus beta (β) and gamma (γ) oscillations to exclude the possibility that slow drift was associated with aperiodic (i.e. broadband) changes in spectral power over time (1/f noise) [43–46].

Figure 1. Experimental methods. (A) Orientation-change detection task. After an initial fixation period, a sequence of stimuli (orientated Gabor pairs separated by fixation periods) was presented. The subjects' task was to detect an orientation change in one of the stimuli and make a saccade to the changed stimulus. (B) Electrophysiological recordings. We simultaneously recorded: 1) spiking responses of populations of neurons in V4 using 100-channel microelectrode ('Utah') arrays; and 2) EEG from 8 electrodes positioned on the scalp.

Correlation between pre-stimulus α power and P1 amplitude

First, we explored the relationship between pre-stimulus α power and the amplitude of the P1 component of the VEP. As described above, these two signals appear to operate in an antagonistic

manner on detection tasks with decreases in pre-stimulus α power/increases in P1 amplitude being associated with improved performance [19–26,39,40]. Hence, we hypothesized that these two large-scale EEG signals would be negatively correlated over time. To investigate the relationship between pre-stimulus α power and P1 amplitude, EEG data for each session was binned using a 30-minute sliding window stepped every 6 minutes (Figure 2A and Figure 2B). The width of the window, and the step size, were chosen to isolate slow changes over time based on previous studies we performed [17,18]. An example session is shown in Figure 2C. In support of our hypothesis, pre-stimulus power in the α frequency band was negatively associated with P1 amplitude.

Figure 2. Isolating slow fluctuations in spontaneous and evoked EEG signals. (A) Spontaneous EEG signals. On each trial, we calculated the mean amplitude of oscillations in distinct frequency bands for each pre-stimulus period using a FFT. The data was then binned using a 30-minute sliding window stepped every 6 minutes. Each bin is represented by a shaded grey line. Note that the duration of the fixation period was held constant at 300ms in this plot for illustration purposes only. In our analysis, we used the full duration of the fixation period, which was randomized between 300 to 500ms. (B) Evoked EEG signals. On each trial, we calculated a VEP for each stimulus presentation. The data was then binned using the same 30-minute sliding window so that direct comparisons could be made between spontaneous and evoked EEG signals. Each bin is represented by a shaded grey line. (C) Example session from Monkey 1 showing that pre-stimulus α power was negatively correlated with P1 amplitude over time. Each metric has been z-scored for illustration purposes. (D) Histogram showing distributions of Pearson's r values (black) across sessions between pre-stimulus α power and P1 amplitude. Actual distributions were compared to shuffled distributions (grey) using two-sided permutation tests (difference of medians). Median r values are indicated by dashed lines. $p < 0.05^*$, $p < 0.01^{**}$, $p < 0.001^{***}$. See Figure S1 for additional example sessions from both monkeys.

229 Next, we explored if a similar pattern was present across sessions. We computed correlations
 230 (Pearson product-moment correlation coefficient) between pre-stimulus power in the α band and the
 231 amplitude of the P1 component for each session and compared the distribution of Pearson's r values
 232 obtained to shuffled distributions using permutation tests (two-sided, difference of medians).
 233 Consistent with the pattern of results observed in several individual sessions for Monkey 1 and
 234 Monkey 2 (Figure S1), we found that pre-stimulus α power was significantly and negatively
 235 correlated with P1 amplitude (Figure 2D, median $r = -0.36$, $p = 0.024$). These results motivated us to
 236 ask if pre-stimulus α power and P1 amplitude are correlated with other task performance and eye
 237 metrics that are often taken to be hallmarks of arousal e.g. hit rate, false alarm rate, pupil size,
 238 microsaccade rate, reaction time and saccade velocity. A direct prediction of our results is that an
 239 opposite pattern should emerge when these measures are correlated with pre-stimulus α power and
 240 P1 amplitude.

241 Correlating pre-stimulus α power/P1 amplitude with other arousal-related variables

242 In order to directly compare pre-stimulus α power and P1 amplitude with a host of arousal-
 243 related variables we binned the task performance (hit rate and false rate) and eye metrics (pupil size,
 244 microsaccade rate, reaction time, saccade velocity) in the manner described above using a 30-minute
 245 sliding window stepped every six minutes. We computed Pearson product-moment correlations
 246 between these metrics for each session and we compared the subsequent distributions of Pearson's r
 247 values to shuffled distributions using permutation tests (two-sided, difference of medians). First, we
 248 found that the amplitude of pre-stimulus oscillations in the α band was negatively correlated with
 249 false alarm rate (Figure 3A, median $r = -0.39$, $p < 0.001$). No significant correlation was found
 250 between pre-stimulus α power and hit rate (median $r = 0.04$; $p = 0.783$) but false alarm rate was itself
 251 positively correlated with hit rate across sessions (Figure S2, median $r = 0.49$, $p < 0.001$). Hence, our
 252 results support an emerging body of work showing that the amplitude of pre-stimulus α oscillations
 253 is associated with changes in response criterion as opposed to sensitivity [32,43]. In addition, a
 254 negative correlation was found across sessions between pre-stimulus α power and pupil size in

255 Monkey 1 (median $r = -0.32$, $p = 0.037$), but no such correlation was found in Monkey 2 (median $r =$
256 0.07 , $p = 0.623$). We found a significant ($p < 0.05$) correlation between pre-stimulus α power and pupil
257 size in 7/20 sessions for Monkey 1 and 2/16 sessions for Monkey 2. Across sessions and monkeys,
258 we found positive correlations of pre-stimulus α power with reaction time (Figure 3A, median $r =$
259 0.26 , $p = 0.012$), and P1 amplitude with saccade velocity (Figure 3B, median $r = 0.21$, $p = 0.026$).
260 Negative correlations of pre-stimulus α power with saccade velocity (median $r = -0.27$, $p = 0.002$),
261 and P1 amplitude with microsaccade rate (median $r = -0.38$, $p = 0.008$) were also observed. No
262 significant correlation was found between P1 amplitude and reaction time (median $r = 0.08$, $p =$
263 0.728), but reaction time was itself negatively correlated with saccade velocity across sessions (Figure
264 S2, median $r = -0.52$, $p < 0.001$). Taken together, these results demonstrate that slow time scale
265 changes in spontaneous and evoked EEG signals were accompanied by changes in the subjects'
266 arousal levels over time. In particular, decreases in pre-stimulus α power and increases in P1
267 amplitude were accompanied by increases in false alarm rate and saccade velocity as well as decreases
268 in microsaccade rate and reaction time.

