Beyond establishing involvement: quantifying the contribution of anticipatory α- and β-band suppression to perceptual improvement with attention

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van Ede F, Köster M, Maris E. Beyond establishing involvement: quantifying the contribution of anticipatory α- and β-band suppression to perceptual improvement with attention. *J Neurophysiol* 108: 2352–2362, 2012. First published August 15, 2012; doi:10.1152/jn.00347.2012.—Systems and cognitive neuroscience aim at understanding the neurophysiological mechanisms that underlie cognition and behavior. Many studies have revealed the involvement of many types of neural signals in diverse cognitive and behavioral phenomena. Here, we go beyond establishing such involvement and address two fundamental, yet largely unaddressed, questions: *1*) exactly how much does a given neural signal contribute to a cognitive or behavioral phenomenon of interest; and *2*) to what extent are distinct neural signals independently related to this phenomenon? We recorded brain activity using magnetoencephalography while human participants performed a cued somatosensory detection task. Using a novel method, we then quantified the contribution (in a predictive but not causal sense) of two well-established neural phenomena to the improvement in perception with attentional orienting. In our sample, the anticipatory suppression of extracranially recorded oscillatory α- and β-band amplitudes from contralateral primary somatosensory cortex could account for maximally 29% of the attention-induced improvement in tactile perception. In addition, although amplitude suppressions in the α- and β-frequency bands both contributed to this improvement, their contribution was largely shared. These data reveal the upper limit of the cognitive/behavioral relevance of this type of signal and show that at least 71% of the perceptual improvement with attention must be accounted for by other signals.

attentional orienting; behavioral relevance; magnetoencephalography; neuronal oscillations; somatosensory perception

SYSTEMS AND COGNITIVE NEUROSCIENCE aim at understanding the neurophysiological mechanisms that underlie cognition and behavior. To date, this has resulted in a wealth of knowledge concerning the involvement of particular neural signals in cognitive functions and behavior. Despite this progress, studies up to now have left two fundamental questions largely unaddressed. First, how much does a given neural signal contribute to a cognitive or behavioral phenomenon of interest? Second, to what extent are distinct neural signals independently related to this phenomenon?

We report on a study in which both of these questions were addressed. In particular, we quantified the contribution of well-established neural phenomena (anticipatory suppression of oscillatory amplitude in sensory cortex occurring in multiple frequency bands) to a well-established behavioral phenomenon: the improvement in perception that occurs with attentional orienting.

Knowing when and where a stimulus will occur allows for orienting of attention and improves perception (Posner 1980). Such attentional orienting involves a prestimulus modulation of oscillatory electromagnetic signals with their amplitude being lower over relevant compared with irrelevant sensory cortex. This has been established for posterior α-band (8–14 Hz) oscillations during visual (Foxe et al. 1998; Gould et al. 2011; Siegel et al. 2008; Snyder and Foxe 2010; Thut et al. 2006; Worden et al. 2000; Wyart and Tallon-Baudry 2008) and somatosensory α- and β-band (15–30 Hz) oscillations during tactile (Anderson and Ding 2011; Haegens et al. 2011a; Jones et al. 2010; van Ede et al. 2010, 2011) orienting of attention. These modulations are specific to the timing (Rohenkohl and Nobre 2011; van Ede et al. 2011) and features (Snyder and Foxe 2010) of an expected stimulus, and their deployment depends on stimulus probability (Gould et al. 2011; Haegens et al. 2011a). These properties suggest that oscillatory amplitude modulation reflects an important mechanism underlying the orienting of attention. This is further supported by the observation that low amplitude in the α- and β-band is associated with an enhancement in *1*) cortical excitability (Haegens et al. 2011b; Romei et al. 2008; Sauseng et al. 2009), *2*) blood oxygenation level-dependent (BOLD) activity (Ritter et al. 2009; Scheeringa et al. 2011), and *3*) psychophysical performance in visual (Thut et al. 2006; van Dijk et al. 2008) and tactile (Haegens et al. 2011a; Jones et al. 2010; van Ede et al. 2011) tasks.

Despite this coherent picture, two fundamental aspects remain unclear. First, exactly how much of the improvement in perception with attentional orienting can be accounted for by these anticipatory amplitude modulations? Although we acknowledge that the relation between prestimulus amplitude and perception has previously been investigated with quantitative measures (e.g., correlation coefficients), inferences have so far remained qualitative. Specifically, it was assessed whether a correlation of interest was statistically significant, allowing the qualitative inference of whether a relation existed between the two variables ("establishing involvement"). As we will outline in our DISCUSSION, based on these conventional correlational analyses, only this type of qualitative inference is justified. In the present paper, our goal is not qualitative but quantitative inference. In other words, our goal is determining the effect size. A second fundamental aspect that has remained unclear is to what extent modulations in the α- and the β-band independently contribute to the improvement in perception that occurs with attentional orienting. To address these two questions, we developed a novel method that allows for a quantification of

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the cognitive/behavioral relevance (in a predictive but not causal sense) of a particular set of neural signals. In contrast to conventional quantitative measures (in casu the correlation coefficient), our method produces a quantification that is affected by neither noise in our predictor variables (i.e., our amplitude estimates) nor variability in our criterion variable (detection responses) that is independent of the cognitive variable under investigation (attentional orienting; see DISCUSSION). We applied our method to extracranial signals that were acquired during a somatosensory detection task using magnetoencephalography (MEG). In our sample, the anticipatory suppression of the rolandic α- and β-band oscillatory amplitudes together accounted for maximally 29% of the improvement in tactile perception with attentional orienting. Moreover, although amplitude suppressions in both frequency bands contributed to this improvement, their contributions were largely shared.

MATERIALS AND METHODS

Participants and Experimental Design

Our study was conducted in accordance with the Declaration of Helsinki and approved by the local ethics committee (CMO Regio Arnhem-Nijmegen). Informed written consent was obtained from all subjects.

Fourteen healthy subjects (5 male; 22–49 yr) participated in the study. Two subjects were excluded from analysis: for one subject, no stable behavioral performance could be obtained, and the other one fell asleep.

Subjects performed a somatosensory detection task in which the spatial and temporal locations of target stimuli was either cued or not (Fig. 1). The central event in a trial was the occurrence of a brief auditory stimulus (50 ms, white noise) that was paired with an electrotactile stimulus (0.5-ms electric pulse close to threshold intensity) in half of the trials. This tactile stimulus was delivered using either of two constant-current, high-voltage stimulators (type DS7A; Digitimer) to the left or right thumb. Stimulus intensity was set before the experiment. For this, we used a Bayesian staircase algorithm (QUEST; Watson and Pelli 1983) that adjusts the stimulus intensity such that the hit rate in the cued condition (see below) was ~80% (see also Fig. 2A). This algorithm was implemented in the MATLAB (The MathWorks) Psychophysics Toolbox (Brainard 1997). Intensities were 1.400 ± 0.066 and 1.747 ± 0.077 mA for the left and the right hand, respectively.

In the experiment, subjects indicated whether a tactile stimulus was presented at the time of the central event, which was marked by a brief auditory stimulus. In the uncued condition, it was made as difficult as possible to predict the time of the central event as well as the location at which the tactile stimulus might occur. Concerning time, we drew the interval between a response and the next central event from a truncated negative exponential distribution (mean 3.5 s; limited to the range 2.5–12 s). A negative exponential distribution has a hazard rate (the probability of an event at time t given that it has not occurred yet) that does not depend on this time t and therefore is unpredictable. Concerning location, the tactile stimulus could, in case of a stimulus-present trial, occur at either thumb. In the cued condition, the time of the central event and the location at which the tactile stimulus might occur were fully predictable: 1.5 s before the central event, an auditory cue (150 ms, pure tone) was presented, informing with 100% validity about the time (after 1.5 s) and location (left/right thumb) at which the stimulus might occur. Cue pitch (500/1,000 Hz) informed location and was counterbalanced across participants. Cued and uncued trials were randomly interleaved. In total, one-third of the trials were cued.

