Prestimulus Neural Oscillations Inhibit Visual Perception via Modulation of Response Gain

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Abstract

■ The ongoing state of the brain radically affects how it processes sensory information. How does this ongoing brain activity interact with the processing of external stimuli? Spontaneous oscillations in the alpha range are thought to inhibit sensory processing, but little is known about the psychophysical mechanisms of this inhibition. We recorded ongoing brain activity with EEG while human observers performed a visual detection task with stimuli of different contrast intensities. To move beyond qualitative description, we formally compared psychometric functions obtained under different levels of ongoing alpha power and evaluated the inhibitory effect of ongoing alpha oscillations in terms of contrast or response gain models. This procedure opens the way to understanding the actual functional mechanisms by which ongoing brain activity affects visual performance. We found that strong prestimulus occipital alpha oscillations—but not more anterior mu oscillations—reduce performance most strongly for stimuli of the highest intensities tested. This inhibitory effect is best explained by a divisive reduction of response gain. Ongoing occipital alpha oscillations thus reflect changes in the visual system's input/output transformation that are independent of the sensory input to the system. They selectively scale the system's response, rather than change its sensitivity to sensory information. ■

INTRODUCTION

The brain is never completely at rest. Numerous studies have demonstrated that high levels of internally generated neuronal activity occur continuously, even in the absence of any external stimulation. These activity patterns are referred to as "ongoing," "spontaneous," "resting," or "prestimulus," as opposed to responses evoked by and, thus, following sensory events. Spontaneous brain activity is not mere neural noise but rather is organized in coherent patterns reflecting the underlying local (Arieli, Sterkin, Grinvald, & Aertsen, 1996) and global (Fox, Snyder, Zacks, & Raichle, 2006) brain architecture. It accounts for a substantial portion of the variability of evoked responses and behavioral performance (Fox, Snyder, Vincent, & Raichle, 2007; Fox et al., 2006), constituting what has been termed a neural context for information processing in the brain (Mcintosh, 1999).

In electrophysiological recordings, spontaneous neuronal activity takes the form of oscillations—wave-like signal fluctuations that reflect rhythmic variations of membrane potentials, which in turn are associated with periodic fluctuations of neuronal excitability (Buzsáki & Draguhn, 2004). The most prominent and most studied brain oscillation in the awake state is the so-called alpha rhythm (Dalal et al., 2011) at a frequency of about 10 Hz. Numerous studies have demonstrated that these spontaneous oscillations interact with processing of sensory information. Specifically, strong spontaneous alpha oscillations over the visual cortex just before stimulus presentation impair detection of visual stimuli (Busch, Dubois, & Vanrullen, 2009; Van Dijk, Schoffelen, Oostenveld, & Jensen, 2008; Hanslmayr et al., 2007; Ergenoglu et al., 2004) and TMS-induced phosphenes (Romei et al., 2008). In a similar fashion, alpha oscillations over the somatosensory cortex (also referred to as the rolandic mu rhythm) impair somatosensory detection (Haegens, Händel, & Jensen, 2011; Haegens, Nácher, Luna, Romo, & Jensen, 2011; Jones et al., 2010; Zhang & Ding, 2010). Furthermore, it is becoming increasingly clear that this inhibition can be controlled via attention. Indeed, several studies have shown alpha oscillations increase over the cortical areas representing unattended or task-irrelevant information. Thus, when paying attention to one visual hemifield, alpha oscillations increase over brain areas representing the unattended hemifield (Busch & VanRullen, 2010; Kelly, Lalor, Reilly, & Foxe, 2006; Worden, Foxe, Wang, & Simpson, 2000). Other studies have demonstrated that alpha-mediated inhibition may be a general gating mechanism of cortical processing. For instance, when participants are instructed to attend either to the visual or to the somatosensory modality, alpha oscillations increase over the task-irrelevant sensory cortices (Foxe, Simpson, & Ahlfors, 1998). Furthermore, during working memory tasks, alpha oscillations serve to inhibit the processing of potentially distracting information (Bonnefond & Jensen,

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2012; Busch & Herrmann, 2003). Together, these findings demonstrate that alpha oscillations are not an idling rhythm, as was originally thought (Pfurtscheller, Stancák, & Neuper, 1996), but rather suggests that they allow a dynamic functional inhibition of task-irrelevant cortical processing (Foxe & Snyder, 2011; Mathewson, Beck, Fabiani, Ro, & Gratton, 2011; Jensen & Mazaheri, 2010; Klimesch, Sauseng, & Hanslmayr, 2007).

Although there is a strong consensus on the inhibitory effect of alpha oscillations, the functional mechanism by which they exert this effect has remained speculative. Some authors have suggested that alpha oscillations inhibit input to the visual system (Lange, Oostenveld, & Fries, 2013; Romei, Gross, & Thut, 2010), specifically by affecting the visual threshold (Ergenoglu et al., 2004). Others have surmised that alpha oscillations inhibit communication of the visual system with other areas (Van Dijk et al., 2008). It is important to note that addressing this issue requires moving beyond the mere demonstration that correct and incorrect behavioral responses are characterized by differential levels of prestimulus alpha power or that prestimulus power affects behavioral performance. Because functional mechanisms of inhibition are largely theoretical concepts that cannot be observed directly (as opposed to hit rates or oscillatory power), identification of the functional mechanisms by which alpha oscillations impair performance requires a formal model of how these empirical data are related to candidate inhibitory mechanisms.

In this study, we consider two candidate functional mechanisms by which alpha oscillations may inhibit perception: contrast gain and response gain (Figure 1). These mechanisms refer to specific changes in the psychometric function (PMF), which describes the relation between stimulus intensity (e.g., stimulus luminance) and how well these stimuli are perceived. Both gain mechanisms have been studied as mechanisms underlying effects of spatial attention (Ling & Carrasco, 2006; Reynolds, Pasternak, & Desimone, 2000; McAdams & Maunsell, 1999) and adaptation (Kohn, 2007) on neuronal responses and behavioral performance. The signature of a contrast gain effect is a horizontal shift of the PMF (Figure 1B, left curves), reflecting a change in threshold and in the stimulus intensity required to achieve a given level of performance. Thus, a contrast gain effect is equivalent to a change in the effective physical intensity of the stimulus. If alpha oscillations impair perception by reducing contrast gain, this would translate to a rightward shift in the PMF. Accordingly, the strongest effects of alpha power should be found for stimuli of intermediate intensity within the dynamic portion of the PMF with little or no effect at higher stimulus intensities. Alternatively, alpha oscillations may inhibit performance by means of response gain modulation—a divisive suppression that is proportional to the response of the system (Figure 1B, right curves). This effect is characterized in particular by a reduction of peak performance at highest signal intensities and, notably, does not lead to a change in threshold. The distinction between contrast gain and response gain modulation is important, because an effect on contrast gain is equivalent to a change in the physical intensity of the input signal, whereas an

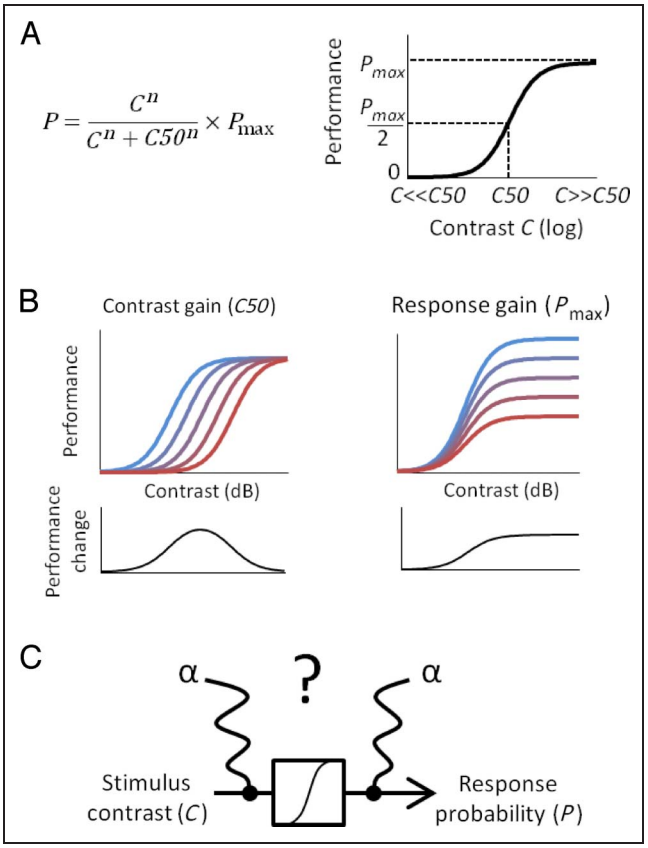

Figure 1. Two putative effects of alpha oscillations on performance. (A) The observer's response in a detection task is driven by the transformation of input contrast C into response probability P. This transformation is described parametrically in a model of the PMF with parameters:

- P_{max}: maximum performance for strongest contrast stimuli. P_{max} is an index of the result or output of the transformation process.
- $C50$: threshold contrast, the input contrast required to achieve half maximum performance ($P_{max}/2$).
- n: the slope (steepness) of the PMF (not illustrated in the figure).