269

270

271

272

273

274

275

276

277

278

279

Figure 3. Correlations between EEG signals and other arousal-related variables. (A) Pre-stimulus α power. Each blue point corresponds to the median r value across sessions between pre-stimulus α power and a given metric. In contrast, grey points represent median r values computed using shuffled data. Actual distributions of r values were compared to shuffled distributions using two-sided permutation tests (difference of medians). Asterisks indicate a significant effect with a p value at least < 0.05 . See Figure S2 for histograms showing distributions of r values (black) across sessions between: A) hit rate and false alarm rate; and B) reaction time and saccade velocity.

Correlations between pre-stimulus α power, P1 amplitude and slow drift

As described above, we and others have shown that pre-stimulus α power and P1 amplitude are related to global changes in arousal. Therefore, we were keen to explore if these indirect and non-invasive measures of neural activity are associated with low-dimensional modes of neural

activity acquired directly from the spiking activity of a population of neurons. Previously, we reported a co-fluctuation of neural activity in macaque visual and prefrontal cortex [17]. This ‘slow drift’ was related to the subject’s tendency to make impulsive decisions in a change detection task, ignoring sensory evidence (false alarms), and multiple eye metrics that together are indicative of global changes in brain state [18]. Here we sought to determine if pre-stimulus α power and P1 amplitude are associated with slow drift.

To calculate slow drift, we binned spike counts in V4 using the same 30-minute sliding window described above and used in previous research (Figure 4A, see Methods). We then applied principal component analysis (PCA) to the data and estimated slow drift by projecting the binned residual spike counts along the first principal component (i.e. the loading vector that explained the most variance in the data). Because the sign of the loadings in PCA is arbitrary [47], the correlation between slow drift and a given variable can be positive or negative. This would not affect the overall pattern of results if one were solely interested in total variance explained. However, we were interested in whether slow drift was associated with a characteristic pattern of EEG activity that we and others have shown to be indicative of changes in the subjects’ arousal levels over time i.e. decreased pre-stimulus α power and increased P1 amplitude (Figure 2 and Figure 3). Hence, the sign of the correlation between slow drift and the spontaneous/evoked EEG signals was critical. In order to establish a common frame of reference across sessions, and preserve the sign of the correlations, we constrained the slow drift for each session to have the same relationship to the spontaneous and evoked activity of the neurons. That is, the slow drift was flipped if the mean projection value for evoked responses recorded during stimulus periods was less than that for spontaneous responses recorded during pre-stimulus periods i.e., if the relationship that was naturally observed for unprojected data did not hold true [18]. This served to align the data across sessions such that relatively large slow drift values were associated with higher firing rates. To put it another way, a negative correlation between pre-stimulus α power and slow drift would be indicative of a pattern in

which decreased pre-stimulus α power would lead to higher firing rates. Our hypothesis predicts that the opposite should be true for P1 amplitude.

We computed the slow drift of the neuronal population in each session using the above-mentioned method, and then compared it to our two spontaneous and evoked EEG measures: pre-stimulus α power and P1 amplitude. An example session is shown in Figure 4B (same sessions as in Figure 2C). In support of our hypothesis, a characteristic pattern was found in which slow drift was negatively associated with pre-stimulus α power and positively associated with P1 amplitude. Next, we investigated if a similar pattern was found across sessions.

Figure 4. Calculating slow drift. (A) Three example neurons from a single session (Monkey 1). Each point represents the mean residual spike count during a 400ms stimulus period. The data was then binned using a 30-minute sliding window stepped every six minutes (solid line) so that direct comparisons could be made with the EEG signals. PCA was used to reduce the dimensionality of the data and slow drift was calculated by projecting binned residual spike counts along the first principal component. (B) Example session from Monkey 1 showing that slow drift was negatively correlated with pre-stimulus α power and positively correlated with P1 amplitude. Each metric has been z-scored for illustration purposes.

We computed the Pearson product-moment correlation coefficient between pre-stimulus α power/P1 amplitude and slow drift for each session. We then compared the actual distribution of Pearson's r values to shuffled distributions using permutation tests (two-sided, difference of medians). Because the slow drift was aligned across sessions based on the neural activity alone and not the EEG metrics, the shuffled distributions were centered on a correlation value of zero. We also computed

363 correlations between slow drift and pre-stimulus oscillations in the beta (β) and gamma (γ) frequency
 364 bands to rule out the possibility that slow drift was associated with aperiodic (i.e. broadband) changes
 365 in spectral power over time (1/f noise) [43–46]. If this was the case, one might expect a significant
 366 correlation between slow drift and the amplitude of pre-stimulus oscillations in the α , β and γ
 367 frequency bands.

368 Consistent with the pattern of results observed in several individual sessions for Monkey 1
 369 and Monkey 2 (Figure S1), we found that slow drift was negatively correlated with pre-stimulus α
 370 power (median $r = -0.38$, $p = 0.004$), whereas it was positively correlated with P1 amplitude (median
 371 $r = 0.31$, $p = 0.027$). No significant correlation was found between slow drift and the amplitude of
 372 pre-stimulus power in the β (median $r = 0.04$, $p = 0.078$) or γ (median $r = 0.09$, $p = 0.621$) bands.
 373 Although these findings suggest that slow drift was not associated with changes in 1/f noise over time
 374 [43–46] we carried out an additional analysis in which aperiodic signals were removed prior to
 375 computing mean oscillatory power in different frequency bands. Importantly, the overall pattern of
 376 results remained the same when slow drift was correlated with aperiodic-adjusted power in the α
 377 (median $r = -0.18$, $p = 0.016$), β (median $r = -0.04$, $p = 0.145$) and γ (median $r = 0.07$, $p = 0.773$)
 378 bands (Figure S3). Therefore, we found that spontaneous and evoked EEG signals are associated with
 379 slow drift even after controlling for changes in 1/f noise [43–46].

380

381

382

383

384

385

386

387

388

Figure 5. Correlations between EEG signals and slow drift in visual cortex. Histogram showing distributions of r values (colors) across sessions between slow drift and spontaneous/evoked EEG signals. Actual distributions were compared to shuffled distributions (grey) using two-sided permutation tests (difference of medians). Median r values are indicated by dashed lines. $p < 0.05^*$, $p < 0.01^{**}$, $p < 0.001^{***}$. See Figure S3 for histograms showing distributions of r values across sessions between slow drift and aperiodic-adjusted power in different frequency bands.