Participants indicated whether a stimulus was presented by pressing a button with the left or the right index finger. To indicate the presence of a tactile stimulus, subjects pressed a button on the side where a lateralized auditory response-mapping tone (150 ms, 1,000 Hz) was presented, and to indicate its absence, they pressed the button on the other side. This mapping of the perceptual decision onto the response was varied from trial to trial to prevent specific motor preparation in the anticipation period of interest. Auditory feedback was presented, indicating hits and misses (50 ms upgoing, respectively, downgoing frequency sweeps between 500 and 1,000 Hz).

Participants completed ~1,000 trials in 2 consecutive MEG sessions lasting ~70 min each. Within each block (72 trials), cue presence, cue side, stimulus presence, and stimulus side were counterbalanced. After each session, a primary somatosensory cortex (S1) localizer experiment was performed: 200 suprathreshold stimuli were delivered to each thumb. The day before the experiment, subjects practiced the task for approximately 1 h.

Data Acquisition and Preprocessing

The MEG system (CTF MEG; MISL, Coquitlam, British Columbia, Canada) contained 275 axial gradiometers and was housed in a magnetically shielded room. We also recorded bipolar surface electromyogram (EMG) from the flexors of the forearm (cf. van Ede et al. 2011). Localization coils, fixed to anatomic landmarks (nasion and left and right ear), determined head position. All data were low-pass filtered (300-Hz cutoff), digitized at 1,200 Hz, and stored for offline analysis. Line noise was removed offline using a discrete Fourier transform filter. Epochs contaminated by artifacts were removed based on visual inspection. T1-weighted MR images were acquired, and subject-specific, single-shell models (Nolte 2003) for source reconstruction were calculated.

Data Analysis

Data were analyzed using FieldTrip (Oostenveld et al. 2011), an open-source MATLAB toolbox developed at the Donders Institute for Brain, Cognition and Behaviour (Nijmegen, The Netherlands).

Frequency analysis. We calculated oscillatory amplitude by means of the Fourier transform with and without time resolution. For calculations with time resolution (Fig. 2C), we used a 500-ms sliding time
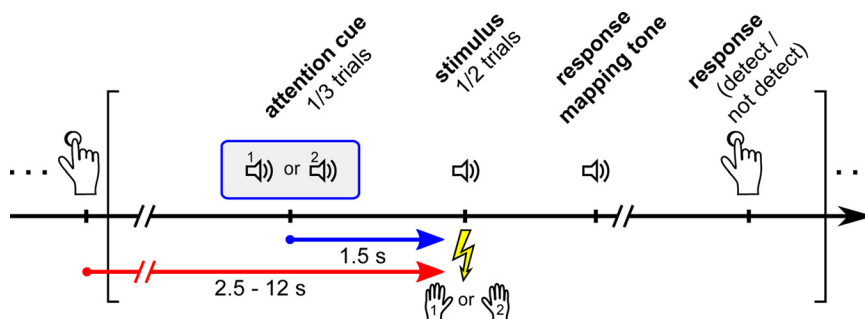

Fig. 1. Experimental paradigm. Subjects performed a somatosensory detection task in which time (after 1.5 s) and location (left/right thumb) of the upcoming event was either cued or uncued. This event consisted of a brief tone that, in half of the trials, was paired with an electric stimulus on 1 of the subject's thumbs. In each trial, subjects had to indicate whether they had felt a stimulus. Responses were delayed, and the mapping of the perceptual decisions onto the response buttons was indicated by a response mapping tone (see MATERIALS AND METHODS). Uncued and cued trials were randomly interleaved.

window (advanced in steps of 50 ms) and a Hanning taper. Nontime-resolved amplitude estimates (Figs. 3–5) were calculated for the α- and the β-band using the multitaper method (Percival and Walden 1993). The α-band was defined as 8–14 Hz for all subjects, whereas the β-band (which is more variable in frequency) was individually determined per subject based on the poststimulus response (cf. van Ede et al. 2010, 2011). On average, β-bands ranged from 16 to 28 Hz.

Source reconstruction. We reconstructed oscillatory brain activity originating from putative S1. This involved three steps. First, we used the localizer data to find, per participant, the left and right sources that showed the strongest response to right and left tactile stimuli, respectively. This was done in source space using beamforming (dynamic imaging of coherent sources; Gross et al. 2001). Beamforming reconstructs source power using spatial filters that have unit gain for the point source of interest while maximally attenuating other sources. This is accomplished by taking the cross-spectral density matrix into account. Per voxel (0.5-cm resolution), we calculated the difference between the β-band amplitudes recorded after left and right thumb stimulation. The maximum and minimum voxels in this contrasted volume were used as loci for left and right sources of interest (S1; cf. Haegens et al. 2011a; van Ede et al. 2011). We then estimated optimal spatial filters for reconstructing activity from left and right S1, again using beamforming (now using the cross-spectral density for the 5- to 50-Hz band, calculated across all epochs of interest). Finally, we applied the obtained filters to our time-domain data (275 channels) to reduce it to 1 left and 1 right virtual S1 channel.

Quantifying the contribution of amplitude to behavior. For every trial, we calculated four prestimulus amplitude estimates: α- and β-amplitude in left and right S1. To allow pooling of data over recording sessions and hemispheres, we normalized all amplitude estimates by linearly transforming them to the percentage change from the mean amplitude in the uncued condition of the respective session and hemisphere. Amplitudes from left and right S1 were reassigned as contralateral or ipsilateral to the stimulus.

Our quantification procedure, which was performed per subject, involved the following steps. First, we modeled the relation between perceptual performance and prestimulus amplitude using linear (for reaction times) and logistic regression (for hit rate) in the uncued trials (Fig. 3, *A–C, middle*). Second, we quantified the anticipatory amplitude modulation in the cued condition. This modulation is the difference between the mean amplitudes in the cued and the uncued trials (red and blue vertical lines in Fig. 3, *A* and *B, bottom*). Third, we used the amplitude-perception regression coefficients from *step 1* to predict the improvement in perceptual performance that would follow from the anticipatory modulation from *step 2*. Finally, we compared this prediction with the actual improvement that occurred with cueing. By dividing the predicted improvement with the observed improvement (and multiplying it by 100), we quantified how much of the improvement in perception with attentional orienting can be accounted for by anticipatory amplitude modulation: the percentage explained improvement.

Because the regression lines of the cued and uncued condition were roughly linear and parallel over the range in which our amplitudes occurred (Fig. 3), our quantification could be based on the regression line of only the uncued condition together with the average attention-induced change in amplitude and perception in the cued condition. If the regression lines would not have been linear and parallel, one would have to use a calculation that is more generally applicable (i.e., to nonlinear and nonparallel regression lines). This more general calculation comes from the statistical literature on the contribution of covariates (to correct for them) when estimating treatment effects in observational studies (Maris 1998; Rubin 1974, 1977). The formal theory behind this literature also applies to other estimation problems, such as ours, and involves estimating the extent to which the difference between groups (cued and the uncued trials) on some dependent variable (detection performance) can be explained by a another variable (oscillatory amplitude).