Note how the output of the ratio $C^n/(C^n + C50^n)$ changes with changing values of C. It is close to zero when C is small in comparison with $C50$ ($C^n/(C^n + C50^n) \approx 0$ with $C << C50$). It is equal to 0.5 when contrast C is equal to $C50$, and it is close to 1 when C is largely greater than $C50$ ($C^n/(C^n + C50^n) \approx 1$ with $C >> C50$). (B) We model the effects of alpha oscillations on performance by allowing each of the two variables $C50$ and P_{max} to vary with alpha amplitude. Allowing $C50$ to vary is equivalent to changing the input contrast C. This modulation is called contrast gain and allows the PMF to shift horizontally on the contrast axis (left curves). Note that an effect of alpha oscillations on contrast gain would be most pronounced for stimuli with a contrast close to threshold contrast ($C50$). Alternatively, allowing P_{max} to vary is equivalent to changing output performance. This modulation is called response gain and allows the upper asymptote of the PMF to shift vertically along the performance axis (right curves). Note that an effect of alpha oscillations on response gain would be most pronounced for stimuli with maximal contrast. The slope n (not illustrated here) is assumed unaffected by alpha oscillations in these models. (C) We can equivalently represent the stimulus–response transformation graphically and locate the effects of alpha oscillations either before (contrast gain) or after (response gain) the nonlinear transformation of contrast (C) into response probability (P).

effect on response gain reflects a change in the visual system's input/output transformation that is independent of the input to the visual system and thus selectively scales the system's response.

In previous studies, the effect of prestimulus alpha oscillations on perception was evaluated with stimuli of a single near-threshold contrast (e.g., Van Dijk et al., 2008; Hanslmayr et al., 2007; Ergenoglu et al., 2004), making it impossible to distinguish between effects on contrast gain or response gain, because both contrast gain and response gain lead to performance changes at near-threshold stimulus intensities (Figure 1B, bottom curves). Here, we measured the full PMFs of naive human observers while they performed a visual detection task on a wide range of stimulus intensities and compared four different models of how spontaneous oscillations recorded with EEG might affect performance: a contrast gain model, a response gain model, a mixed model combining contrast and response gain, and a null model of no effect of oscillations on performance. We demonstrate that alpha oscillations impair visual perception via a response gain mechanism in most observers. Furthermore, to establish the neural specificity of this effect, we dissociated occipital visual alpha oscillations from the more anterior somatosensory mu rhythm using independent component analysis (ICA; Delorme & Makeig, 2004) and showed that mu oscillations had no effect on visual performance, suggesting that the inhibitory effect of alpha oscillations on performance is modality specific.

METHODS

Participants

Twelve participants (eight women, 27 ± 4.7 years, 11 right-handed), with normal or corrected-to-normal vision and with no reported history of neurological or psychiatric disorders, were analyzed (17 participants were recorded, but 5 were excluded because of performance at highest signal intensity below 60%). All participants gave signed informed consent. All experimental procedures were approved by the Charité University Medicine Ethics Committee.

Stimuli and Procedure

The experiment was written in MATLAB (The Mathworks, Natick, MA) using the Psychophysics Toolbox 3 (Brainard, 1997). Stimuli were presented on a black background (1.1 Cd/m^2) using a cathode ray tube monitor operated at 100 Hz and situated in a dark room.

A small central fixation cross and two thin vertical bars on each side of the fixation marking the locations were target stimuli could appear were constantly visible of the screen (Figure 2). Target stimuli were small gray dots (diameter: 7° visual angle) presented for 10 msec at an eccentricity of 5.7° visual angle either to the left or right of the fixation cross. The ISI ranged from 2100 to 3300 msec. Participants were instructed to maintain fixation at the center of the screen and to press a key whenever they detected a target. Accuracy was emphasized over speed. Correct detections were counted when a key press occurred between 200 and 1500 msec after a target (only 2.6% of all responses occurred after 1000 msec).

Each participant completed first one session, during which the individual target luminance threshold for 50% detection was determined with an adaptive staircase procedure (Watson & Pelli, 1983). During this part of the experiment, some stimuli (2/5) were presented at the maximum intensity allowed by the screen so that participants would also experience strong stimuli similar to those they would see later during the main part of the experiment (see Results section). The estimated threshold was then used for the main part of the experiment to spread seven different luminance levels at −3, −1, −0.5, 0, 0.5, 1, 3 dB around threshold. These luminance levels were presented in random order so that each level was presented 160 times (1120 trials total).

Recordings and Preprocessing

We recorded continuous EEG with a 64-channel ActiveTwo system (Biosemi, Amsterdam, The Netherlands). Two additional electrodes, CMS (common mode sense) and DRL (driven right leg), were used as reference and ground. Details of the circuitry can be found on the Biosemi Web site (www.biosemi.com/faq/cms&drl.htm). Electrodes were placed according to the international 10–10 system.

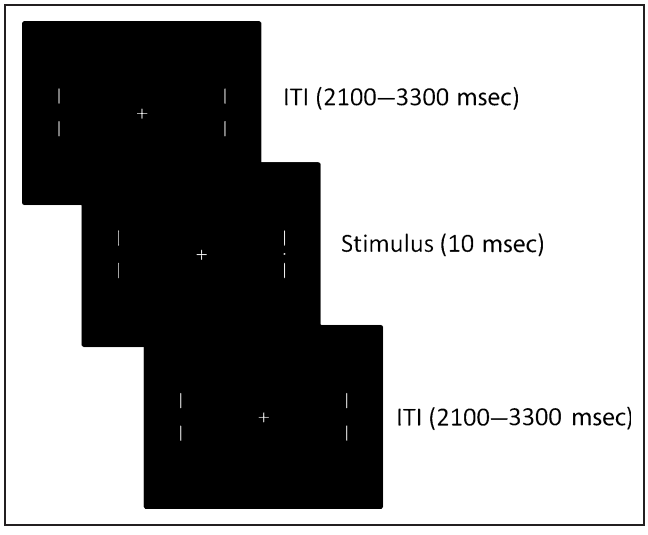

Figure 2. Stimuli. A fixation cross surrounded by two pairs of placeholders (7° eccentricity) was constantly present in the center of the screen. At unpredicable times, a stimulus appeared randomly to the left or right between placeholders (here to the right), and participants signaled detection by a button press.

The horizontal and vertical EOGs were recorded by attaching additional electrodes at the outer canthi of both eyes and below the right eye. All signals were digitized at 512 Hz, 24-bit A/D conversion.

The EEGLAB toolbox version 11 running on MATLAB (R2010b) was used to process and analyze the data (Delorme & Makeig, 2004). Data were converted to an average reference offline, band-pass filtered between 1 and 45 Hz, and epoched from −1000 to 1000 msec relative to target onset. Data were screened manually for major artifacts, and trials were discarded if a blink occurred within ±100 msec of target onset. This trial rejection served to exclude trials on which eyes were closed during stimulus presentation. Artifacts because of eye blinks at other latencies were removed with ICA (see below). Global field power was computed as the standard deviation of all electrodes at each time point (Murray, Brunet, & Michel, 2008).

Independent Component Analysis, Source Localization, and Clustering

Channel EEG data reflect a mixture of electric currents originating from various brain and nonbrain sources. To separate sources originating from oscillatory alpha generators located in occipital cortex from mu generators around the somatosensory cortex and from nonneural generators like muscle activity, EEG data were transformed using infomax ICA (Delorme & Makeig, 2004). ICA identified a set of statistically independent components, each represented by a component time course and spatial filter (topography). The cerebral sources of these components were localized with equivalent dipole fitting (dipfit 2.2 implemented in EGLAB) using a standard boundary element head model.

We then used a three-step procedure to isolate alpha and mu components within and across participants. (1) We discarded components that were not well modeled by a dipolar source and for which the residual variance after subtraction of the forward modeled fitted dipole from the data exceeded 15%. By doing this, we effectively discarded most of the nonneural or noise components. A total of 141 components from the 12 participants were included in the subsequent analyses. (2) We used a *k*-means clustering algorithm (implemented in EEGLAB) to group independent components with similar characteristics across participants according to their topographies, Fourier power spectra, and dipole location (with equal weights). The number of clusters requested was set to 12, so that an average of one component per participant would be included in each cluster. Tests with larger or smaller numbers of requested clusters yielded qualitatively similar results. (3) We merged all posterior alpha clusters satisfying two criteria: the presence of a peak in the alpha band in the power spectrum and a posterior location (according to scalp maps and dipole fits). This merged cluster is subsequently referred to as the occipital alpha cluster. Similarly, we merged components that had a peak in the alpha frequency band and were located more anteriorly, close to the central sulcus, into a final mu cluster. Subsequently, all the analyses were performed on these clusters.

In addition, we also grouped alpha clusters by laterality (i.e., whether the x coordinate of the centroid of the cluster was positive [right-lateralized cluster] or negative [left-lateralized cluster] to examine whether effects of alpha oscillations on performance were lateralized). Specifically, we tested whether detection of lateralized stimuli was influenced by the amount of lateralization of alpha power (i.e., the difference in alpha amplitude between ipsi- and contralateral clusters).