Discussion

In this study, we investigated if pre-stimulus α oscillations and the amplitude of the P1 component of the VEP could be utilized as an external signature of an internal brain state, a recently discovered neural activity pattern called slow drift [17]. We know from previous work that slow drift in macaque visual and prefrontal cortex is significantly associated with a host of eye metrics that are strongly indicative of changes in arousal [18]. Since pre-stimulus α power and P1 amplitude are also related to arousal, we wondered if a link could be established between a neural measure of an internal

420 brain state acquired directly from the spiking activity of populations of neurons (i.e., slow drift) and
 421 indirect signals recorded non-invasively using EEG. Results showed that slow drift was significantly
 422 associated with a pattern that is indicative of changes in the subjects' arousal levels over time. When
 423 pre-stimulus α power was low and P1 amplitude was high, pupil size, false alarm rate and saccade
 424 velocity increased, whereas microsaccade rate and reaction time decreased.

425 A variety of studies have shown that pre-stimulus α power and P1 amplitude are related to a
 426 behavioral pattern that is associated with arousal level. For example, decreases in the amplitude of
 427 pre-stimulus α oscillations are typically accompanied by improved performance on detection tasks
 428 [19–26] and decreased reaction time [35–38], whereas the opposite is true of P1 amplitude [19,25,39–
 429 41]. This motivated us to ask if pre-stimulus α power was negatively associated with P1 amplitude.
 430 In support of previous research [43], we found that decreases in pre-stimulus α power were
 431 accompanied by increases in P1 amplitude. This antagonistic effect might be related to functional
 432 inhibition. Evidence suggests that neuronal excitability is reduced when pre-stimulus α oscillations
 433 are relatively strong [48–53]. Since early VEP components are thought to be generated in an additive
 434 manner on top of spontaneous activity [54–57] inhibition of low-level sensory areas might lead to a
 435 reduction in P1 amplitude. Regardless of the underlying mechanism, a direct prediction of our results
 436 is that pre-stimulus α oscillations and P1 amplitude should be correlated in an opposite manner with
 437 a constellation of arousal-related variables.

438 In this study, we explored if pre-stimulus α oscillations and P1 amplitude were associated
 439 with various task performance and eye metrics that have been strongly linked to global changes in
 440 brain state. Results showed that pre-stimulus α power was negatively correlated with false alarm rate
 441 and saccade velocity, whereas it was positively correlated with reaction time. In contrast, P1
 442 amplitude was negatively correlated with saccade velocity and microsaccade rate. Taken together,
 443 these findings suggest that the subjects' arousal levels were changing slowly over time. As described
 444 above, decreases in pre-stimulus α power and increases in P1 amplitude are known to be associated
 445 with improved performance on detection tasks [19–26,39,40]. That these metrics were also coupled

446 with increases in false alarm rate and saccade velocity as well as decreases in microsaccade rate and
447 reaction time suggests that they can be taken as external measures of heightened arousal levels. In
448 addition, our findings extend recent work in humans [31,32] showing that pre-stimulus α power is
449 associated with changes in response criterion as opposed to sensitivity.

450 The principles of signal detection theory [27] dictate that improved performance on detection
451 tasks can arise due to changes in sensitivity or response criterion [28–30]. Recent work has shown
452 that decreased pre-stimulus α power is associated with increased hit rate and false alarm rate, which
453 suggests that performance improvements linked to decreased pre-stimulus α power might occur due
454 to changes in response criterion [31,32]. Our results support this hypothesis, as pre-stimulus α power
455 was negatively associated with false alarm rate. Furthermore, we found that slow drift was positively
456 correlated with hit rate (median $r = 0.41$, $p < 0.001$) and false alarm rate (median $r = 0.53$, $p < 0.001$)
457 across sessions, consistent with previous research [17]. Luo and Maunsell (2015) found that changes
458 in sensitivity, not response criterion were related to single-neuron and pairwise statistics in V4. In
459 contrast, our results suggest that V4 activity is related to a shift in response criterion that was indexed
460 by decreased pre-stimulus α power. One possible explanation for this discrepancy is that Luo and
461 Maunsell (2015) manipulated response criterion in a spatially selective manner to avoid shifts arising
462 due to global changes in arousal. In our study, response criterion was not manipulated directly, so
463 shifts are likely to have occurred spontaneously due to global changes in the subjects' arousal levels.
464 In addition, we averaged across electrode sites because decreases in pre-stimulus α power/increases
465 in P1 amplitude are associated with improved performance on detection tasks across large swathes of
466 cortex [19,22,31]. These findings suggest that spatially selective and global shifts in response
467 criterion might be governed by distinct neural mechanisms. This is an important issue to address, not
468 just because it has the potential to resolve a discrepancy in the literature, but it also might explain
469 why pre-stimulus α power on spatial attention tasks is associated with a pattern that is more akin to
470 changes in sensitivity i.e. decreased/increased pre-stimulus α power over electrodes
471 contralateral/ipsilateral to the attended hemifield [58].

In summary, we found that two commonly used metrics of cognitive state in human EEG studies, pre-stimulus α power and P1 amplitude, are associated with gradual shifts in the underlying population structure of neural activity throughout the brain. Together, these measures at the scalp, and in the cortex, were predictive of changes in the monkeys' arousal levels over time. These findings show that indirect measures of neural activity can be used to index a global signature of arousal. They support recent work showing that pre-stimulus α power is associated with response criterion [59]. Finally, by linking a vast EEG literature in humans with simultaneous scalp/microelectrode array recordings in macaques our results bridge the gap between large-scale signals and the responses of single cortical neurons.

Methods

Subjects

Two adult rhesus macaque monkeys (*Macaca mulatta*) were used in this study. A previous report [60] presented analysis of different aspects of the same experiments described here. Surgical procedures to chronically implant a titanium head post (to immobilize the subjects' heads during experiments) and microelectrode arrays were conducted in aseptic conditions under isoflurane anesthesia, as described in detail by Smith and Sommer (2013). Opiate analgesics were used to minimize pain and discomfort during the perioperative period. Neural activity was recorded using 100-channel "Utah" arrays (Blackrock Microsystems) in V4 (Monkey 1 = right hemisphere; Monkey 2 = left hemisphere). The arrays comprised a 10x10 grid of silicon microelectrodes (1 mm in length) spaced 400 μ m apart. Experimental procedures were approved by the Institutional Animal Care and Use Committee of the University of Pittsburgh and were performed in accordance with the United States National Research Council's Guide for the Care and Use of Laboratory Animals.