Our logistic regression modeled the probability of a hit as a function of prestimulus amplitude according to:

$$P(\text{hit}) = \frac{\exp(\beta 0 + \beta 1 \times \text{amplitude})}{1 + \exp(\beta 0 + \beta 1 \times \text{amplitude})}$$

We also performed logistic regression using two predictor variables, the single-trial α- and β-band amplitudes. For this, we added a $\beta 2 \times$ amplitude term in the exponent. Coefficients were estimated using the maximum likelihood criterion.

Correcting for noisy-amplitude estimates. Noise in our amplitude estimates will attenuate the slopes of the amplitude-perception regression lines that form the basis of our quantification. To obtain a veridical (i.e., noise-free) quantification, the slope-attenuating effect of this noise must be corrected for. We did this in two ways.

First, we corrected single-trial amplitudes for variability in single-trial head position (which was derived from our localization coils) by *1*) calculating head position as the Euclidian distance between the center of the head and the center of the MEG helmet, *2*) modeling the relation between head position and amplitude using linear regression, and *3*) using these regression coefficients (per subject, session, and frequency band) to correct single-trial amplitudes for variability in single-trial head position. We used this metric of height in the helmet because we expected amplitude estimates from left and right S1 (which are positioned at the top center of the helmet) to be most affected by it (this might be different for different cortical regions). Because head height accounted for only a small percentage of the variance in amplitude across trials (<5%), we decided not to perform more sophisticated head-position correction algorithms.

Second, we corrected for noise in the single-trial estimates. For this, we used a simulation approach (depicted in Fig. 5, *A–C*) with which we derived which true amplitude-perception regression slope formed the basis of our observed slope, given the observed amount of noise. First, we log-transformed our amplitude estimates such that their distribution closely approximated a normal distribution (Fig. 5, *A* and *D*). As before, we linearly transformed them as percentage change from the mean amplitude in the uncued trials. Second, we estimated the sampling variance of these log- and linearly transformed amplitude estimates. For this, we made use of the fact that the multitaper method calculates multiple independent Fourier coefficients per trial, one per taper. Per trial, we calculated the log- and linearly transformed amplitudes of these taper-specific Fourier coefficients, calculated their variance across tapers, and divided this variance by the number of tapers to estimate the trial-specific sampling variance. We then averaged this quantity across trials. It is crucial to specify under which conditions this trial-averaged sampling variance estimate is unbiased, that is, under which conditions it neither over- nor underestimates the true sampling variance. We will consider this point in detail below. In the third step, we used the estimated sampling variance to reconstruct the true log-amplitude distribution as a normal distribution with mean equal to the observed mean and variance equal to the observed across-trial variance minus the estimated sampling variance (Fig. 5A). Using this distribution together with the noise distribution (normal distribution with 0 mean and variance equal to the estimated sampling variance), by means of simulation, we derived which true amplitude-perception regression slope would, after adding noise, yield the empirically observed 1 (Fig. 5, *B* and *C*).

In our simulation method, we used a 1-dimensional grid (100 steps) of regression coefficients centered on the empirically observed coefficient. For every point in this grid, we randomly drew trial-specific amplitudes from our reconstructed true distribution and assigned every trial to be a hit or miss depending on the probability of a hit given the simulated regression coefficient and the drawn amplitude value. We then randomly drew noise from our noise distribution, added it to the amplitudes drawn from the reconstructed true distribution, performed a logistic regression, and calculated the simulated percentage explained improvement. To obtain a reliable estimate, we

repeated all the above steps for the full grid numerous times. After every repetition, we calculated, for each grid point, the running average of the simulated percentage explained improvement. We then identified the best grid point as the point for which this running average was closest to the empirically observed percentage. Absolute deviations were as small as $0.109 \pm 0.024\%$ for the single predictor model and $0.015 \pm 0.004\%$ for the dual-predictor model (discussed below). We continued until the 95% confidence interval (calculated across simulations) of this simulated percentage for the best grid point fell within $\pm 1\%$. As our estimate of the true regression slope, we took the regression coefficient belonging to the best grid point. With this estimated true regression coefficient, the true percentage explained improvement was derived. These simulations were performed for each participant separately, and outcomes were averaged across participants.

This quantification of the explained improvement is only unbiased if the signal is stationary, because only in this case our estimate of the true amplitude-perception regression slope is unbiased. This is because only under this assumption of a stationary signal can we rely on the standard theory of multitaper estimation to obtain an unbiased estimate of the single-trial noise variance from the Fourier coefficients of the multiple independent tapers (Percival and Walden 1993, p. 360, point 2; Thomson 1982, section IV). Under violation of this assumption, the noise of the signal is overestimated because part of the within-trial variance across tapers is due to the nonstationarity of the true underlying signal. This leads to an overestimation of the true amplitude-perception regression slope and thereby also the percentage explained improvement. It is important to realize that the percentage explained improvement is potentially overestimated but cannot be underestimated. For this reason, it is a conservative strategy to consider our quantification as the maximal percentage explained improvement. Because perfect stationarity is unlikely to hold, the real percentage explained improvement likely lies between the percentages that are obtained before and after noise correction.

Finally, we extended our simulations to the combined α- and β-amplitude model by drawing from bivariate normal distributions. Besides the sampling variances of the α- and the β-log amplitudes, we also calculated their sampling covariance, making use of the within-trial covariance across the tapers. Likewise, we calculated the covariance across trials. This allowed us to reconstruct noise and true bivariate distributions required for our simulation. Simulated percentage explained improvement was calculated using multiple logistic regression, and simulations were performed for a 2-dimensional grid (25×25 steps) containing coefficients for α and β. As our estimate of the true pair of regression coefficients, we took the pair of coefficients belonging to the best grid point.

Statistics

All reported statistical tests were performed across subjects by means of 1-sample or paired-samples t-tests (2-tailed, $\alpha = 0.05$). All reported measures of spread are ± 1 SE.

RESULTS

Attentional Orienting Improves Perception and Involves Suppression of Contralateral Oscillatory α- and β-Band Amplitude

As depicted in Fig. 2, *A* and *B*, orienting attention improved perception by increasing the hit rate from $52 \pm 3\%$ (mean over subjects $\pm$ SE) in the uncued to $81 \pm 2\%$ in the cued condition [$t(11) = 8.491$, $P < 0.001$] and by decreasing reaction times from 685 ± 83 ms in the uncued to 513 ± 40 ms in the cued condition [$t(11) = -3.291$, $P < 0.01$]. False alarm rates did not differ between conditions [uncued $27 \pm 3\%$; cued $24 \pm$

3%; $t(11) = 1.023$, $P = 0.328$] and were lower than hit rates for all subjects. This indicates performance well above chance.

It is well-established that attentional orienting to upcoming sensory events involves an anticipatory modulation of oscillatory amplitude within sensory cortex (Anderson and Ding 2011; Foxe et al. 1998; Gould et al. 2011; Haegens et al. 2011a; Jones et al. 2010; Rohenkohl and Nobre 2011; Siegel et al. 2008; Snyder and Foxe 2010; Thut et al. 2006; van Ede et al. 2010, 2011; Worden et al. 2000; Wyart and Tallon-Baudry 2008). To verify this in our data, we computed time- and frequency-resolved oscillatory amplitudes for the reconstructed source signals originating from left and right S1 (see MATERIALS AND METHODS). Figure 2*C* shows the time-frequency representation of oscillatory amplitude in S1 following a cue to the contralateral side, expressed as the percentage change from the uncued condition. Orienting attention to an upcoming tactile event involved an attenuation of oscillatory amplitude in both the α- and the β-band [contralateral α: $t(11) = -5.523$, $P < 0.001$; contralateral β: $t(11) = -7.215$, $P < 0.001$]. Figure 2*D* shows source reconstructions of this anticipatory modulation, separately for anticipation of a left and a right thumb stimulus. Consistent with previous findings (van Ede et al. 2011), the anticipatory suppression occurs predominantly contralateral to the cued hand and includes S1.