Time–Frequency Analysis

Oscillatory power was computed by means of a continuous wavelet transform of single-trial data for the frequency range from 3 to 40 Hz. The length of the wavelet increased linearly from one cycle at 3 Hz to five cycles at 40 Hz. This modified wavelet transform is typically used to optimize the tradeoff between temporal resolution at lower frequencies and stability at higher frequencies. At each time t and frequency f, the result of the wavelet transform for trial k is a complex number, in which A represents the amplitude of the signal and φ its phase:

$$A_{k(t,f)}e^{i\varphi_{k(t,f)}}$$

For each participant, single-trial prestimulus alpha (or mu) amplitude was quantified as the amplitude averaged over a time–frequency window of −400 msec and −200 msec, 8 and 12 Hz for all components included in the alpha (or mu) cluster for that participant. This time window was chosen so that our measure of prestimulus amplitude would not be affected by the poststimulus response. We then used a quintile split on the distributions of alpha (and mu) amplitudes to sort trials into five bins of increasing alpha amplitude with a roughly equal number of trials in each bin.

Modeling

The goal of this study was to characterize the effect of prestimulus alpha oscillations in terms of contrast gain and response gain mechanisms. The signature of a contrast gain effect is a horizontal shift of the PMF, which describes the relation between stimulus intensity (e.g., stimulus luminance) and how well these stimuli are perceived (Figure 1B, left curves). A contrast gain effect reflects a change in the system's threshold and, thus, is equivalent to a change in the effective physical intensity of the stimulus. If alpha oscillations impair perception by reducing contrast gain, this would translate to a rightward

shift in the PMF. Accordingly, the strongest effects of alpha power should be found for stimuli of intermediate intensity within the dynamic portion of the PMF with little or no effect at higher stimulus intensities. Alternatively, response gain is proportional to the response of the system (Figure 1B, right curves). Thus, this effect is characterized by a reduction of peak performance at highest signal intensities and, notably, does not lead to a change in threshold.

To distinguish between these gain mechanisms, we fitted PMFs to performance at all stimulus intensities using maximum likelihood, as implemented in the Palamedes toolbox for MATLAB (Prins & Kingdom, 2009) using a Naka-Rushton contrast response model (Ling & Carrasco, 2006; Albrecht & Hamilton, 1982).

$$P = P_{max} \times \frac{C^n}{C^n + C50^n} \quad (1)$$

where P represents proportion of correct detections, C is the contrast level of the stimulus, *C50* is the contrast at half the maximum performance (threshold), n is an exponent that determines the steepness of the function (slope), and $P_{\max}$ is the maximum performance level. In a first descriptive analysis, we set threshold, slope, and maximum performance parameters free and obtained best estimates for each of the five bins of alpha amplitude. We obtained confidence intervals using 400 parametric bootstrap simulations (Wichmann & Hill, 2001). This allowed us observing qualitatively how parameters varied as a function of alpha amplitude but did not allow further mechanistic inference. We therefore then turned to our hypothesis-driven approach, as follows.

We first estimated the best-fitting parameters to the average performance, without splitting trials based on alpha amplitude. We fixed these parameters and named this model the "null model." We then measured the goodness of fit of this null model by computing the likelihood of observing the performance we observed in each of the alpha amplitude bins under this model. In other words, with the null model, we measured the likelihood of observing the data from each alpha amplitude bin if alpha had no effect on performance. We used Akaike Information Criterion (AIC) here and for all subsequent measures of goodness of fit. AIC is a better measure than likelihood because it accounts for changes in the number of free parameters in the models (Burnham, 2004). Subsequently, we fit three other models, freeing parameters in turn as follows to account for potential effects of alpha oscillations on performance. In the contrast gain model, we allowed only *C50* to vary freely to account for performance changes across alpha bins. As mentioned already, under the contrast gain model, only horizontal shifts are allowed to account for performance changes (see Figure 1, left curves). In the response gain model, we allowed $P_{\max}$ to vary freely to account for performance changes across alpha bins. As mentioned already, in the response gain model, only vertical (multiplicative) shifts of performance are allowed to account for performance changes (see Figure 1B, right curves). Finally, we also tested a "full model," incorporating both contrast and response gain modulation, that is, in which both *C50* and $P_{\max}$ were allowed to vary.

To decide which model was best, we normalized the AIC of each model with respect to the AIC of all the other models in the set to obtain Akaike weights (Burnham, 2004). This measure reflects the weight of evidence that the data provide in favor of each model in the set. However, because Akaike weights are a relative measure that depends on the set of tested models, it does not provide any information about the size of the observed effect. A large Akaike weight in favor of one model means that it is the best of the tested models but does not mean that the effect it models is large. Furthermore, it provides no indication as to the direction of a potential effect. To evaluate the size of the measured effect, we also obtained confidence intervals around the free parameter estimates of the best model using 400 parametric bootstrap simulations. We subsequently evaluated whether these bootstrapped confidence intervals overlapped between the most extreme alpha bins and considered that nonoverlapping confidence intervals indicated significant modulation of the given parameter.

To confront this component space analysis with more conventional channel space analyses, we isolated peak alpha channels (PO3, POz, and PO4), as well as central channels typically used to measure mu activity (C3 and C4), and performed the same analysis as above on trials split according to average amplitude in the 8–12Hz, 400–200 msec time–frequency window on these channels.

Previous studies have shown that attention-induced lateralization of alpha oscillations during visual detection affects performance (Kelly, Gomez-Ramirez, & Foxe, 2009; Kelly et al., 2006; Worden et al., 2000). These studies have generally reported that higher alpha amplitude over ipsilateral occipital cortex enhances performance, whereas higher alpha activity over contralateral occipital cortex hinders performance. We thus separated alpha clusters by laterality and measured whether amplitude in these lateral clusters affected detection performance for ipsi- or contralateral stimuli differently. To do so, we computed a trial-by-trial laterality index by taking the ipsi- minus contralateral alpha amplitude (with respect to the upcoming stimulus). Positive values of this laterality index indicate higher ipsi- than contralateral prestimulus alpha and are expected to lead to better performance than negative values. We split trials in five bins of laterality index and used the same procedure as above to evaluate which of the four models presented above best explained performance changes and whether these performance changes were significant.

We also looked for potential effects of the phase of ongoing alpha oscillations on performance. If there was a preferred phase at which performance was best in this

paradigm, a model that allows varying performance as a function of phase should fit the data better than a model that does not. We used the same rationale as with amplitude, splitting single trials in bins according to the phase of the signal in the 8–12 Hz frequency range at stimulus onset (at 0 msec). Trials were sorted in bins according to the extracted phase. Because the best phase angle does not need to be identical across participants (Busch & VanRullen, 2010; Busch et al., 2009) and to summarize the data across participants, we set the bin with best performance to be the first one, so that, similar to the amplitude modeling, the best bin would always be the same across participants. We used six bins here to be able to compare the best (first) bin with its opposite phase angle (third) bin. We used the same procedure as above to evaluate which of the four models presented above best explained performance changes and whether these performance changes were significant.

RESULTS

We measured ongoing alpha oscillations with EEG while human participants performed a visual detection task. Target stimuli (small gray dots on a black background) were presented for 10 msec at one of seven luminance levels that were logarithmically spaced around the 50% detection threshold.

Behavior

Overall, behavioral performance followed an expected sigmoid shape. Importantly, performance did not reach ceiling level at highest stimulus intensities but rather peaked at 83 ± 2.9% (*SEM*), allowing for potential variations of maximum performance (response gain). Average RTs were 555 ± 16 msec (*SEM*). Noteworthy, stimuli presented at the 50% detection threshold, as estimated by the initial staircase procedure, resulted in only 39% (±4%, *SEM*) correct performance in the main part of the experiment. We suspect that this discrepancy was because of the different range of stimulus intensities used in the staircase and in the main experiment and a resulting change in observers' decision criterion. During the staircase, most of the stimuli were presented at the current threshold estimate and were difficult to detect, whereas only 2/5 of all stimuli were clearly visible. By contrast, about half of the stimuli were above threshold and well detectable during the main experiment. Thus, observers may have been led to believe that the task had gotten easier and report predominantly the high-intensity targets, resulting in more conservative criterion during the main experiment than during the staircase. This change in decision criterion would explain why fewer targets at the 50% intensity were reported during the main experiment.

Stimulus-evoked Activity

To check for the presence of normal stimulus-evoked activity, we averaged ERPs at channels PO3, PO4, and POz across all stimulus intensities for trials where the stimulus was detected. The first peak response occurred at 222 msec poststimulus with an average amplitude of −1.9 μV, followed by a peak at 439 msec with an average amplitude of 1.4 μV. These ERP components were absent for undetected stimuli.