Microelectrode array recordings

Signals from each microelectrode in the array were amplified and band-pass filtered (0.3–7500 Hz) by a Grapevine system (Ripple). Waveform segments crossing a threshold (set as a multiple

of the root mean square noise on each channel) were digitized (30KHz) and stored for offline analysis and sorting. First, waveforms were automatically sorted using a competitive mixture decomposition method [62]. They were then manually refined using custom time amplitude window discrimination software (code available at <https://github.com/smithlabvision/spikesort>), which takes into account metrics including (but not limited to) waveform shape and the distribution of interspike intervals [63]. A mixture of single and multiunit activity was recorded, but we refer here to all units as “neurons”. The mean number of V4 neurons across sessions was 70 (SD = 11) for Monkey 1 and 31 (SD = 16) for Monkey 2.

506 EEG recordings

We recorded EEG from 8 Ag/AgCl electrodes (Grass Technologies) adhered to the scalp with electrically conductive paste. The electrodes were positioned roughly at the following locations: Fz, Iz, CP3, CP4, F5, F6, PO7, and PO8 (see Fig. 1B). Signals were referenced online to a steel screw on the titanium head post, digitized at 1 kHz and amplified by a Grapevine system (Ripple) and low-pass filtered online at 250 Hz. Segments of EEG data (a segment is defined as a 400ms stimulus period or a 300-500ms pre-stimulus period) were considered excessively noisy and removed if any of the channels had a standard deviation 10 times greater than the mean of the entire session. We also removed segments of data if any of the channels exhibited a flat signal defined as a standard deviation less than 300 nanovolts.

516 Visual stimuli

Visual stimuli were generated using a combination of custom software written in MATLAB (The MathWorks) and Psychophysics Toolbox extensions [64,65]. They were displayed on a CRT monitor (resolution = 1024 X 768 pixels; refresh rate = 100Hz), which was viewed at a distance of 36 cm and gamma-corrected to linearize the relationship between input voltage and output luminance using a photometer and look-up-tables.

522 Behavioral task

Subjects fixated a central point (diameter = 0.6°) on the monitor to initiate a trial (Figure 1A).

Each trial comprised a sequence of stimulus periods (400ms) separated by fixation periods (duration drawn at random from a uniform distribution spanning 300-500ms). The 400ms stimulus periods comprised pairs of drifting full-contrast Gabor stimuli. One stimulus was presented in the aggregate receptive field (RF) of the recorded V4 neurons, whereas the other stimulus was presented in the mirror-symmetric location in the opposite hemifield. Although the spatial (Monkey 1 = $0.85\text{cycles}/^\circ$; Monkey 2 = $0.85\text{cycles}/^\circ$) and temporal frequencies (Monkey 1 = 8cycles/s ; Monkey 2 = 7cycles/s) of the stimuli were not optimized for each individual V4 neuron they did evoke a strong response from the population. The orientation of the stimulus in the aggregate RF was chosen at random to be 45 or 135° , and the stimulus in the opposite hemifield was assigned the other orientation. There was a fixed probability (Monkey 1 = 30%; Monkey 2 = 40%) that one of the Gabors would change orientation by ± 1 , ± 3 , ± 6 , or $\pm 15^\circ$ on each stimulus presentation. The sequence continued until the subject) made a saccade to the changed stimulus within 400ms (“hit”); 2) made a saccade to an unchanged stimulus (“false alarm”); or 3) remained fixating for more than 400ms after a change occurred (“miss”). If the subject correctly detected an orientation change, they received a liquid reward. In contrast, a time-out occurred if the subject made a saccade to an unchanged stimulus delaying the beginning of the next trial by 1s. It is important to note that the effects of spatial attention were also investigated (although not analyzed in this study) by cueing blocks of trials such that the orientation change was 90% more likely to occur in one hemifield relative to the other hemifield.

Eye tracking

Eye position and pupil diameter were recorded monocularly at a rate of 1000Hz using an infrared eye tracker (EyeLink 1000, SR Research).

Microsaccade detection

Microsaccades were defined as eye movements that exceeded a velocity threshold of 6 times the standard deviation of the median velocity for at least 6ms [66–68]. They were required to be separated in time by at least 100ms. In addition, we removed microsaccades with an amplitude greater

549 than 1° and a velocity greater than $100^\circ/\text{s}$. To assess the validity of our microsaccade detection
550 method, the correlation (Pearson product-moment correlation coefficient) between the amplitude and
551 peak velocity of detected microsaccades (i.e., the main sequence) was computed for each session.
552 The mean correlation between these two metrics across sessions was 0.86 (SD = 0.07) indicating that
553 our detection algorithm was robust [69].

554 Pre-stimulus power

555 To calculate the mean amplitude of oscillations in different frequency bands during pre-
556 stimulus periods the signals from all 8 EEG electrodes were averaged together. We adopted this
557 approach because previous research has shown that pre-stimulus α power across a wide range of
558 frontal, midline and occipital locations is associated with performance on detection tasks [19,22,31].
559 Furthermore, the overall pattern of results did not change when signals were averaged across frontal,
560 midline and posterior electrodes. We then computed a FFT of the Hanning-windowed segment of data
561 spanning the entire duration of the pre-stimulus period (300 - 500ms) and computed mean power in
562 the α , β and γ bands. To isolate slow changes, the data for each trial and power band was binned using
563 a 30-minute sliding window stepped every 6 minutes (Figure 2A).

564 P1 amplitude

565 To calculate the VEP during stimulus periods the signals from all 8 EEG electrodes were
566 averaged together. We adopted this approach for similar reasons to those described above i.e., because
567 P1 amplitude across a wide range of frontal, midline and occipital locations is associated with
568 performance on detection tasks [19]. In addition, the overall pattern of results remained the same
569 when signals were averaged across frontal, midline and posterior electrodes. We then computed the
570 VEP for the period spanning 200ms before to 200ms after stimulus onset, filtered the segment
571 between 1 and 25Hz, and baseline-corrected by subtracting the average voltage for the period
572 spanning 200ms before stimulus onset. To isolate slow changes over time, we divided the session into
573 30-minute bins stepped every 6 minutes and computed the mean VEP for each time bin. We were
574 interested in whether slow drift was associated with the amplitude of the P1 component. Hence, we

575 calculated the maximum amplitude of the peak occurring ~100ms after stimulus onset from the VEP
576 for each time bin (Figure 2B). Note that we were only able to reliably compute VEPs in 3 sessions
577 for Monkey 2, which means the majority of evoked EEG data is from Monkey 1 (17 sessions).