We now asked how much of the perceptual improvement can be accounted for by these neural signals.

Quantifying the Contribution of Anticipatory Amplitude Suppression to Perceptual Improvement with Attention

Spontaneous fluctuations in oscillatory amplitude are correlated with perceptual performance (Jones et al. 2010; Linkenkaer-Hansen et al. 2004; van Dijk et al. 2008). We capitalized on this relation to quantify the contribution of anticipatory amplitude suppression to attentional improvement.

Figure 3 illustrates the rationale of our quantification (see also MATERIALS AND METHODS). First, we used data from the uncued condition to estimate the relation between prestimulus amplitude and perceptual performance in the absence of attentional orienting (red data points). For visualization of this relation, we sorted trials by amplitude and grouped them into 8 nonoverlapping bins (cf. Jones et al. 2010; Linkenkaer-Hansen et al. 2004). As can be seen at the *top* of Fig. 3, there is a close-to-monotone relation between prestimulus amplitude and perceptual performance, with lower prestimulus amplitude predicting higher perceptual performance. As part of our quantification, we modeled this relation at the level of the single-trial data. As adequate models for these empirically observed close-to-monotone relations, we used logistic regression for detection responses (hit/miss; a dichotomous variable) and linear regression for reaction times (a continuous variable; Fig. 3, *middle*). These models will be denoted as amplitude-perception regression lines. Our rationale is as follows: if perception were fully determined by α- and β-band amplitudes, then attention-induced changes in perception should be fully accounted for by attention-induced changes in these variables. In this case, the amplitude-perception regression lines should overlap between the cued and the uncued conditions. Alternatively, the degree to which these regression lines do not overlap (as quantified by the vertical distance between them) indicates the degree to
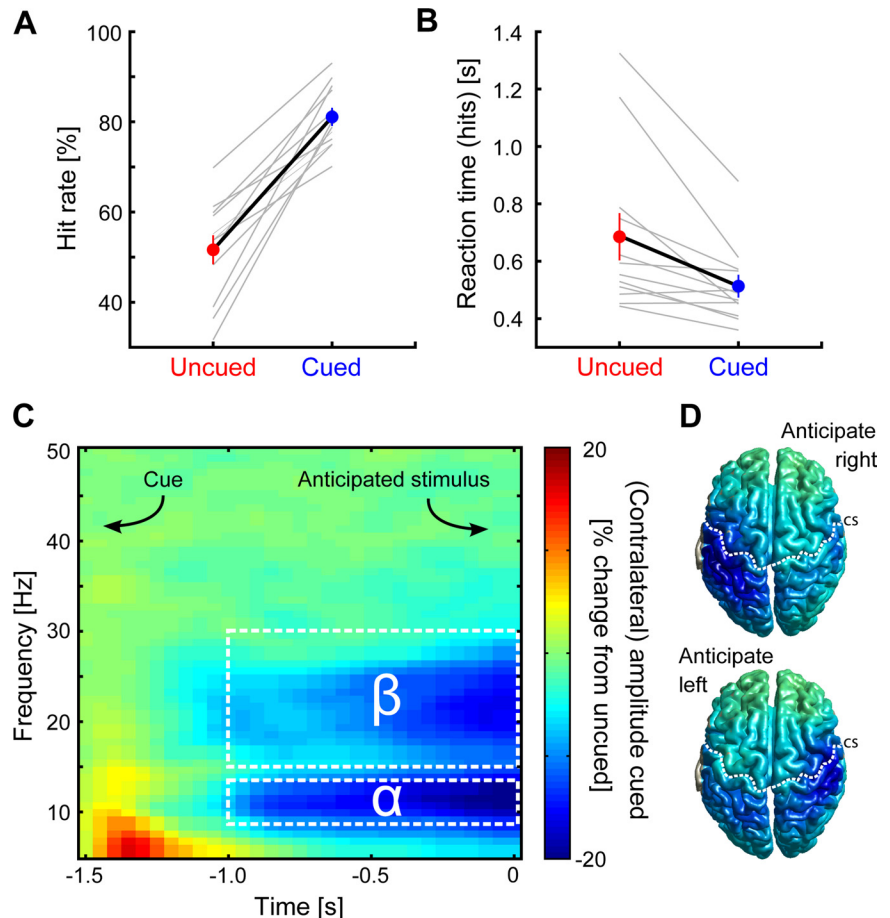
Fig. 2. Attentional orienting improves perception and involves anticipatory suppression of contralateral α- and β-oscillatory amplitudes. *A*: hit rate in the uncued and the cued condition. Colored data points indicate means ± SE. Gray lines show individual subjects. *B*: same as *A* but now showing reaction times in hit trials. *C*: time-frequency representation of oscillatory amplitude in S1 following a cue to the contralateral side expressed as the percentage change from the uncued condition. White boxes indicate the time-frequency window used in subsequent analyses. *D*: source-reconstructed amplitude modulation in anticipation of a right and left thumb tactile stimulus averaged over the α- and β-band in a 1-s prestimulus window. Dashed white lines indicate the central sulci (cs).

which attention-induced changes in perception cannot be explained by these variables.

Before describing the steps in our quantification, note that the relations between amplitude and perception are highly similar for the uncued (red data points) and the cued (blue data points) conditions. This shows that the relation between amplitude and perception is itself not altered by attentional cueing. Moreover, for the ranges in which our amplitudes occurred, the cued and uncued regression lines were roughly linear and parallel. Because of this, our quantification could be based on the regression line of only the uncued condition together with the average attention-induced change in amplitude and perception in the cued condition (see MATERIALS AND METHODS).

Our quantification involved three steps (numbered 1–3 in Fig. 3). First, we quantified the anticipatory amplitude suppression as the difference between the mean amplitudes in the cued and uncued condition (blue and red vertical lines in Fig. 3, *A* and *B*, *bottom*). Second, we used the amplitude-perception regression line from the uncued condition to predict the improvement in perception that would follow from this anticipatory amplitude suppression in the cued condition (i.e., the leftward shift along the regression line equal to the decrease in amplitude after cueing; Fig. 2*C*). Third, we calculated the percentage of the attentional improvement that can be explained by the amplitude suppression (denoted the percentage explained improvement) by taking the ratio of predicted over actual attentional improvement and scaling it as a percentage. As illustrated by the vertical distance between the amplitude-

perception regression line and the mean cued amplitude (blue data point), only part of the attentional improvement can be explained by the anticipatory amplitude suppression. For example, according to the relation between amplitude and perception in Fig. 3*A*, *top*, the anticipatory amplitude suppression in the α-band would lead to an improvement of ~5% in hit rate. The actual improvement was ~65%, and therefore the anticipatory amplitude suppression explains $5 \div 65 \times 100 = 7.7\%$ of the attentional improvement.

The percentage explained improvement was calculated for each subject and averaged subsequently. Explained improvement was highest for contralateral amplitudes: for the α-band this was 5.8 ± 1.5% ($P < 0.005$), and for the β-band this was 7.0 ± 1.5% ($P < 0.001$).