Detection Performance Depends on Prestimulus Alpha Amplitude

Before modeling the effects of alpha oscillations on performance, we visually examined differences in prestimulus oscillatory amplitude for correct and incorrect trials separately. Figure 3 shows the power spectrum at peak alpha channels (PO3, POz, PO4) for detected and undetected stimuli separately (independent of stimulus intensity). The comparison of prestimulus activity for detected and undetected trials showed, as expected, that undetected targets were associated with higher prestimulus amplitudes in the alpha range (8–12 Hz, −400 to −200 msec, two-tailed paired *t* tests: $t(11) = 2.34$; $p < .039$). At channels

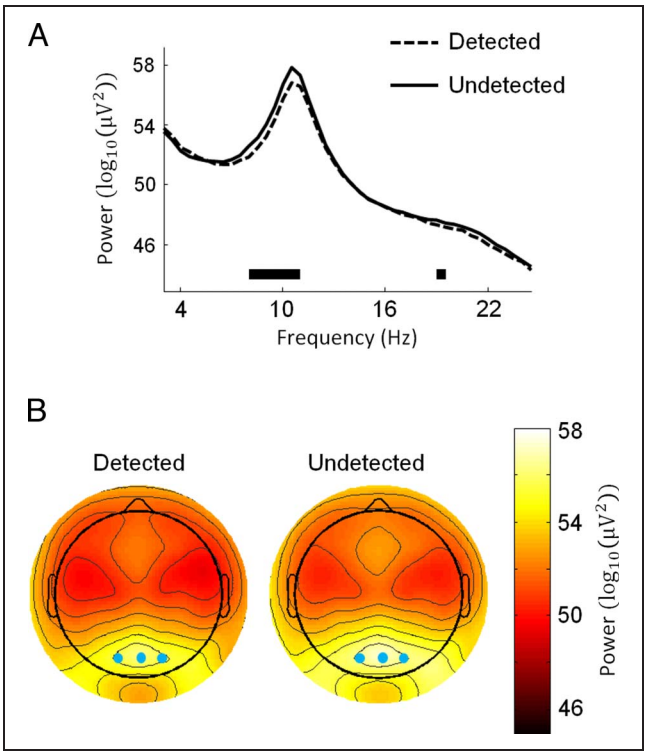

Figure 3. Comparison of prestimulus alpha amplitude for detected and undetected trials at channel level. (A) Power spectrum averaged across channels POz, PO3, and PO4 for Detected and Undetected trials. The black bars show significant differences between Detected and Undetected trials (paired *t* tests, $p < .05$ at two or more consecutive frequencies). (B) Topographical maps of power spectrum at 10 Hz. The three highlighted electrodes are those used for the average spectrum in A.

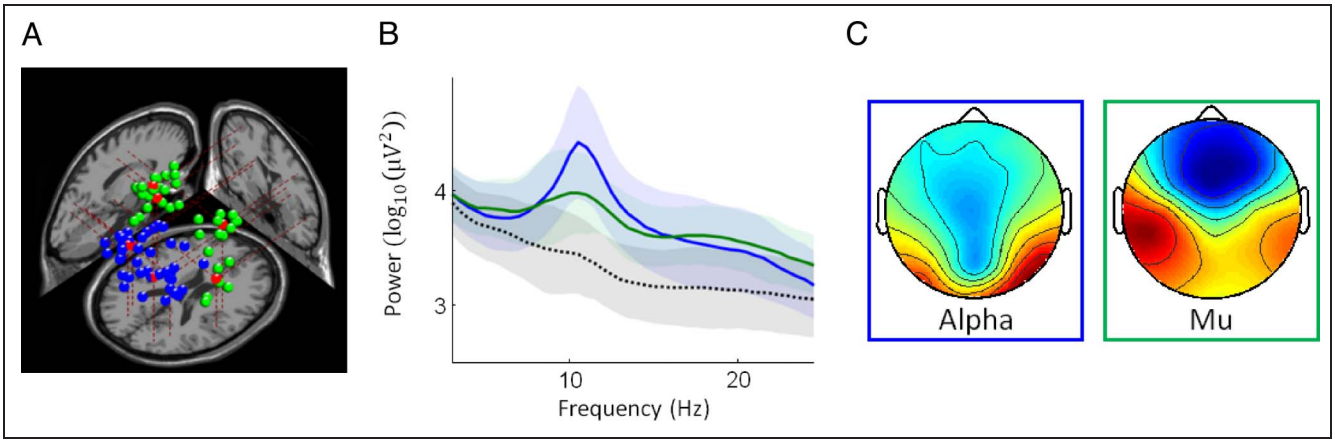

Figure 4. Component cluster properties. Twelve clusters were created from the 141 components of all participants. Four of these clusters were included in each of the larger alpha and mu clusters. (A) Equivalent dipole locations for all components included in the occipital alpha (blue dots) and anterior mu (green dots) cluster. Red dots are the centroids of each of the original clusters. Red dotted lines show the projections of each centroid on the sagittal, coronal, and frontal slices of a template brain. (B) Average power spectrum for the alpha (blue), mu (green), and all other clusters (black dotted line). The gray areas show the standard deviation around the average across components. (C) Average topographies of the two component clusters.

classically used to analyze mu activity (C3 and C4), the difference between Hit and Miss trials was not significant (8–12 Hz, −400 to −200 msec, two-tailed paired t tests $t(11) = 1.73$; $p > .1$). Note that, because of volume conduction, activity at the channel level is a mixture of multiple intracerebral sources. To achieve better signal separation, all subsequent analyzes are performed on independent components. Results at the channel level are reported only for comparison with previous studies employing channel level analyses.

Alpha Components Clustering

We first separated 12 clusters of independent components (Figure 4). The numbers of participants and components included in each cluster are shown in Table 1. Four clusters met the criteria defined for classification as alpha cluster (i.e., having a peak at 10 Hz and a posterior source; see Methods). These are highlighted in blue in Figure 4. The final occipital alpha cluster contained 44 components from 11 participants (one participant had no visible alpha activity and thus no component in the final alpha cluster). Four clusters met the criteria for inclusion into the final mu cluster (having a peak at 10 Hz and a central location; see Methods). These are highlighted in green in Figure 4. Three of these clusters also showed another peak in the lower beta band (18 Hz, Clusters 3, 7, and 10 on Figure 4C). The final mu cluster contained 39 components from 11 participants. All subsequent analyses were performed separately for both the alpha and the mu cluster.

Clusters of components were lateralized (i.e., each cluster centroid was clearly localized either in the right or in the left hemisphere, see projections of the centroids of the clusters on the horizontal slice on Figure 4A). We thus also isolated alpha and mu clusters predominantly located to the right or to the left of the brain and performed subsequent analyses on these lateralized alpha and mu component clusters, separately.

The Relative Level of Prestimulus Occipital Alpha Amplitude Inhibits Detection Performance in a Linear Fashion

We examined the effect of prestimulus occipital alpha amplitude on performance and show that lower/higher alpha amplitude is generally associated with better/worse detection (results for the mu cluster are presented below). Figure 5A shows detection performance broken down by prestimulus alpha amplitude bin at each signal level. This result suggests that greater alpha amplitude is associated with worse performance and that this effect is most pronounced at the highest stimulus intensity. At this intensity, the difference in performance for the bins with strongest and weakest alpha power amounted to an average difference of 15 ± 5% (*SEM*) correct detection.

Others have reported that alpha amplitude has a quadratic (inverted U-shape) effect on performance

Table 1. Number of Independent Components and Participants in All Clusters

Cluster #	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*	*Alpha Cluster (2 + 4 + 5 + 11)*	*Mu Cluster (1 + 3 + 7 + 10)*
Nb participants	5	7	8	5	9	5	9	11	7	7	8	10	11	9
Nb components	7	11	11	8	12	8	13	16	12	8	13	22	44	39

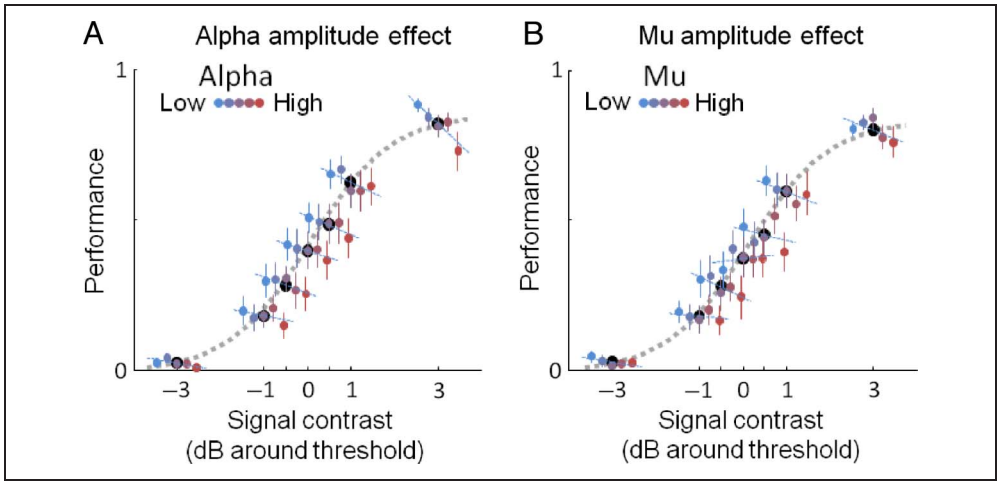

Figure 5. Linear effects of alpha and mu amplitude on performance at all signal contrast intensities. (A) Detection performance at all levels of contrast for the five bins of alpha amplitude. At each of the seven levels of signal contrast, the large black marker shows average performance. The gray-dotted curve illustrates a PMF fit through this average performance. At each signal contrast, the data are also split according to alpha amplitude bins. The dashed blue lines show the best-fitting polynomial through performance for each amplitude bin. (B) Same as A for mu amplitude.