578 Aperiodic-adjusted pre-stimulus power

579 We wanted to rule out the possibility that slow drift was associated with gradual changes in
580 1/f noise [43–46]. Therefore, we carried out a supplementary analysis in which slow drift was
581 correlated with aperiodic-adjusted power in different frequency bands. As before, signals from all 8
582 EEG electrodes were averaged together. We then computed a FFT of the Hanning-windowed segment
583 of data spanning the first 300ms of the pre-stimulus period. To isolate slow changes over time, we
584 divided the session into 30-minute bins stepped every 6 minutes and computed the mean FFT for each
585 time bin. We then subtracted the aperiodic signal from the FFT for each time bin, which was computed
586 by fitting an exponential function [46]. Finally, mean pre-stimulus power in the α , β and γ bands was
587 calculated from the aperiodic-adjusted FFT for each time bin. Note that the frequency axis of the FFT
588 for each pre-stimulus interval had to be identical in this analysis so as to reliably estimate the aperiodic
589 signal. Since accounting for broadband changes in 1/f noise did not change the overall pattern of
590 results (Figure S3) we decided to correlate slow drift with pre-stimulus power in different frequency
591 bands using the full duration of the pre-stimulus interval (300-500ms) to enhance frequency
592 resolution.

593 Task performance metrics

594 Hit rate was defined as the number of saccades toward a stimulus that changed orientation
595 divided by the total number of stimuli presented that changed orientation. False alarm rate was the
596 number of saccades toward a stimulus that did not change orientation divided by the total number of
597 stimuli presented that did not change orientation. To isolate slow changes, hit rate and false alarm rate
598 were calculated in 30-minute bins stepped every 6 minutes. The width of the bins and step size were
599 chosen based on previous research to ensure reliable estimates over a relatively small number of trials
600 [17].

601 Eye metrics

602 Mean pupil diameter was measured during stimulus periods, whereas microsaccade rate was
603 measured during fixation periods between the visual stimulus presentations [18]. We did not include
604 the initial fixation period when measuring microsaccade rate as there was an increase in eye position
605 variability during this period resulting from fixation having been established a short time earlier (300-
606 500ms). Such variability was not present in subsequent fixation periods. Reaction time and saccade
607 velocity were measured on trials in which the subjects were rewarded for correctly detecting an
608 orientation change. Reaction time was defined as the time from when the change occurred to the time
609 at which the saccade exceeded a velocity threshold of 100°/s. Saccade velocity was the peak velocity
610 of the saccade to the changed stimulus. To isolate slow changes in the eye metrics over time the data
611 for each session was binned using a 30-minute sliding window stepped every 6 minutes.

612 Calculating slow drift

613 The spiking responses of populations of neurons in V4 were measured during a 400ms period
614 that began 50ms after stimulus presentation (Figure 4A). To control for the fact that some neurons
615 had a preference for one orientation (45 or 135°) over the other residual spike counts were calculated.
616 We subtracted the mean response for a given orientation across the entire session from individual
617 responses to that orientation. To isolate slow changes in neural activity over time, residual spike
618 counts for each V4 neuron were binned using a 30-minute sliding window stepped every 6 minutes
619 (Figure 4A). PCA was then performed to reduce the high-dimensional residual data to a smaller
620 number of latent variables [70]. Slow drift in V4 was estimated by projecting the binned residual
621 spike counts for each neuron along the first principal component.

622 Aligning slow drift across sessions

623 As described above, slow drift was calculated by projecting binned residual spike counts along
624 the first principal component. The weights in a PCA can be positive or negative [47], which meant
625 the sign of the correlation between slow drift and a given metric was arbitrary. Preserving the sign of
626 the correlations was particularly important in this study because we were interested in whether slow

627 drift was associated with a pattern that is indicative of changes in the subjects' arousal levels over
628 time i.e., decreased pre-stimulus α power and increased P1 amplitude. The method for aligning slow
629 drift across sessions is described in detail by Johnston et al. [18]. Briefly, we projected evoked
630 responses acquired during stimulus periods and spontaneous responses acquired during pre-stimulus
631 periods onto the first principal component. The sign of the slow drift was flipped if the mean
632 projection value for the evoked responses was less than that for the spontaneous responses i.e., if the
633 relationship that was naturally observed for unprojected data did not hold true.

634

635

636

637

638

639

640

641

642

643

644

645

646

647

648

649

650

651

652

653

654

655

656

657

Figure S1. Relationships between slow drift, pre-stimulus α power and P1 amplitude (A) Example sessions from Monkey 1. Each metric has been z-scored for illustration purposes. (B) Same as (A) but for Monkey 2.

Figure S2. (A) Histogram showing distributions of r values (black) across sessions between hit rate and false alarm rate. (B) Same as (A) but for r values computed between reaction time and saccade velocity. In (A) and (B) actual distributions were compared to shuffled distributions (grey) using two-sided permutation tests (difference of medians). Median r values are indicated by dashed lines. $p < 0.05^*$, $p < 0.01^{**}$, $p < 0.001^{***}$.

Figure S3. Correlations between aperiodic-adjusted power and slow drift. Histograms showing distributions of r values (colors) across sessions between slow drift and EEG power in different frequency bands. Actual distributions were compared to shuffled distributions (grey) using two-sided permutation tests (difference of medians). Median r values are indicated by dashed lines. $p < 0.05^*$, $p < 0.01^{**}$, $p < 0.001^{***}$.