For reaction times (Fig. 3*C*), only contralateral β-band amplitude predicted behavior [$t(11) = 2.938, P < 0.05$] and explained 12.5 ± 6.0% of the attentional improvement. This phenomenon was less robust, and therefore we will exclusively focus on hit rate in the following.

We investigated to what extent α- and β-band amplitude in the contralateral hemisphere contributed independently to the explained improvement. We found that combining contralateral α and β explained only an additional 1.7 ± 0.8% [$t(11) = 2.065, P = 0.063$] of the improvement compared with using only contralateral β (an increase in explained improvement from 7 to 8.7%). This small and nonsignificant improvement is a consequence of the fact that amplitude fluctuations in the α- and β-band are highly correlated, and therefore their influence on somatosensory perception is not unique. In fact, we ob-

1 Anticipatory amplitude suppression
2 Improvement predicted from suppression
3 Improvement observed with attentional orienting

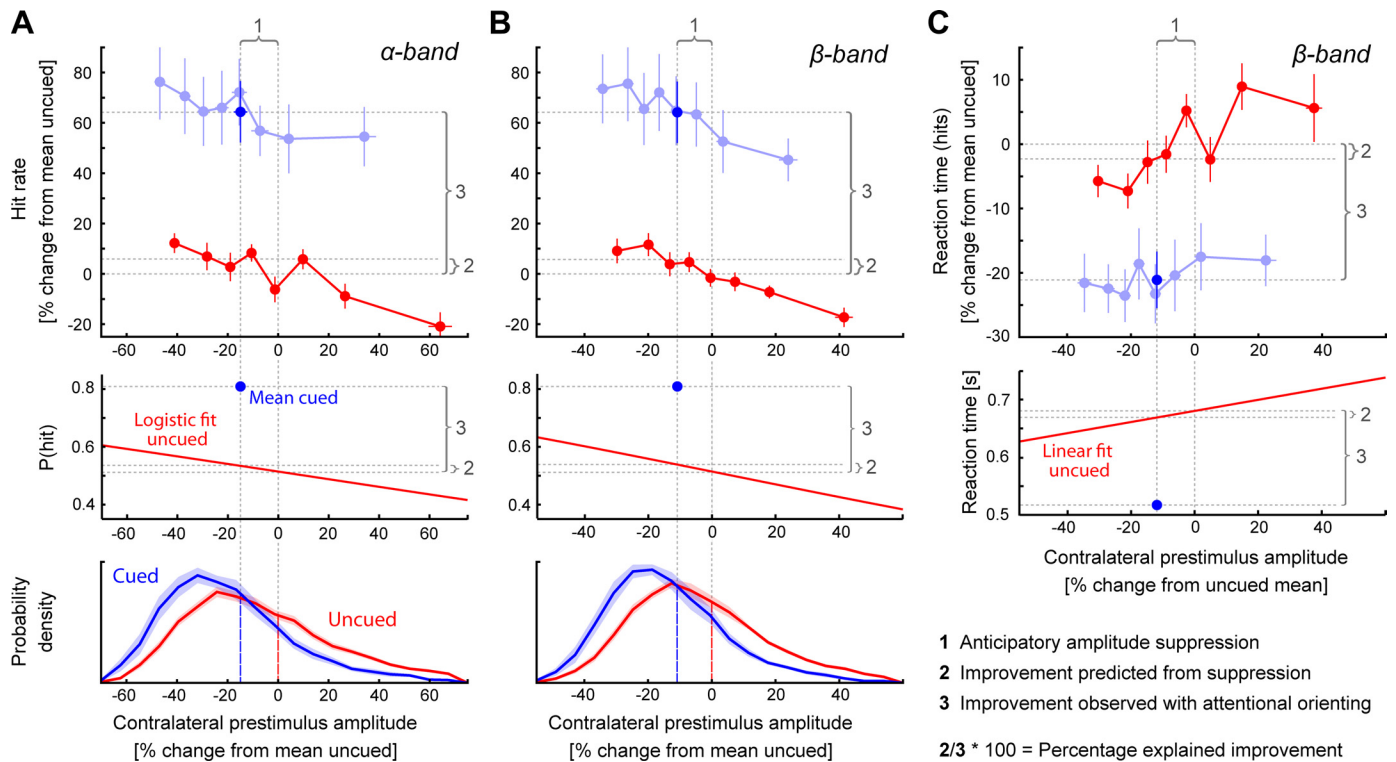**2/3** * 100 = Percentage explained improvement

Fig. 3. Rationale for the quantification of the percentage of attentional improvement explained by amplitude suppression. *A, top*: hit rate as a function of prestimulus amplitude in the α-band. Trials from the uncued (red) and cued (blue) condition were separately sorted by amplitude and grouped into 8 bins. Data are normalized as the percentage change from the mean in the uncued condition. The dark blue data point shows mean amplitude and hit rate in the cued condition, which was used for calculation of the percentage explained improvement. Error bars indicate SE. *Middle*: fitted regression slope, analog to binned data on *top*. Slope is depicted for the average subject. In practice, quantification was performed per subject. P(hit) represents the probability of a correct detection. *Bottom*: distribution of prestimulus amplitudes, separately for cued and uncued trials. Vertical dashed lines indicate mean amplitude. *B*: same as *A* for amplitudes in the β-band. *C*: same as *A* showing reaction time (in hit trials) as a function of β-band amplitude. Quantification of the percentage explained improvement involved *1*) estimating the anticipatory amplitude suppression, *2*) calculating the predicted improvement in perception that would follow from this anticipatory suppression, and *3*) comparing the predicted improvement with the observed improvement. This calculation was based on the fitted regression lines (*middle*).

served an average across-trial correlation between α- and β-amplitude of 0.564 ± 0.038. (Because of the inherent unreliability of the amplitude estimates, this correlation underestimates the true correlation. We will correct for this later.)

We may have underestimated the explained improvement because our predictor variables may not have been the most predictive ones. To assess this, we investigated to what extent the explained improvement depends on the spatial, spectral,
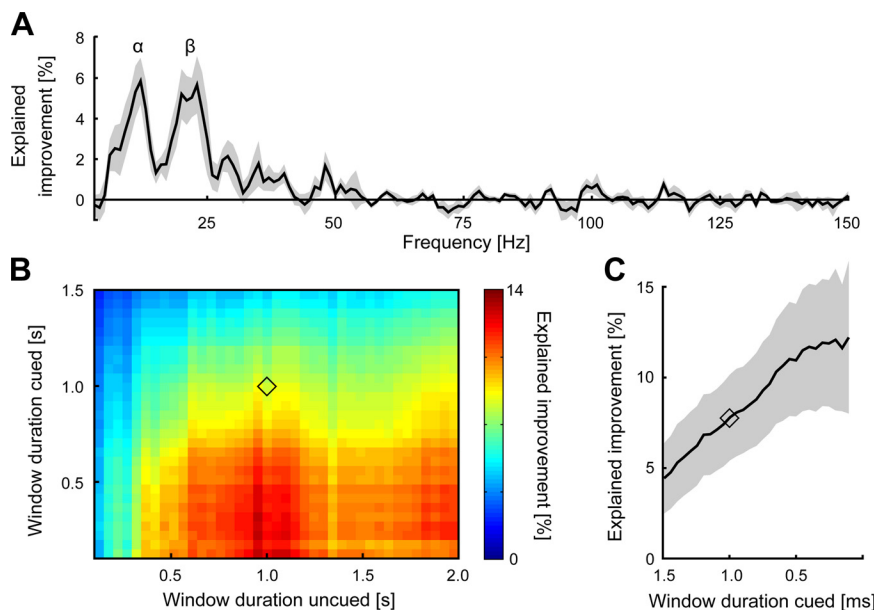