(Lange, Halacz, van Dijk, Kahlbrock, & Schnitzler, 2012; Zhang & Ding, 2010), having a maximum effect not at highest but at intermediate alpha amplitude levels. To evaluate whether performance varied in a linear or a quadratic manner as a function of alpha amplitude, we used a hierarchical testing of the variance explained by two nested models at each signal contrast. The full regression model included both a quadratic and a linear effect of alpha amplitude bin on performance, and the simpler regression model included only the linear effect. The linear model best accounted for the effects of alpha amplitude on performance at all levels of stimulus intensity (Figure 5A, all $F(1, 2) < 3.2$, $p > .2$), showing that alpha amplitude was linearly related to the decrease in performance.

Modeling the Effect of Occipital Alpha Amplitude on Detection Performance

To further characterize the psychometric contrast response functions of the observers, we first used a descriptive approach, examining how the parameters of the PMF varied as a function of alpha amplitude. We modeled PMFs separately for each alpha amplitude bin in each

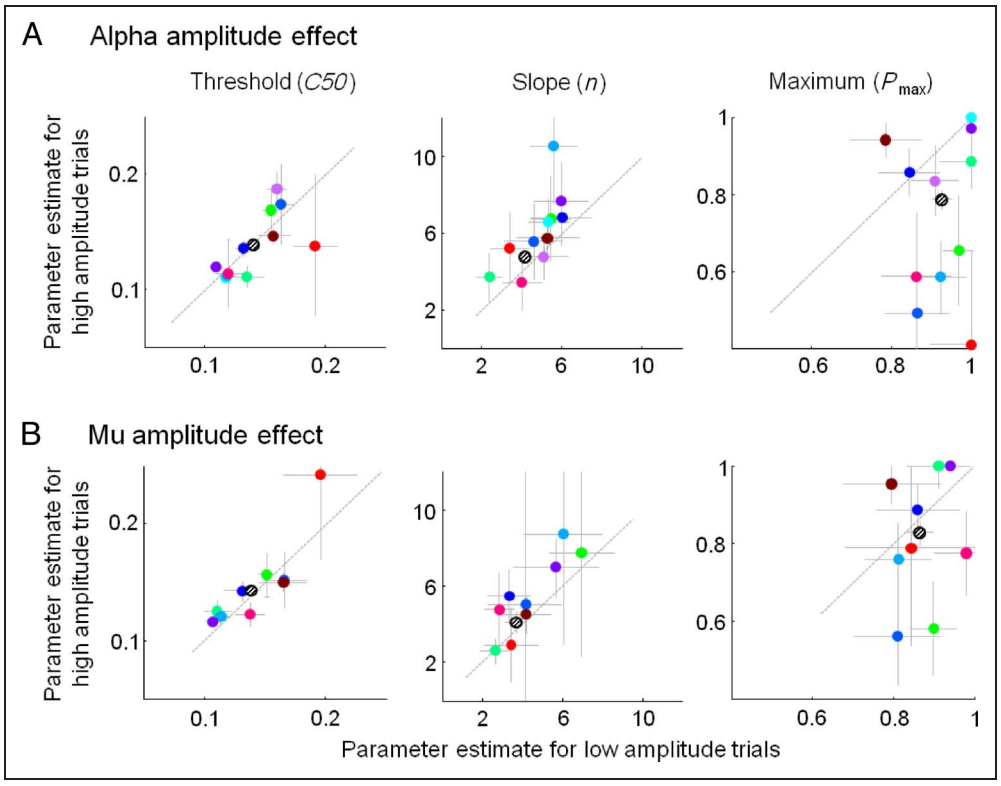

Figure 6. Bootstrapped model parameter estimates of the model fit with all free parameters. (A) Model parameters estimated separately for trials with lowest (x axis) and highest (y axis) prestimulus alpha amplitudes. Colored markers represent single participants; individuals are represented by the same color across panels. In each panel, the black marker shows the estimates for the pooled performance across all participants. The dashed line represents the equality diagonal. Points falling on this diagonal indicate that prestimulus alpha amplitude had no effect on a given parameter, whereas points above/below the diagonal indicate higher parameter estimates for high/low prestimulus amplitudes. Error bars indicate the bootstrapped confidence intervals around the parameter estimates. (B) Same as A for mu amplitudes.

observer using a standard Naka-Rushton equation (Equation 1) with three free parameters: threshold (*C50*), slope (n), and maximum performance (P_{max}). As shown in Figure 6A, participants' threshold and slope parameter estimates for highest and lowest amplitude bins were scattered along the equality diagonal, indicating similar thresholds and slopes for trials with strong and weak prestimulus alpha amplitude. By contrast, the maximum performance parameter estimates were located below the equality diagonal in 6 of 11 participants, indicating lower maximum performance on trials with strong prestimulus occipital alpha power. For these participants, the modeled maximum performance was up to 60% lower in high alpha amplitude trials than in low alpha amplitude trials, suggesting that a response gain model is likely to describe the data best.

To formally test this hypothesis, we compared four different models of how alpha amplitude could affect performance. Figure 7 shows the results of the model comparison based on performance pooled across participants. Each panel of Figure 7A shows the best fitting curves from each model as well as the residuals (difference between data and model). Figure 7B shows Akaike weights (also shown in Table 2, bottom row) for each model, confirming that the response gain model best fitted the data. We assessed the size of the effect by evaluating whether the P_{max} estimates were significantly different between the most extreme amplitude bins. The results are shown on the scatter plot of Figure 7C and Table 2 for all participants separately and for the pooled data across participants. This analysis shows that the response gain modulation was significant at the group level. The results at the individual participant level were more nuanced. The response gain model was best and significant in 4 of the 11 participants analyzed. One was best fitted by the response gain model but showed an effect in the opposite direction, five other participants were best fitted by the null model, and one other by the contrast gain model (although in the unexpected direction, threshold *C50* was lowest with high levels of alpha in this participant). Together, these analyses support the hypothesis that prestimulus occipital alpha amplitude influences behavior