765 References

- 766 1. Britten, K.H., Newsome, W.T., Shadlen, M.N., Celebrini, S., and Movshon, J.A. (1996). A
767 relationship between behavioral choice and the visual responses of neurons in macaque MT.
768 *Vis. Neurosci.* *13*, 87–100.
- 769 2. Moran, J., and Desimone, R. (1985). Selective attention gates visual information processing
770 in extrastriate cortex. *Science* (80-.). *229*, 782–784.
- 771 3. Fuster, J.M., and Alexander, G.E. (1971). Neuron activity related to short-term memory.
772 *Science* (80-.). *173*, 652–654.
- 773 4. Cohen, M.R., and Maunsell, J.H.R. (2009). Attention improves performance primarily by
774 reducing interneuronal correlations. *Nat. Neurosci.* *12*, 1594–1600.
- 775 5. Leavitt, M.L., Pieper, F., Sachs, A.J., and Martinez-Trujillo, J.C. (2017). Correlated
776 variability modifies working memory fidelity in primate prefrontal neuronal ensembles. *Proc.*
777 *Natl. Acad. Sci. U. S. A.* *114*, E2494–E2503.
- 778 6. Zohary, E., Shadlen, M.N., and Newsome, W.T. (1994). Correlated neuronal discharge rate
779 and its implications for psychophysical performance. *Nature* *370*, 140–143.
- 780 7. Murray, J.D., Bernacchia, A., Roy, N.A., Constantinidis, C., Romo, R., and Wang, X.J.
781 (2017). Stable population coding for working memory coexists with heterogeneous neural
782 dynamics in prefrontal cortex. *Proc. Natl. Acad. Sci. U. S. A.* *114*, 394–399.
- 783 8. Harvey, C.D., Coen, P., and Tank, D.W. (2012). Choice-specific sequences in parietal cortex
784 during a virtual-navigation decision task. *Nature* *484*, 62–68.
- 785 9. Khanna, S.B., Snyder, A.C., and Smith, M.A. (2019). Distinct sources of variability affect
786 eye movement preparation. *J. Neurosci.* *39*, 4511–4526.
- 787 10. Mante, V., Sussillo, D., Shenoy, K. V., and Newsome, W.T. (2013). Context-dependent
788 computation by recurrent dynamics in prefrontal cortex. *Nature* *503*, 78–84.
- 789 11. Sohn, H., Narain, D., Meirhaeghe, N., and Jazayeri, M. (2018). Bayesian computation
790 through cortical latent dynamics. *bioRxiv*, 465419. Available at:

- 791 <https://www.biorxiv.org/content/early/2018/11/08/465419>.
- 792 12. Remington, E.D., Narain, D., Hosseini, E.A., and Jazayeri, M. (2018). Flexible Sensorimotor
793 Computations through Rapid Reconfiguration of Cortical Dynamics. *Neuron* 98, 1005-
794 1019.e5. Available at: <https://doi.org/10.1016/j.neuron.2018.05.020>.
- 795 13. Wang, J., Narain, D., Hosseini, E.A., and Jazayeri, M. (2018). Flexible timing by temporal
796 scaling of cortical responses. *Nat. Neurosci.* 21, 102–112. Available at:
797 <http://dx.doi.org/10.1038/s41593-017-0028-6>.
- 798 14. Sadtler, P.T., Quick, K.M., Golub, M.D., Chase, S.M., Ryu, S.I., Tyler-Kabara, E.C., Yu,
799 B.M., and Batista, A.P. (2014). Neural constraints on learning. *Nature* 512, 423–426.
800 Available at: <http://dx.doi.org/10.1038/nature13665>.
- 801 15. Stringer, C., Pachitariu, M., Steinmetz, N., Reddy, C.B., Carandini, M., and Harris, K.D.
802 (2019). Spontaneous behaviors drive multidimensional, brainwide activity. *Science* (80-.).
803 364.
- 804 16. Musall, S., Kaufman, M.T., Juavinett, A.L., Gluf, S., and Churchland, A.K. (2019). richly
805 varied movements. *Nat. Neurosci.* 22. Available at: [http://dx.doi.org/10.1038/s41593-019-](http://dx.doi.org/10.1038/s41593-019-0502-4)
806 0502-4.
- 807 17. Cowley, B., Snyder, A., Acar, K., Williamson, R., Yu, B., and Smith, M. (2020). Slow drift of
808 neural activity as a signature of impulsivity in macaque visual and prefrontal cortex. *Neuron*,
809 1–17. Available at: <https://doi.org/10.1016/j.neuron.2020.07.021>.
- 810 18. Johnston, R., Snyder, A.C., Khanna, S., Issar, D., and Smith, M.A. (2020). The eyes reflect
811 an internal cognitive state embedded in the population activity of cortical neurons. *bioRxiv*,
812 2020.06.29.178251. Available at:
813 <http://biorxiv.org/content/early/2020/06/30/2020.06.29.178251.abstract>.
- 814 19. Ergenoglu, T., Demiralp, T., Bayraktaroglu, Z., Ergen, M., Beydagi, H., and Uresin, Y.
815 (2004). Alpha rhythm of the EEG modulates visual detection performance in humans. *Cogn.*
816 *Brain Res.* 20, 376–383.

- 817 20. Babiloni, C., Vecchio, F., Bultrini, A., Romani, G.L., and Rossini, P.M. (2006). Pre- and
818 poststimulus alpha rhythms are related to conscious visual perception: A high-resolution EEG
819 study. *Cereb. Cortex* 16, 1690–1700.
- 820 21. Van Dijk, H., Schoffelen, J.M., Oostenveld, R., and Jensen, O. (2008). Prestimulus
821 oscillatory activity in the alpha band predicts visual discrimination ability. *J. Neurosci.* 28,
822 1816–1823.
- 823 22. Busch, N.A., Dubois, J., and VanRullen, R. (2009). The phase of ongoing EEG oscillations
824 predicts visual perception. *J. Neurosci.* 29, 7869–7876.
- 825 23. Thut, G., Nietzel, A., Brandt, S.A., and Pascual-Leone, A. (2006). α -Band
826 electroencephalographic activity over occipital cortex indexes visuospatial attention bias and
827 predicts visual target detection. *J. Neurosci.* 26, 9494–9502.
- 828 24. Hanslmayr, S., Aslan, A., Staudigl, T., Klimesch, W., Herrmann, C.S., and Bäuml, K.H.
829 (2007). Prestimulus oscillations predict visual perception performance between and within
830 subjects. *Neuroimage* 37, 1465–1473.
- 831 25. Mathewson, K.E., Gratton, G., Fabiani, M., Beck, D.M., and Ro, T. (2009). To see or not to
832 see: Prestimulus α phase predicts visual awareness. *J. Neurosci.* 29, 2725–2732.
- 833 26. Romei, V., Gross, J., and Thut, G. (2010). On the role of prestimulus alpha rhythms over
834 occipito-parietal areas in visual input regulation: Correlation or causation? *J. Neurosci.* 30,
835 8692–8697.
- 836 27. Green, D.M., and Swets, J.A. (1966). Signal detection theory and psychophysics (Wiley).
- 837 28. Luo, T.Z., and Maunsell, J.H.R. (2015). Neuronal Modulations in Visual Cortex Are
838 Associated with Only One of Multiple Components of Attention. *Neuron* 86, 1182–1188.
839 Available at: <http://dx.doi.org/10.1016/j.neuron.2015.05.007>.
- 840 29. Luo, T.Z., and Maunsell, J.H.R. (2018). Attentional Changes in Either Criterion or Sensitivity
841 Are Associated with Robust Modulations in Lateral Prefrontal Cortex. *Neuron* 97, 1382-
842 1393.e7. Available at: <https://doi.org/10.1016/j.neuron.2018.02.007>.