Fig. 4. Explained improvement is frequency-specific and depends on the duration of the time window. *A*: percentage explained improvement in hit rate as a function of the frequency of the prestimulus amplitude. Amplitude was estimated in a 1-s prestimulus window with 4-Hz frequency smoothing. Shading indicates SE. α And β are the only frequency bands that contribute to the explanation of the attentional improvement. *B*: percentage explained improvement as a function of the time-window duration used to estimate the amplitude in the uncued (horizontal axis) and the cued (vertical axis) trials. Contralateral α- and β-band amplitude were used as joint predictors. *C*: percentage explained improvement as a function of the time-window duration in the cued trials. Time-window duration for the uncued trials was fixed to 1 s. The diamonds in *B* and *C* indicate the time-window durations used in previous analyses. Time windows always ended at stimulus onset.
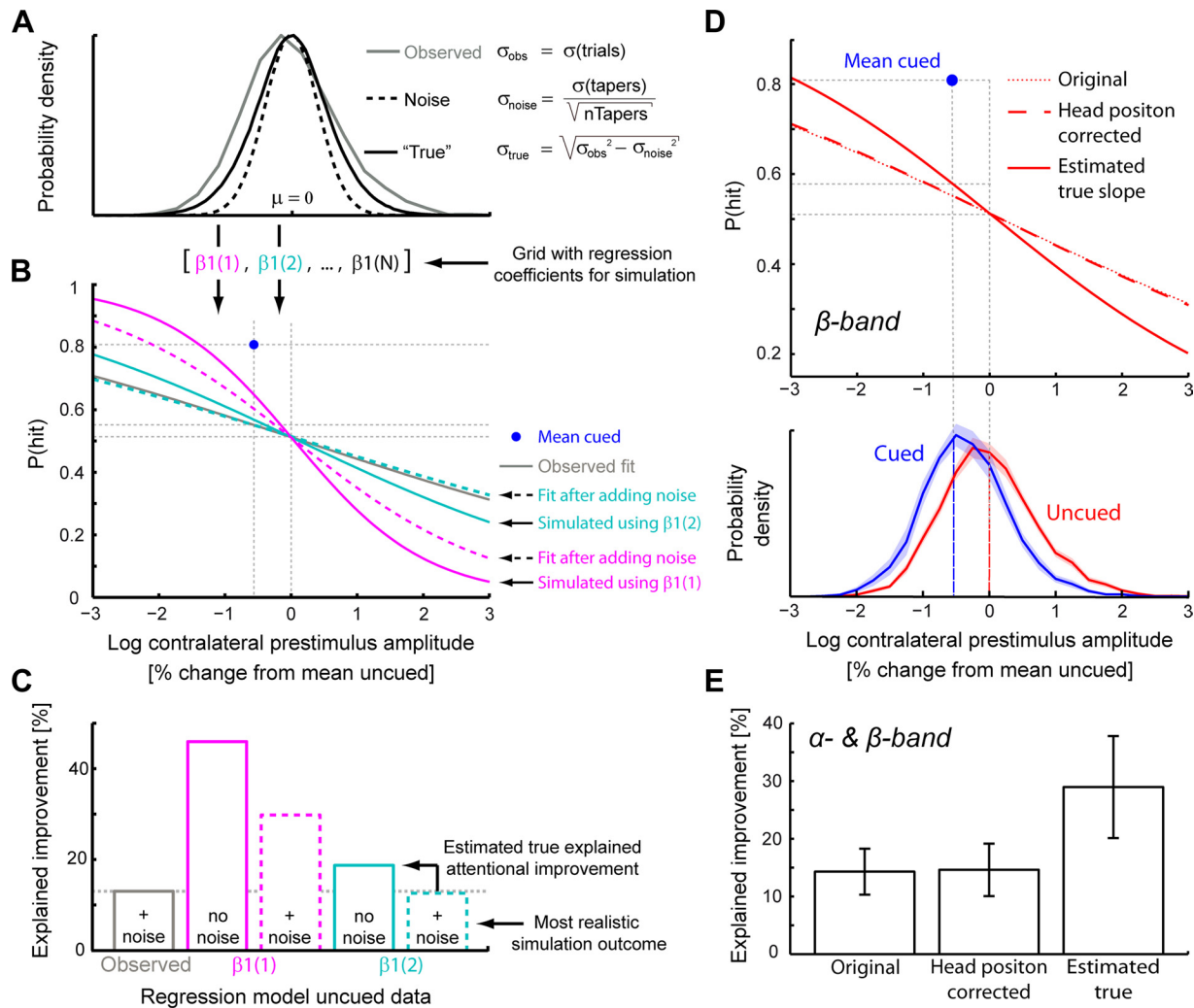
Fig. 5. Correcting for noise reveals maximal explained improvement. *A*: observed amplitude, derived noise, and reconstructed true amplitude distributions used in the simulation procedure. The observed distribution involves log-transformed amplitude and resembles a normal distribution. The noise distribution is modeled as a normal distribution with SD equal to the SE calculated across tapers. The true amplitude distribution is modeled as a normal distribution with variance equal to the difference between the observed and the noise variance. σ_{obs}, σ_{noise}, and σ_{true} refer to the SD of the observed, noise, and true distributions. μ Refers to their means. nTapers refers to the amount of tapers used in the estimation of oscillatory amplitude. *B*: true slopes were estimated by simulating the effect of adding the observed amount of noise to numerous (see grid between *A* and *B*) noise-free slopes. $\beta1(1)$, $\beta1(2)$, and $\beta1(N)$ refer to the logistic regression coefficients (see MATERIALS AND METHODS) used in *simulations 1*, *2*, and *N*. See MATERIALS AND METHODS for the amount of simulations (*N*) performed. *C*: the simulated noise-free slope that, after adding noise, most closely resembled our observed slope was taken as the estimated true slope. With this slope, estimated true percentage explained improvement was calculated. Data in *A–C* represent the average participant (we actually performed this simulation per participant and averaged the outcomes). For our multiple-predictor model (involving both α- and β-amplitude), we performed similar simulations using bivariate observed, noise, and true distributions. *D*: logistic regression fits for the originally observed, head-position-corrected, and combined head-position- and noise-corrected (estimated true) relation between prestimulus β-amplitude and hit-rate probability. *Bottom* shows log-transformed amplitude distributions, which approximate normal distributions (a requirement for our simulations). *E*: percentage explained improvement using the original, head-position-corrected, and estimated true slopes. Error bars indicate SE. α- And β-band amplitudes were used as combined predictors and estimated over 950- and 250-ms windows for uncued and cued data, respectively (Fig. 4).

and temporal aspects of our predictor variables. First, it may be that the S1 sources, which were extracted using a localizer, did not reflect the perceptually relevant α- and β-band amplitudes. We therefore extracted sources from the spatial topography of the anticipatory modulation (Fig. 2*D*) and performed the same analysis. With these new sources, we obtained highly similar results (α: 4.9 ± 1.5%; β: 7.1 ± 1.8%). Second, also pertaining to the spatial aspect, instead of unilateral amplitudes, we tested amplitude lateralization (the difference between contra- and ipsilateral S1 amplitude; cf. Thut et al. 2006) as a predictor. Fluctuations in amplitude lateralization did not predict performance (α: 1.8 ± 0.9%, $P = 0.066$; β: 0.5 ± 1.2%, $P = 0.669$).

