Enhanced Alpha-oscillations in Visual Cortex during Anticipation of Self-generated Visual Stimulation

Max-Philipp Stenner[1,2], Markus Bauer[1], Patrick Haggard[1], Hans-Jochen Heinze[2], and Ray Dolan[1]

Abstract

■ The perceived intensity of sensory stimuli is reduced when these stimuli are caused by the observer's actions. This phenomenon is traditionally explained by forward models of sensory action–outcome, which arise from motor processing. Although these forward models critically predict anticipatory modulation of sensory neural processing, neurophysiological evidence for anticipatory modulation is sparse and has not been linked to perceptual data showing sensory attenuation. By combining a psychophysical task involving contrast discrimination with source-level time–frequency analysis of MEG data, we demonstrate that the amplitude of alpha-oscillations in visual cortex is enhanced before the onset of a visual stimulus when the identity and onset of the stimulus are controlled by participants' motor actions. Critically, this prestimulus enhancement of alpha-amplitude is paralleled by psychophysical judgments of a reduced contrast for this stimulus. We suggest that alpha-oscillations in visual cortex preceding self-generated visual stimulation are a likely neurophysiological signature of motor-induced sensory anticipation and mediate sensory attenuation. We discuss our results in relation to proposals that attribute generic inhibitory functions to alpha-oscillations in prioritizing and gating sensory information via top–down control. ■

INTRODUCTION

Immediate consequences of voluntary actions are subject to sensory attenuation. A prominent example is the perceived intensity of self-applied tactile stimuli, which is reduced when compared with identical, externally generated stimuli (Bays, Wolpert, & Flanagan, 2005). Similarly, a reduction of perceived loudness and visual contrast have been demonstrated for self-generated auditory (Weiss, Herwig, & Schütz-Bosbach, 2011) and visual (Cardoso-Leite, Mamassian, Schütz-Bosbach, & Waszak, 2010) stimuli, respectively.

Across modalities, sensory attenuation has been considered within theoretical frameworks that link motor processing to perception of sensory action consequences (Waszak, Cardoso-Leite, & Hughes, 2012; Schütz-Bosbach & Prinz, 2007; Wolpert, Ghahramani, & Jordan, 1995). Here, sensory attenuation is often explained by consistency between forward models of anticipated action effects (corollary discharge; Sperry, 1950) and actual sensory feedback. Neural processes that functionally correspond to corollary discharge have been reported based on invasive recordings in animals (Poulet & Hedwig, 2007; Sommer & Wurtz, 2006). Equivalent evidence in humans is sparse, predominantly indirect (i.e., speaking in favor of motor prediction pathways upstream of sensory areas), and rarely linked to sensory attenuation (Ostendorf, Kilias, & Ploner, 2012; Kühn, Seurinck, Fias, & Waszak, 2010; Voss, Ingram, Haggard, & Wolpert, 2009; Haggard & Whitford, 2004).

Because of its presumed motor origin, corollary discharge is regarded as physiologically distinct from sensory predictions that are purely based on external events. Because corollary discharge is thought to reflect motor forward modeling, it implies anticipated control over a stimulus. Corollary discharge signals should therefore vary with the extent to which an action is expected to control a subsequent stimulus and should not simply reflect predictability of this stimulus from other sensory events. However, in a recent review, Hughes, Desantis, and Waszak (2012) concluded that the majority of studies on sensory attenuation potentially confound stimulus control and stimulus predictability. Because stimulus predictability is well known to modulate sensory processing (e.g., Alink, Schwiedrzik, Kohler, Singer, & Muckli, 2010), this confound can lead to misinterpretations. Other studies of sensory attenuation are based on a task design that introduces another potential confounding factor. These studies typically compare stimuli that are contingent on an action with identical stimuli that are not preceded by a motor response (e.g., Weiss et al., 2011; see Hughes et al., 2012, for a review). Such a comparison introduces a difference in motor output in addition to differences in control, which complicates interpretation of any observed effects as specific to control.

Here, we used a task that varied the extent to which an action controlled a subsequent stimulus while keeping both stimulus predictability and motor output comparable