- 843 30. Crapse, T.B., Lau, H., and Basso, M.A. (2018). A Role for the Superior Colliculus in
844 Decision Criteria. *Neuron* 97, 181-194.e6. Available at:
845 <https://doi.org/10.1016/j.neuron.2017.12.006>.
- 846 31. Iemi, L., Chaumon, M., Crouzet, S.M., and Busch, N.A. (2017). Spontaneous neural
847 oscillations bias perception by modulating baseline excitability. *J. Neurosci.* 37, 807–819.
- 848 32. Limbach, K., and Corballis, P.M. (2016). Prestimulus alpha power influences response
849 criterion in a detection task. *Psychophysiology* 53, 1154–1164.
- 850 33. de Gee, J.W., Colizoli, O., Kloosterman, N.A., Knapen, T., Nieuwenhuis, S., and Donner,
851 T.H. (2017). Dynamic modulation of decision biases by brainstem arousal systems. *Elife* 6,
852 1–36.
- 853 34. Hong, L., Walz, J.M., and Sajda, P. (2014). Your eyes give you away: Prestimulus changes in
854 pupil diameter correlate with poststimulus task-related EEG dynamics. *PLoS One* 9.
- 855 35. Van Kempen, J., Loughnane, G.M., Newman, D.P., Kelly, S.P., Thiele, A., O’Connell, R.G.,
856 and Bellgrove, M.A. (2019). Behavioural and neural signatures of perceptual decision-
857 making are modulated by pupil-linked arousal. *Elife* 8, 1–27.
- 858 36. Kelly, S.P., and O’Connell, R.G. (2013). Internal and external influences on the rate of
859 sensory evidence accumulation in the human brain. *J. Neurosci.* 33, 19434–19441.
- 860 37. Zhang, Y., Wang, X., Bressler, S.L., Chen, Y., and Ding, M. (2008). Prestimulus cortical
861 activity is correlated with speed of visuomotor processing. *J. Cogn. Neurosci.* 20, 1915–
862 1925.
- 863 38. Bompas, A., Sumner, P., Muthumaraswamy, S.D., Singh, K.D., and Gilchrist, I.D. (2015).
864 The contribution of pre-stimulus neural oscillatory activity to spontaneous response time
865 variability. *Neuroimage* 107, 34–45.
- 866 39. Del Cul, A., Baillet, S., and Dehaene, S. (2007). Brain dynamics underlying the nonlinear
867 threshold for access to consciousness. *PLoS Biol.* 5, 2408–2423.
- 868 40. Pourtois, G., De Pretto, M., and Hauert, C. (2006). Time course of brain activity during

- 869 change blindness and change awareness: performance J. Cogn. Neurosci., 2108–2129.
- 870 Available at:
- 871 [http://www.mitpressjournals.org/doi/abs/10.1162/jocn.2006.18.12.2108%5Cnfile:///Users/Megan/Documents/Papers2/Articles/2006/Pourtois/Time course of brain activity during change](http://www.mitpressjournals.org/doi/abs/10.1162/jocn.2006.18.12.2108%5Cnfile:///Users/Megan/Documents/Papers2/Articles/2006/Pourtois/Time%20course%20of%20brain%20activity%20during%20change%20blindness%20and%20change%20awareness%20performance%20...%202006%20Pourtois.pdf%5Cnpapers2://pu)
- 872 [blindness and change awareness performance ... 2006 Pourtois.pdf%5Cnpapers2://pu](http://www.mitpressjournals.org/doi/abs/10.1162/jocn.2006.18.12.2108%5Cnfile:///Users/Megan/Documents/Papers2/Articles/2006/Pourtois/Time course of brain activity during change blindness and change awareness performance ... 2006 Pourtois.pdf%5Cnpapers2://pu).
- 873
- 874 41. Mangun, G.R., and Hillyard, S.A. (1991). Modulations of Sensory-Evoked Brain Potentials
- 875 Indicate Changes in Perceptual Processing During Visual-Spatial Priming. J. Exp. Psychol.
- 876 Hum. Percept. Perform. 17, 1057–1074.
- 877 42. Zoefel, B., ten Oever, S., and Sack, A.T. (2018). The involvement of endogenous neural
- 878 oscillations in the processing of rhythmic input: More than a regular repetition of evoked
- 879 neural responses. Front. Neurosci. 12, 1–13.
- 880 43. Iemi, L., Busch, N.A., Laudini, A., Haegens, S., Samaha, J., Villringer, A., and Nikulin, V. V.
- 881 (2019). Multiple mechanisms link prestimulus neural oscillations to sensory responses. Elife
- 882 8, 1–34.
- 883 44. Voytek, B., Kramer, M.A., Case, J., Lepage, K.Q., Tempesta, Z.R., Knight, R.T., and
- 884 Gazzaley, A. (2015). Age-related changes in 1/f neural electrophysiological noise. J.
- 885 Neurosci. 35, 13257–13265.
- 886 45. Peterson, E.J., Rosen, B.Q., Campbell, A.M., Belger, A., and Voytek, B. (2017). 1/F Neural
- 887 Noise Is a Better Predictor of Schizophrenia Than Neural Oscillations. Doi.Org, 113449.
- 888 Available at: <https://www.biorxiv.org/content/early/2017/03/03/113449>.
- 889 46. Haller, M., Donoghue, T., Peterson, E., Varma, P., Sebastian, P., Gao, R., Noto, T., Knight,
- 890 R.T., Shestyuk, A., and Voytek, B. (2018). Parameterizing neural power spectra. bioRxiv,
- 891 299859. Available at:
- 892 <https://www.biorxiv.org/content/biorxiv/early/2018/04/11/299859.full.pdf>.
- 893 47. Jolliffe, I.T., and Cadima, J. (2016). Principal component analysis: A review and recent
- 894 developments. Philos. Trans. R. Soc. A Math. Phys. Eng. Sci. 374.