Third, we performed a frequency-resolved analysis of the percentage explained improvement, of which the results are depicted in Fig. 4*A*. We found that α and β are the only frequency bands that contribute to the prediction. Finally, we investigated the dependence of our results on the prestimulus time windows used to estimate oscillatory amplitude. In line with studies in other groups (Jones et al. 2010; Linkenkaer-Hansen et al. 2004), we had initially used a 1-s prestimulus window for both cued and uncued trials. We now independently varied the duration of the two windows: the prestimulus window in the uncued trials used to estimate the regression slope and the prestimulus window in the cued trials used to

estimate the anticipatory modulation. Figure 4*B* shows the explained improvement as a function of both window durations, which we obtained using the combined model with contralateral α- and β-band amplitude as predictors. The relation between amplitude and hit rate in the uncued condition is relatively stable for time windows >600 ms. However, the anticipatory suppression in the cued trials increases with shorter time windows leading to a higher percentage explained improvement. After a cue, it takes some time to suppress oscillatory amplitudes, and hence the closer the window to the actual stimulus, the stronger the modulation (Fig. 4*C*; see also Fig. 2*C*). Using the best selection of time windows (950 ms uncued and 250 ms cued), the explained improvement was 13.1 $\pm$ 4.0% (compared with the 8.7 $\pm$ 1.8% using the original time windows; Fig. 4, *B* and *C*, $\diamond$).

Correcting for Noisy-Amplitude Estimates

Up to this point, we have treated our amplitude estimates as an accurate representation of neuronal activity. However, in fact, these estimates reflect a combination of true amplitudes (i.e., produced by α- and β-oscillations originating from contralateral S1) and noise. This is important to consider because noise in the predictor variable biases the slope of the regression line toward 0. This phenomenon is known as regression attenuation. This implies that noise in the amplitude estimates will lead to an underestimation of the slope of the true amplitude-perception regression lines and therefore of the explained improvement. (Note that variability in the criterion variable around the regression line does not affect our quantification; see DISCUSSION).

We dealt with this concern in two ways, one dealing with across-trial and another with within-trial noise. First, we corrected for the across-trial amplitude fluctuations caused by trial-by-trial fluctuations in head position (i.e., height of the head in the MEG helmet). Fluctuations in head position accounted for <5% ($r^2 = 0.048 \pm 0.015$) of the total variance in amplitude across trials. Correcting single-trial amplitudes based on single-trial head positions did not alter the slope of the amplitude-perception regression lines [α: $t(11) = 0.392$, $P = 0.703$; β: $t(11) = -0.318$, $P = 0.757$].

Second, we corrected for within-trials noise. For estimation of the noise variance, we made use of the fact that the multitaper method (Percival and Walden 1993) of spectral estimation provides multiple independent amplitude estimates from a single data window, namely one per taper (see MATERIALS AND METHODS). Using a simulation approach (see MATERIALS AND METHODS), we reconstructed the true (noise-free) amplitude-perception regression slope with which we could estimate the true percentage explained improvement. (Note that this percentage is only true under the assumption of signal stationarity. Alternatively, it may be considered an upper bound to the percentage explained improvement; see MATERIALS AND METHODS). Specifically, we drew data from a reconstructed true amplitude distribution (Fig. 5*A*) and investigated the consequences of adding the empirically observed amount of noise (Fig. 5*B*). By simulating multiple amplitude-perception regression slopes and adding noise to these, we derived which true amplitude-perception regression slope would have yielded the one that was actually observed (Fig. 5*C*). This is called the estimated true amplitude-perception relation. Figure 5*C* shows

the fitted logistic regression lines belonging to the originally observed (noise-dependent and thus attenuated), the head-position-corrected, and the estimated true slopes, all three obtained using β-amplitude as predictor.

We performed similar simulations using the model with both α- and β-amplitudes as predictors. For these simulations, we used bivariate true and noise distributions for which the covariance structures were determined by the true amplitude correlation (across trials) and the noise correlation (across tapers), respectively. Because the within-trial noise of α- and β-band amplitude estimates were largely uncorrelated ($r = 0.048 \pm 0.009$), we had previously underestimated the signal correlation: the estimated true correlation was 0.791 $\pm$ 0.037. Figure 5*D* shows the results for this combined model. When correcting for noise in our amplitude estimates, anticipatory suppression of oscillatory amplitudes can account for maximally 29 $\pm$ 8.8% of the improvement in perception that occurs with attentional orienting.

DISCUSSION

Systems and cognitive neuroscience have been very successful in identifying neural signals that are involved in cognitive functions and behavior. This is an important first step in understanding the neurophysiological mechanisms by which cognition and behavior are realized. An essential next step is quantifying how much particular signals, either alone or in combination, contribute to cognition and behavior. Here, we employed a novel method that produces such a quantification by using the relation between spontaneous neural and behavioral fluctuations to determine how much of the task-induced behavioral modulation can be explained by the cooccurring neural modulations. We show that *1*) extracranially recorded amplitude modulations from contralateral primary sensory cortex can, at best, account for 29% of the perceptual improvement with attentional orienting, and *2*) distinct aspects of this signal, α- and β-band oscillatory amplitudes, have a largely shared contribution.

Discussing the results of our study, one can focus on the explained or the unexplained part of the attentional improvement. The maximal percentage explained (29%) was obtained from local oscillatory amplitudes extracted from extracranial signals that were recorded using MEG. It is commonly believed that this oscillatory activity reflects synchronized postsynaptic currents (Hari and Salmelin 1997) with amplitude suppression resulting from desynchronization of these currents (Naruse et al. 2010; Pfurtscheller and Lopes da Silva 1999). Crucially, postsynaptic currents are not related in a one-to-one fashion to neuronal spiking, the signal for targeted communication between brain areas. From this perspective, it is revealing that a signal that depends on desynchronization of neuronal input, and that is recorded extracranially, might explain up to 29% of the attentional improvement. An important question therefore pertains to how anticipatory desynchronization in S1 influences the efficacy by which upcoming sensory information (coded as spiking activity) is represented and transmitted between brain areas. Concerning representation, desynchronization might reduce common (noise) fluctuations between neurons, thereby increasing the coding capacity of the neuronal population (Zohary et al. 1994). Indeed, the desynchronized state is a fundamental aspect of cortical processing (Harris and

Thiele 2011), and a reduction of correlated spike-rate fluctuations by attention has been observed in sensory cortex (Cohen and Maunsell 2009; Mitchell et al. 2009). Concerning transmission, recordings in multiple layers of sensory cortex have revealed that attention-induced desynchronization of α-oscillations occurs primarily in infragranular layers (Buffalo et al. 2011). Because these layers project to upstream areas (Buffalo et al. 2011), the observed anticipatory desynchronization in S1 might reflect preparatory regulation of the efficacy by which thalamic relay nuclei can impact on S1 (Suffczynski et al. 2001).