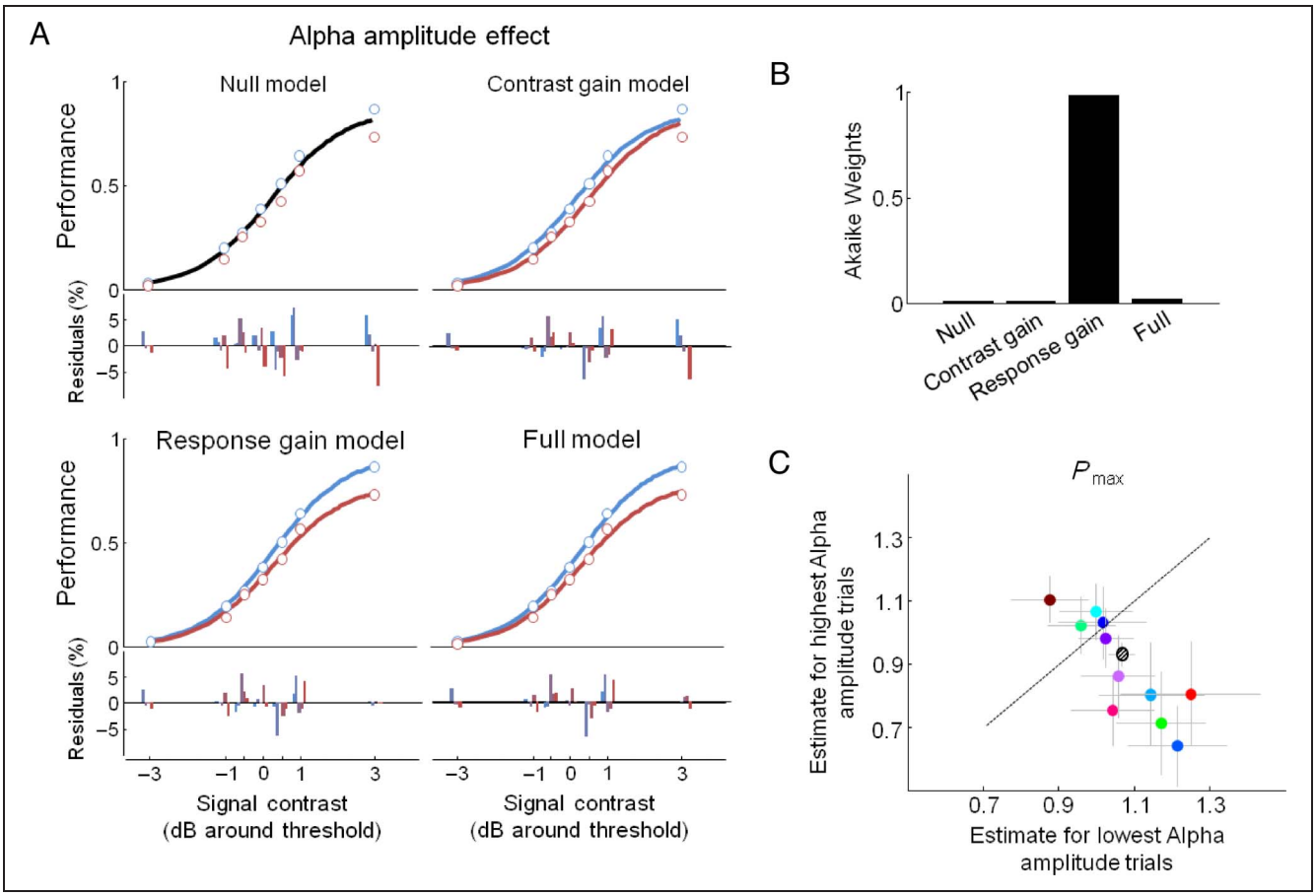

Figure 7. Comparison of four models of the effect of alpha amplitude on detection performance. (A) The four panels show the maximum likelihood estimate of performance variations according to the model mentioned above each panel. At each of the seven levels of signal contrast, the two markers show average performance for the highest (magenta) and lowest (blue) prestimulus alpha amplitude quintile (other quintiles are not shown for clarity). The bar plot under each panel shows the residuals of the fit (the difference between the estimated and actual performance at each contrast level and for each alpha amplitude quintile, including those not shown above). (B) Akaike weights for the tested model set on the data pooled across participants. (C) Response gain parameter (P_{max}) estimated separately for trials with lowest and highest alpha amplitude quintiles in each participant. Same conventions as for Figure 6 apply.

Table 2. Comparison of the Four Models for Alpha and Mu Cluster Amplitudes

	Akaike Weights for Each Model					*Lowest Amplitude Bin*			*Highest Amplitude Bin*			
Participant	*Null*	*Contrast Gain*	*Response Gain*	*Full*	*Best Model*	*−SE*	*Mean*	*+SE*	*−SE*	*Mean*	*+SE*	*Significance*
Alpha Cluster												
1	0.00	0.13	0.82	0.05	Response	1.07	1.17	1.28	0.55	0.71	0.87	*
2	0.54	0.42	0.04	0.00	Null							
3	0.89	0.04	0.06	0.01	Null							
4	0.04	0.17	0.76	0.03	Response	1.02	1.14	1.27	0.64	0.80	0.97	*
5	0.00	0.04	0.95	0.02	Response	1.09	1.21	1.34	0.50	0.64	0.78	*
6	0.97	0.01	0.01	0.00	Null							
7	0.88	0.06	0.06	0.00	Null							
8	0.02	0.01	0.97	0.01	Response	0.93	1.04	1.16	0.62	0.74	0.86	*
9	0.71	0.02	0.26	0.01	Null							
10	0.18	0.28	0.52	0.01	Response	0.78	0.88	0.98	1.04	1.10	1.17	−*
11	0.18	0.58	0.22	0.01	Contrast	1.12	1.55	1.98	0.25	0.61	0.97	−*
All	0.00	0.00	0.98	0.01	Response	1.03	1.07	1.11	0.87	0.91	0.95	*
Mu Cluster												
1	0.02	0.03	0.92	0.03	Response	1.01	1.12	1.24	0.58	0.73	0.88	*
2	0.97	0.01	0.02	0.00	Null							
3												
4	0.88	0.07	0.05	0.00	Null							
5	0.32	0.24	0.40	0.04	Response	0.99	1.12	1.25	0.76	0.88	1.01	
6	0.92	0.04	0.03	0.01	Null							
7	0.79	0.01	0.20	0.00	Null							
8												
9	0.96	0.03	0.02	0.00	Null							
10	0.49	0.21	0.28	0.02	Null							
11	0.03	0.82	0.13	0.02	Contrast	0.72	0.83	0.94	1.00	1.08	1.16	*
All	0.57	0.23	0.17	0.03	Null	0.98	1.02	1.06	0.91	0.95	0.99	

The top and bottom tables show the results of the model fits to performance split according to prestimulus power in the alpha and mu cluster, respectively. Shaded cells show the best model for each participant. In each table, the last row shows the modeling results on average performance of the entire sample. The right part of the table shows estimates and confidence intervals for the lowest and highest amplitude trial bins for the free parameter of the best model (note that there is no free parameter in the Null model, hence rows where the Null model was the best one are empty). Nonoverlapping confidence intervals are marked in the last column (* indicates change in the hypothesized direction, −* indicates change in the opposite direction). Participants 3 and 8 did not have any component in the Mu cluster and are thus not included in the Mu cluster analysis (blank rows in the table).

predominantly by enhancing the response gain of the contrast response function.

Mu Rhythm Amplitude Does Not Affect Visual Performance

To test the specificity of the effect of prestimulus occipital alpha oscillations on visual performance, we applied the same analyses described above to prestimulus activity in the mu cluster. Figure 5B shows detection performance associated with each prestimulus mu amplitude bin at each stimulus intensity. Figure 6B shows that no modulation of threshold or slope was observed in any participant, and inconsistent effects of mu amplitude on maximum performance occurred (three participants with positive and three participants with negative effects). We fitted

the four models presented above to performance split according to mu amplitude and found that the null model best accounted for the data pooled across participants (Figure 8). Akaike weights corresponding to this comparison are shown in Table 2. The results at the individual participant level were more nuanced. The response gain model was best and significant in 4 of the 11 participants analyzed. One was best fitted by the response gain model but showed an effect in the opposite direction, five other participants were best fitted by the null model and one other by the contrast gain model (although in the unexpected direction, threshold *C50* was lowest with high levels of alpha in this participant). Together, these analyses support the hypothesis that prestimulus occipital alpha amplitude influences behavior predominantly by enhancing the response gain of the contrast response function.

We evaluated whether mu amplitude had an inverted U-shaped effect on performance by using the same nested regression model analysis as for posterior alpha components. The linear model best accounted for the effects of mu amplitude on performance (all $F(1, 2) < 9.91$; $p > .08$). Thus, mu amplitude did not have a U-shaped effect on visual performance.

The modeling analysis was also performed on trials separated by amplitude at the channel level. With trials separated by amplitude on alpha channels (PO3, PO4, and POz), Akaike weights favored the response gain model with 98%. For trials separated by amplitude on mu channels (C3 and C4), Akaike weights favored the contrast model with 42% probability, against 33% for the null model, 22% for the response model, and 3% for the full model.

Effects of Lateralization of Alpha Amplitude on Detection of Ipsi- and Contralateral Stimuli

Prestimulus alpha amplitude lateralization did not affect performance. We show these results in Table 3 (top part). At the individual level, the data were best explained either by the Null, or Contrast, or Response gain models; only one participant showed a significant effect in the expected direction. At the group level, the response gain model was best explaining the data but the parameter estimate did