- 895 48. Haegens, S., Nácher, V., Luna, R., Romo, R., and Jensen, O. (2011). α -Oscillations in the
896 monkey sensorimotor network influence discrimination performance by rhythmical inhibition
897 of neuronal spiking. *Proc. Natl. Acad. Sci. U. S. A.* *108*, 19377–19382.
- 898 49. Jensen, O. (2002). Oscillations in the Alpha Band (9-12 Hz) Increase with Memory Load
899 during Retention in a Short-term Memory Task. *Cereb. Cortex* *12*, 877–882.
- 900 50. Klimesch, W., Doppelmayr, M., Schwaiger, J., Auinger, P., and Winkler, T. (1999).
901 “Paradoxical” alpha synchronization in a memory task. *Cogn. Brain Res.* *7*, 493–501.
- 902 51. Mayhew, S.D., Ostwald, D., Porcaro, C., and Bagshaw, A.P. (2013). Spontaneous EEG alpha
903 oscillation interacts with positive and negative BOLD responses in the visual-auditory
904 cortices and default-mode network. *Neuroimage* *76*, 362–372. Available at:
905 <http://dx.doi.org/10.1016/j.neuroimage.2013.02.070>.
- 906 52. Harvey, B.M., Vansteensel, M.J., Ferrier, C.H., Petridou, N., Zuiderbaan, W., Aarnoutse, E.J.,
907 Bleichner, M.G., Dijkerman, H.C., van Zandvoort, M.J.E., Leijten, F.S.S., *et al.* (2013).
908 Frequency specific spatial interactions in human electrocorticography: V1 alpha oscillations
909 reflect surround suppression. *Neuroimage* *65*, 424–432. Available at:
910 <http://dx.doi.org/10.1016/j.neuroimage.2012.10.020>.
- 911 53. Van Kerkoerle, T., Self, M.W., Dagnino, B., Gariel-Mathis, M.A., Poort, J., Van Der Togt, C.,
912 and Roelfsema, P.R. (2014). Alpha and gamma oscillations characterize feedback and
913 feedforward processing in monkey visual cortex. *Proc. Natl. Acad. Sci. U. S. A.* *111*, 14332–
914 14341.
- 915 54. Mazaheri, A., and Jensen, O. (2006). Posterior α activity is not phase-reset by visual stimuli.
916 *Proc. Natl. Acad. Sci. U. S. A.* *103*, 2948–2952.
- 917 55. Shah, A.S., Bressler, S.L., Knuth, K.H., Ding, M., Mehta, A.D., Ulbert, I., and Schroeder,
918 C.E. (2004). Neural Dynamics and the Fundamental Mechanisms of Event-related Brain
919 Potentials. *Cereb. Cortex* *14*, 476–483.
- 920 56. Mäkinen, V., Tiitinen, H., and May, P. (2005). Auditory event-related responses are generated

- 921 independently of ongoing brain activity. *Neuroimage* 24, 961–968.
- 922 57. Bijma, F., De Munck, J.C., Huizenga, H.M., and Heethaar, R.M. (2003). A mathematical
923 approach to the temporal stationarity of background noise in MEG/EEG measurements.
924 *Neuroimage* 20, 233–243.
- 925 58. Foxe, J.J., and Snyder, A.C. (2011). The role of alpha-band brain oscillations as a sensory
926 suppression mechanism during selective attention. *Front. Psychol.* 2, 1–13.
- 927 59. Samaha, J., Iemi, L., Haegens, S., and Busch, N.A. (2020). Spontaneous Brain Oscillations
928 and Perceptual Decision-Making. *Trends Cogn. Sci.*, 1–15. Available at:
929 <https://doi.org/10.1016/j.tics.2020.05.004>.
- 930 60. Snyder, A.C., Yu, B.M., and Smith, M.A. (2018). Distinct population codes for attention in
931 the absence and presence of visual stimulation. *Nat. Commun.* 9, 4382. Available at:
932 <http://www.nature.com/articles/s41467-018-06754-5>.
- 933 61. Smith, M.A., and Sommer, M.A. (2013). Spatial and Temporal Scales of Neuronal
934 Correlation in Visual Area V4. *J. Neurosci.* 33, 5422–5432. Available at:
935 <http://www.jneurosci.org/cgi/doi/10.1523/JNEUROSCI.4782-12.2013>.
- 936 62. Shoham, S., Fellows, M.R., and Normann, R.A. (2003). Robust, automatic spike sorting
937 using mixtures of multivariate t-distributions. *J. Neurosci. Methods* 127, 111–122.
- 938 63. Kelly, R.C., Smith, M.A., Samonds, J.M., Kohn, A., Bonds, A.B., Movshon, J.A., and Sing
939 Lee, T. (2007). Comparison of Recordings from Microelectrode Arrays and Single Electrodes
940 in the Visual Cortex. *J. Neurosci.* 27, 261–264.
- 941 64. Brainard, D.H. (1997). The Psychophysics Toolbox. *Spat. Vis.* 10, 433–436.
- 942 65. Pelli, D.G. (1997). The VideoToolbox software for visual psychophysics: Transforming
943 numbers into movies. *Spat. Vis.* 10, 437–442.
- 944 66. Scholes, C., McGraw, P. V., Nyström, M., and Roach, N.W. (2015). Fixational eye
945 movements predict visual sensitivity. *Proc. R. Soc. B Biol. Sci.* 282.
- 946 67. Engbert, R., and Kliegl, R. (2003). Microsaccades uncover the orientation of covert attention.

947 Vision Res. *43*, 1035–1045.

948 68. Loughnane, G.M., Newman, D.P., Tamang, S., Kelly, S.P., and O’Connell, R.G. (2018).

949 Antagonistic interactions between microsaccades and evidence accumulation processes

950 during decision formation. *J. Neurosci.* *38*, 2163–2176.

951 69. Zuber, B.L., Stark, L., and Cook, G. (1965). Microsaccades and the velocity-amplitude

952 relationship for saccadic eye movements. *Science* (80-.). *150*, 1459–1460.

953 70. Cunningham, J.P., and Yu, B.M. (2014). Dimensionality reduction for large-scale neural

954 recordings. *Nat. Neurosci.* *17*, 1500–1509.

955