At the same time, our data show that at least 71% of the attentional improvement must be explained by signals from different areas and/or different types of signals. Importantly, this holds only for signals that are at least partially uncorrelated with the amplitude modulations that we observed. For example, a frontoparietal control region might be involved in orienting of attention (Bressler et al. 2008), but its signals will not explain additional attentional improvement if its only function is to induce amplitude modulations in sensory cortex (Capotosto et al. 2009). Concerning signals from different areas, there may be a central role for the thalamus (Saalmann and Kastner 2011). Consistent with this, thalamic activity is modulated by attention (O'Connor et al. 2002), and ongoing fluctuations in thalamic BOLD activity predict somatosensory detection (Boly et al. 2007). Next, secondary somatosensory cortex (S2) is also modulated by attention (Chapman and Meftah el 2005; Steinmetz et al. 2000). Importantly, attentional modulation in S1 and secondary somatosensory cortex may occur independently (Chapman and Meftah el 2005), allowing for additional explained improvement. Finally, previous studies have suggested that perception is influenced by the balance in activity between relevant (contralateral) and irrelevant (ipsilateral) cortical areas (Drevets et al. 1995; Thut et al. 2006; but see Cohen and Maunsell 2011). However, this could not be confirmed in our study: *1*) the anticipatory modulation only involved a contralateral suppression (Fig. 2*D*; in line with van Ede et al. 2011); and *2*) the balanced amplitude measure (contralateral − ipsilateral) did not predict performance. Concerning different types of signals, additional attentional improvement is likely accounted for by signals on a finer spatial scale (e.g., anticipatory spike-rate increases; Luck et al. 1997; Meftah el et al. 2009). Note here that such different signal types might explain additional attentional improvement, not because of differences in signal quality (because noise is corrected for in our quantification) but because they are sensitive to distinct neural processes. An important goal for future experiments will be to reveal the extent to which these signals and the observed oscillatory amplitude suppression independently contribute to attentional improvement.

We found that both α- and β-band amplitude modulations explain the attentional improvement: amplitude in both frequency bands is suppressed during tactile anticipation (present study; Anderson and Ding 2011; Jones et al. 2010; van Ede et al. 2011) and is related to perceptual performance (present study; Haegens et al. 2011a; Jones et al. 2010; van Ede et al. 2011). However, because α- and β-band amplitudes were highly correlated, their contributions to somatosensory perception and its improvement with attentional orienting were largely shared. This contrasts with a number of observations that suggest a dissociation between α- and β-band oscillations:

compared with α-band oscillations, β-band oscillations *1*) localize more anterior (Salmelin and Hari 1994), *2*) are modulated with a higher temporal flexibility during temporal orienting of somatosensory attention (van Ede et al. 2011), and *3*) rebound earlier after tactile input (Cheyne et al. 2003). Open questions remain with respect to the functional overlap between α- and β-band oscillations and their underlying neurophysiological mechanisms (Jones et al. 2009).

A common method to establish the involvement of a neural signal in behavior is demonstrating its correlation with that behavior. However, we believe that demonstrating a correlation is inferior to the quantification proposed in this paper. The main problem with the correlation coefficient is its dependence on sources of variability that are not of interest from the perspective of the phenomenon under investigation (in our case, behavioral improvement with attention). This becomes most clear when this irrelevant variability is reduced by trial-averaging the neural and behavioral measures. For example, we observed that binned prestimulus β-amplitude (Fig. 3*B*) correlated almost perfectly ($r = -0.976$) with hit rate. This contrasts with the trial-by-trial correlation between hit rate and β-amplitude, which was as low as -0.078 ± 0.020. There are two reasons for this low correlation: *1*) the measured neural signals are inherently unreliable; and *2*) single-trial fluctuations in behavioral responses to an identical stimulus are partly determined by stochastic fluctuations that are irrelevant to the neural mechanisms that underlie some cognitive phenomenon of interest (e.g., in our setup, electrical conductivity fluctuations at the thumb due to sweating, which are most likely unrelated to attention). This 2nd reason is the most important 1 because the attenuating effect of the unreliability of the measured neural signals (the 1st reason) can be corrected for in a similar way as we did for our quantification. The crucial point is that, in contrast to conventional correlational analyses, behavioral variability due to irrelevant stochastic fluctuations does not affect our quantification. In fact, this variability contributes to the variability around the regression lines, whereas our quantification only depends on the vertical distance between these regression lines.

As pointed to above, our quantification is not affected by fluctuations in variables that affect behavior as long as these fluctuations occur independently of our experimental conditions (i.e., the cued and uncued conditions). This holds true even for cognitively relevant fluctuations such as those involved in motivation. Throughout the experiment, motivation might fluctuate, and this may affect behavior. (This may occur via fluctuations in amplitude, which might contribute to the slopes of our perception-amplitude regression lines.) Crucially, however, if these fluctuations occur independently of the experimental conditions, they will leave the ratio between the predicted and the actual improvement unaltered. (Analogously, our quantification is unaffected by the motivation of the subject: subjects who pay more attention with cueing will have a larger shift in both amplitude and perception but not a larger ratio between the 2.) Alternatively, those variables for which fluctuations do depend on our experimental conditions (e.g., motivation might be higher in cued trials) are considered attentional by definition, and these will go into our quantification. As desired, the part via which such variables influence perception through amplitude modulations will go into the explained part, whereas the part via which they influence

perception through neural processes that are not reflected in this signal will go into the unexplained part.

The knowledge provided by a cognitive or systems neuroscience experiment relies on the relations between a cognitive state or behavior and a limited set of neural signals, none of which reflect all relevant aspects of neuronal processing. To evaluate the scope of any such observed relation, it is thus important to take explicitly into account the degree to which the investigated signal is behaviorally relevant. Unfortunately, for many signals, a quantification of this relevance has not been established yet. In our study, we used oscillatory amplitudes that were recorded extracranially using MEG and carefully scanned the spatial, spectral, and temporal dimensions of this signal (Fig. 4). We demonstrate substantial behavioral relevance. At the same time, our quantification reveals the degree to which relevant neuronal processes are invisible in extracranially (MEG) recorded oscillatory amplitudes and thus must be explored in different signals or, alternatively, in different aspects of the same signal such as oscillatory amplitude and phase (e.g., Busch et al. 2009).

The paradigm that is introduced here can be more widely applied to investigate the contribution of other neural signals to attentional improvement. First, contribution of similar signals in different sensory modalities can be investigated (e.g., occipital α in a visual task). Second, instead of behavioral performance, the response variable can be a neurophysiological signal that is affected by attention (e.g., an evoked response). Third, the neurophysiological signal that is used to predict behavior can be recorded during stimulus processing rather than during anticipation. Fourth, other signals, such as BOLD or spiking activity, can be used to represent the brain state. In addition, by combining multiple predictors in a regression model, it is possible to investigate to what extent distinct signals independently contribute to the response variable. This can be used to investigate, for example, whether frontoparietal, thalamic, and sensory regions independently contribute to the attentional improvement and also whether this holds for BOLD and simultaneously recorded electrophysiological signals or anticipatory and stimulus-induced signals.

Concluding, we have, to our knowledge, for the first time quantified the cognitive/behavioral relevance of a particular neural signal in the context of attentional orienting: MEG-recorded anticipatory α- and β-band amplitude modulations have a largely shared contribution and account for maximally 29% of the improvement in perception that occurs with attentional orienting. Our study reveals the behavioral relevance of extracranially recorded oscillatory signals, and our method provides a new means to quantify how much a particular set of neural signals contribute to cognitive, behavioral, and neurophysiological phenomena.

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GRANTS

DISCLOSURES

AUTHOR CONTRIBUTIONS

F.v.E., M.K., and E.M. conception and design of research; F.v.E and M.K. performed experiments; F.v.E., M.K., and E.M. analyzed data; F.v.E., M.K., and E.M. interpreted results of experiments; F.v.E. and M.K. prepared figures; F.v.E., M.K., and E.M. drafted manuscript; F.v.E., M.K., and E.M. edited and revised manuscript; F.v.E., M.K., and E.M. approved final version of manuscript.

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