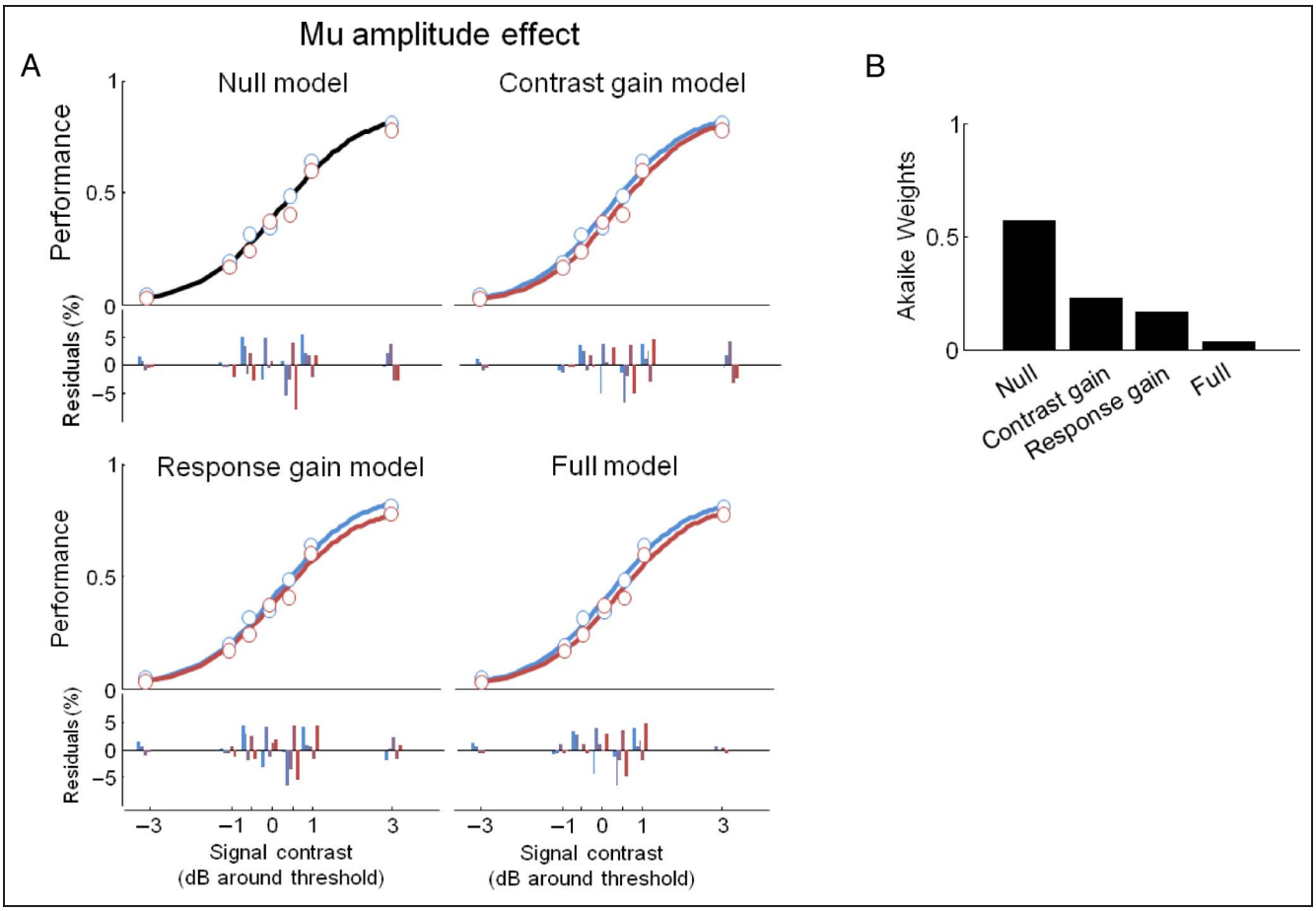

Figure 8. Comparison of four models of the effect of mu amplitude on detection performance. (A) The four panels show the maximum likelihood estimate of performance variations according to the model mentioned above each panel. At each of the seven levels of signal contrast, the two markers show average performance for the highest (magenta) and lowest (blue) prestimulus mu amplitude quintile (other quintiles are not shown for clarity). The bar plot under each panel shows the residuals of the fit (the difference between the estimated and actual performance at each contrast level and for each mu amplitude quintile, including those not shown above). (B) Akaike weights for the tested model set on the data pooled across participants.

Table 3. Comparison of the Four Models for Lateralization and Phase Effects on Performance

	Akaike Weights for Each Model					*Highest Ipsilateral Bin*			*Highest Contralateral Bin*			
Participant	*Null*	*Contrast Gain*	*Response Gain*	*Full*	*Best Model*	*−SE*	*Mean*	*+SE*	*−SE*	*Mean*	*+SE*	*Significance*
Ipsilateral–Contralateral												
1	0.46	0.48	0.03	0.04	Contrast	0.91	1.26	1.62	0.23	0.52	0.81	−*
2	0.00	0.95	0.00	0.05	Contrast	1.49	2.17	2.85	0.10	0.59	1.08	−*
3	0.96	0.01	0.02	0.00	Null							
4	0.16	0.26	0.57	0.02	Response	0.81	0.94	1.07	0.64	0.77	0.90	
5	0.02	0.68	0.26	0.04	Contrast	0.49	0.66	0.84	0.12	0.57	1.01	
6	0.46	0.50	0.01	0.03	Contrast	0.46	0.68	0.91	0.51	0.82	1.13	
7	0.93	0.03	0.04	0.00	Null							
8	0.35	0.03	0.60	0.01	Response	0.74	0.85	0.95	0.85	0.95	1.06	
9	0.02	0.72	0.15	0.11	Contrast	0.23	0.38	0.52	1.40	1.60	1.80	*
10												
11	0.00	0.99	0.00	0.01	Contrast	0.41	0.55	0.70	0.65	0.87	1.08	
All	0.00	0.01	0.93	0.06	Response	0.92	0.96	1.00	0.88	0.92	0.96	

	Akaike Weights for Each Model					*Best Performance Phase Bin*			*Opposite Phase Bin*			
Participant	*Null*	*Contrast Gain*	*Response Gain*	*Full*	*Best Model*	*−SE*	*Mean*	*+SE*	*−SE*	*Mean*	*+SE*	*Significance*
Phase Effect												
1	0.96	0.04	0.01	0.00	Null							
2	0.99	0.00	0.00	0.00	Null							
3	0.79	0.20	0.01	0.00	Null							
4	0.94	0.06	0.00	0.00	Null							
5	0.95	0.02	0.03	0.00	Null							
6	0.81	0.17	0.01	0.00	Null							
7	0.90	0.01	0.09	0.00	Null							
8	0.41	0.08	0.01	0.50	Full[a]	0.39/0.78	1.42/0.98	2.44/1.18	2.72/0.60	2.89/0.76	3.06/0.91	
9	0.96	0.03	0.01	0.00	Null							
10	0.90	0.01	0.09	0.00	Null							
11	0.97	0.01	0.03	0.00	Null							
All	0.02	0.97	0.00	0.01	Contrast	0.99	1.04	1.08	0.96	1.00	1.04	

The top and bottom tables show the results of the model fits to performance split according to prestimulus lateralization (ipsilateral–contralateral prestimulus alpha amplitude) and phase, respectively. Shaded cells show the best model for each participant. In each table, the last row shows the modeling results on average performance of the entire sample. The right part of the table shows estimates and confidence intervals for the lowest and highest amplitude trial bins for the free parameter of the best model (note that there is no free parameter in the null model, hence rows where the Null model was the best one are empty). Nonoverlapping confidence intervals are marked in the last column (* indicates change in the hypothesized direction, −* indicates change in the opposite direction). [a]The full model has two parameters ($C50$ and P_{max}) presented in the confidence interval columns as "$C50/P_{max}$." Participant 10 did not have components in both the ipsi- and contralateral clusters and thus could not be included in the lateralization analysis (blank row in the table).

not differ significantly between trials with highest alpha activity in ipsilateral clusters (expected high performance) and those with highest activity at contralateral clusters (expected low performance).

Alpha Phase Effects on Visual Performance

The phase of the ongoing oscillations at stimulus onset had no consistent effect on performance for single participants. For the tested time–frequency window, the null model best explained the data in all participants but one, as shown in the bottom part of Table 3. Akaike weights in favor of the null model for individual participants were on average above 0.90 ± 0.03 (*SEM*). When phase data were aggregated across participants, Akaike weights favored the contrast gain model (97%). However, when comparing the PMFs between trials with optimal versus opposite phase bin, we found that the confidence intervals of the *C50*

parameter were overlapping, indicating that the effect of alpha phase on contrast gain was less robust than the effect of alpha amplitude on response gain.

DISCUSSION

In this study, we investigated the functional mechanism by which spontaneous alpha oscillations impair visual perception. Specifically, we show that prestimulus alpha oscillations can affect visual detection performance via a divisive response gain mechanism. The effect of alpha oscillations was thus greatest on stimuli with highest intensity. We also show that this effect is specific to oscillations originating from the occipital cortex, because oscillations at the same frequency but originating from more anterior, possibly somatosensory cortices (i.e., the mu rhythm), did not affect performance reliably. These results impose constraints on the processing stages potentially affected by alpha oscillations and thus have implications for our understanding of the functional role of alpha oscillations in brain processing.

Mu components, originating from more anterior sources than the alpha components, did not affect performance reliably. This is consistent with other studies showing that alpha oscillations primarily affect brain processing locally and in a modality-specific manner (Capilla, Schoffelen, Paterson, Thut, & Gross, 2012; Sauseng, Klimesch, Gerloff, & Hummel, 2009). Similarly, in a visual detection task, Van Dijk and coworkers (2008) showed no effect of mu activity on visual performance. Understanding interactions between alpha activity in different sensory cortices and stimulation in different sensory modalities will be an important goal for future research.

Several previous psychophysical investigations of the role of alpha oscillations over the somatosensory cortex reported an inverted U-shaped pattern of effects on detection of somatosensory stimuli (Zhang & Ding, 2010; Linkenkaer-Hansen, Nikulin, Palva, Ilmoniemi, & Palva, 2004): Stimuli preceded by particularly weak or strong alpha oscillations were difficult to detect, whereas stimuli preceded by intermediary alpha amplitudes were associated with improved performance. Interestingly, Linkenkaer-Hansen and coworkers (2004) found that the inverted U-shaped effect of alpha oscillations originating from somatosensory cortex was accompanied by a linear effect of alpha oscillations over the parietal cortex. Using visual stimuli, Rajagovindan and Ding (2011) found an inverted U-shaped effect of occipital alpha oscillations on the amplitude of evoked potentials but did not report any effects on behavioral performance. In this study, performance was clearly monotonically decreasing with increasing occipital alpha power, consistent with previous studies (Van Dijk et al., 2008), whereas frontocentral mu rhythms showed neither monotonic nor inverted U-shaped effects. Thus, it is likely that the inverted U-shaped relationship between amplitude of occipital prestimulus oscillations and behavioral performance is specific to frontocentral mu rhythms in somatosensory detection tasks.

Previous studies reported that not only the amplitude but also the phase of the ongoing alpha oscillations affect performance and perception (e.g., Dugué, Marque, & VanRullen, 2011; Busch & VanRullen, 2010; Busch et al., 2009; Mathewson, Gratton, Fabiani, Beck, & Ro, 2009). In this experiment, we found no significant effect of alpha phase at stimulus onset on detection performance. However, although the modeling analysis indicated that a contrast gain model explained the data better than any of the other models, the contrast thresholds computed for trials with optimal versus opposite phase bin were too overlapping to be considered significantly different. This lack of a robust effect here should be considered with care, because the analysis might be underpowered because of a too small number of trials, which prevented us from doing a more fine-grained analysis of phase information (e.g., by increasing the number of phase bins).

We tested whether the relative amount of spontaneous alpha activity occurring on the ipsi- versus contralateral side relative to the upcoming stimulus affected performance. Indeed, previous studies reported that attention-induced ipsi- minus contralateral alpha activity affects performance (Busch & VanRullen, 2010; Kelly et al., 2006, 2009; Worden et al., 2000). We thus reasoned that spontaneous alpha activity (although not triggered by an attention shift) could reveal a similar pattern. No such effect occurred in this study.

Our study significantly extends previous studies on the effects of prestimulus oscillations on performance. Indeed, most previous studies of the effects of alpha oscillations on perception did not attempt to formally model the underlying suppression mechanism (although see Van Ede, Köster, & Maris, 2012; Van Ede, de Lange, & Maris, 2012; Wyart & Tallon-Baudry, 2009, for other modeling studies) and could thus only speculate as to the functional mechanism underlying the effect. At large, two mutually nonexclusive theoretical interpretations of the functional effects of alpha oscillations have been proposed. The first one postulates that alpha oscillations inhibit processing in cortical regions whose activity is unnecessary or even deleterious to complete the task at hand (Jensen & Mazaheri, 2010; Klimesch et al., 2007). This inhibition of processing is often interpreted in terms of a decrease in excitability of the underlying cortex. Elegant experimental manipulations allowed researchers to suggest that alpha oscillations inhibit the input to the visual system. For example, Lange and coworkers (2013) used stimuli inducing illusory percepts to suggest that states of weak alpha oscillations "might render visual cortex in general more susceptible to input." Similarly, Romei and coworkers (2010) used TMS in several experiments and suggested that "alpha regulates the flow of incoming information," consistent with an early interpretation that alpha oscillations may provide "a gating mechanism for incoming sensory information on its way to the cortex"

(Ergenoglu et al., 2004). The second line of interpretation emphasizes the potential role of alpha oscillations in promoting top–down processing and long distance information transfer (Saalmann, Pinsk, Wang, Li, & Kastner, 2012; Palva & Palva, 2007; Von Stein & Sarnthein, 2000). In this view, the inhibitory effect of alpha oscillations could also be interpreted as reduced communication of the visual system with other areas because of increased top–down demands. Thus, for instance, Van Dijk and coworkers (2008) suggested that alpha oscillations might serve to "gate the information passed from occipital to dorsal parietal areas."

It is important to note that functional mechanisms such as gain effects are theoretical concepts that cannot be directly observed in empirical data such as hit rates or microvolts. Instead, finding and quantifying such mechanisms requires a formal model that describes the relationship between mechanisms and observable data. In this study, we used a formal description of the inhibitory effect of prestimulus oscillations by modeling these effects as free parameters of the PMF. This approach allowed us to measure which of the two interpretations described above best fits human detection performance. Specifically, modulatory effects such as the inhibitory effect of prestimulus occipital alpha oscillations can act on contrast gain or response gain, which are closely related to previous interpretations of the inhibitory effect of prestimulus alpha oscillations. A reduction of contrast gain would be indicated by a horizontal, rightward shift of the PMF (see Figure 1B, left), reflecting an increase in threshold. This effect would be equivalent to a reduction of the effective input to the visual system, corresponding to the previously hypothesized effect of alpha oscillations on visual input (see Lange et al., 2013; Romei et al., 2008; Ergenoglu et al., 2004). Alternatively, response gain is indicated by a reduction of performance that is proportional to the output of the system (Figure 1B, right). Thus, reduced response gain does not impair performance by a change in the visual system's threshold or sensitivity. Rather, reduced response gain marks a reduction in the visual system's input/output transformation or how much a given increase in stimulus intensity leads to an improvement in performance—and thus in the impact of the responses computed in the visual system on higher-level areas. In this study, we found that a response gain model best fitted the data in the majority of participants, thus showing that the inhibitory effect of alpha oscillations on performance is best explained by a change at the level of the output of the model.

This finding is thus consistent with the proposal that alpha oscillations act on the functional communication between the cortical areas (Saalmann et al., 2012; Palva & Palva, 2007; Von Stein & Sarnthein, 2000), during or after the transformation of a continuous representation of sensory stimulus into a binary "seen"/"not seen" decision. This suggests that the effect of alpha oscillations occurs, at a postperceptual, possibly decisional stage. However, we cannot strictly dissociate a perceptual from a decisional effect here. This would require measuring sensitivity and decision criterion of the observers, as defined in signal detection theory (Green & Swets, 1966). Indeed, our measure of performance conflates both quantities. A sensitivity account would argue that alpha amplitude variations would affect the observers' ability to discriminate the very weak stimuli we were using from noise, whereas a decision criterion account would argue that alpha oscillations affect the threshold level of signal at which they decide whether a stimulus is present or not. Estimating decision bias requires no-stimulus trials to calculate participants' false alarm rate. The response rate to signal presented 3 dB under detection threshold (the weakest presented stimuli, which were virtually invisible) could arguably be considered an upper bound to the false alarm rate in this experiment. Notably, participants responded on average only to 4 of 160 of these weak stimuli. The potential impact of these false alarms was thus at most weak in the final performance measure and is unlikely to have generated the massive response gain effects we observed. Future experiments will address this issue explicitly by inducing higher false alarm rates and measuring their rate at various levels of alpha oscillations. Of interest to this issue, one previous study used a modeling approach in a signal detection task and found that alpha oscillations did not bias decisions (Wyart & Tallon-Baudry, 2009). They found that a model in which alpha oscillations explicitly affected decision bias was not better fitting the data than one in which no such bias could occur.

How do these findings relate to what has been observed with other modulatory effects on performance? In particular, as mentioned in the introduction, attention and adaptation both affect the PMF and their effects have been modeled in terms of contrast and response gain modulation. Visual adaptation refers to a change in perceptual and neuronal sensitivity after exposure to a static and unchanging stimulus. Single-cell recording studies in the visual system of animals, as well as in human neuroimaging and psychophysical studies, have consistently shown that adaptation acts via a contrast gain mechanism (see review by Kohn, 2007). It thus seems unlikely that the effect of prestimulus alpha oscillations on performance is mediated by adaptation.

The results of modeling attentional modulations of psychophysical performance, on the other hand, are more varied. Numerous studies (see Carrasco, 2011, for a comprehensive review) have demonstrated that attention can affect neuronal responses and psychophysical performance by contrast gain (Ling & Carrasco, 2006; Martínez-Trujillo & Treue, 2002; McAdams & Maunsell, 1999) or response gain (Reynolds et al., 2000; McAdams & Maunsell, 1999) or by a combination of both (Herrmann, Heeger, & Carrasco, 2012; Herrmann, Montaser-Kouhsari, Carrasco, & Heeger, 2010; Ling & Carrasco, 2006). Recently, a powerful and simple model has been proposed to reconcile these observations across levels of observation (Reynolds & Heeger, 2009). It accounts for response and contrast gain

effects as the simple product of two opposing driving forces during signal processing. The first one, called stimulus drive, influences performance via a positive multiplicative gain proportional to the contrast of the stimulus, whereas the second one, called suppressive drive or normalization, pools sensory information and acts in the opposite direction. This breakdown of the stimulus into a multiplicative and a divisive factor allows this model to reconcile a wide range of results (Reynolds & Heeger, 2009), including single cell electrophysiology and behavioral psychophysics. Recently, it has been used in psychophysical experiments to account for the effect of spatial attention on performance (Herrmann et al., 2010, 2012). Noteworthy, the present results are consistent with the idea that alpha oscillations could intervene in the normalization process. Higher amplitudes of alpha could for instance augment the amount of suppressive drive to the signal, adding to the denominator of the contrast response function. The result of this augmentation would resemble a response gain effect. Further experiments will be required to fully test this hypothesis.

In conclusion, we have used a straightforward modeling approach to disentangle two potential mechanisms by which alpha oscillations can affect visual perception. We show that ongoing alpha oscillations can affect performance via modulation of response gain, suggesting that, rather than affecting the sensitivity of the sensory system to visual information, these oscillations affect postperceptual, potentially decisional processing stages, in line with a role of these oscillations in promoting interareal communication.

Acknowledgments

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