[1]University College London, [2]University of Magdeburg

Journal of Cognitive Neuroscience 26:11, pp. 2540–2551
doi:10.1162/jocn_a_00658

across conditions. We combined MEG and a visual contrast discrimination task to test for an anticipatory, that is, prestimulus, modulation of sensory processing in relation to perceptual evidence of sensory attenuation. The majority of previous MEG/EEG studies on sensory attenuation in humans have restricted their analyses to specific stimulus-evoked responses (Gentsch & Schütz-Bosbach, 2011; Hughes & Waszak, 2011; Hesse, Nishitani, Fink, Jousmäki, & Hari, 2010; Baess, Widmann, Roye, Schröger, & Jacobsen, 2009; Martikainen, Kaneko, & Hari, 2005). Here, we focus on neural oscillations in a prestimulus time interval, motivated by the finding that induced responses convey a corollary discharge signal in speech (Chen et al., 2011). Accumulating evidence points to alpha-oscillations in visual cortex as a representation of pulsed inhibition by feedback projections that prioritize the processing of task-relevant (Jensen & Mazaheri, 2010) and salient stimuli (Jensen, Bonnefond, & VanRullen, 2012). Closely related functions have been proposed for sensory attenuation, namely the prevention of self-induced desensitization and the prioritizing of externally caused stimuli (Lally & Frendo, 2011; Poulet & Hedwig, 2007). On this basis, we hypothesized that anticipation of a visual stimulus that is controlled by one's action would be reflected in an increase in alpha-amplitude in visual cortex and predict sensory attenuation at the perceptual level.

METHODS

Participants

Ten healthy volunteers participated in the study (mean age = 24.8 years, *SD* = 4.7 years, six women). All participants were recruited via an on-line database. They gave written informed consent before participation with the right to exit the study at any time. The study was approved by the local ethics committee (University College London, UK). Participants received £10 per hour as reimbursement.

Task and Experimental Procedure

The task was designed to vary participants' control over the onset and orientation of a visual stimulus while minimizing differences in motor output and stimulus predictability across conditions. There were three blocked conditions: a "motor/control" condition, in which participants controlled the onset and orientation of a static Gabor patch on a computer screen with their button presses; a "motor/no control" condition, in which participants' button presses had no influence on the onset and orientation of this Gabor patch; and a "no motor" condition, in which the Gabor patch was presented without a preceding button press. The "motor/no control" condition served to avoid a potential confound of control with motor output present in many previous studies. In addition, cues that contained information about the orientation and timing of the Gabor patch in this condition controlled for stimulus predictability. The "no motor" condition was included for comparability with previous studies in which conditions without motor output served as a baseline (typically labeled "effect only" conditions in these studies).

All conditions included a contrast discrimination task to test for psychophysical evidence of sensory attenuation. For this discrimination task, each Gabor patch was followed by a second Gabor patch, which was either of higher, lower, or identical contrast relative to the first. Participants had to indicate whether the first or the second of the two stimuli was the one of higher contrast by pressing one of two buttons. Sensory attenuation is known to decrease over the course of a few hundred milliseconds (Aliu, Houde, & Nagarajan, 2009; Bays et al., 2005). Accordingly, we expected sensory attenuation to affect the first of the two Gabor patches, presented about 50 msec after the button press, more strongly than the second, which followed the first after a SOA between 1000 and 1250 msec. Therefore, we could predict that sensory attenuation would become manifest as a stronger bias to report the first stimulus as the low-contrast one, similar to previous studies that reported shifts in the point of subjective equality (PSE) as a measure of sensory attenuation (Desantis, Weiss, Schütz-Bosbach, & Waszak, 2012; Weiss et al., 2011; Haggard & Whitford, 2004).

We used three constant stimulus levels, each presented in one third of trials in each of the three conditions. The contrast of the first Gabor was held constant at 0.25 Michelson contrast to keep it predictable, and only the contrast of the second Gabor was varied from trial to trial. Contrast levels for trials in which the second Gabor was of higher or lower contrast than the first were individually determined by a staircase procedure before the main task.[1] Participants were informed that the two Gabor patches presented per trial were always of different contrasts. Responses in the contrast discrimination task were given by pressing one of two buttons, operated with the left middle and index fingers. There was no time limit for these responses. Gabor patches (spatial frequency = 0.8 cycles/degree, phase = +90°, size (*SD*) = 1.25°) were presented in the lower left visual field (at −3.0°/−3.0°) on a gray background for 250 msec. This eccentric stimulus presentation was chosen for two reasons: First, MEG is less sensitive to signals evoked by foveally presented visual stimuli versus stimuli above or below fixation (due to the orientation and functional architecture of the calcarine sulcus). And second, stimulus presentation in the left visual hemifield helped to spatially dissociate motor-related signals (in sensorimotor areas of the left hemisphere; relevant responses were given with the right hand) and consequence-related signals (in visual areas of the right hemisphere). The paradigm, including all stimuli, was programmed using Presentation software (Neurobehavioral Systems, www.neurobs.com).

"Motor/Control" Condition

In the "motor/control" condition (Figure 1A, top) participants pressed a button to trigger the sequential presentation of the two Gabor patches on the screen. The onset time and orientation of the first Gabor and the orientation of the second Gabor were fully determined by this button press. Participants were required to perform these button presses as follows.

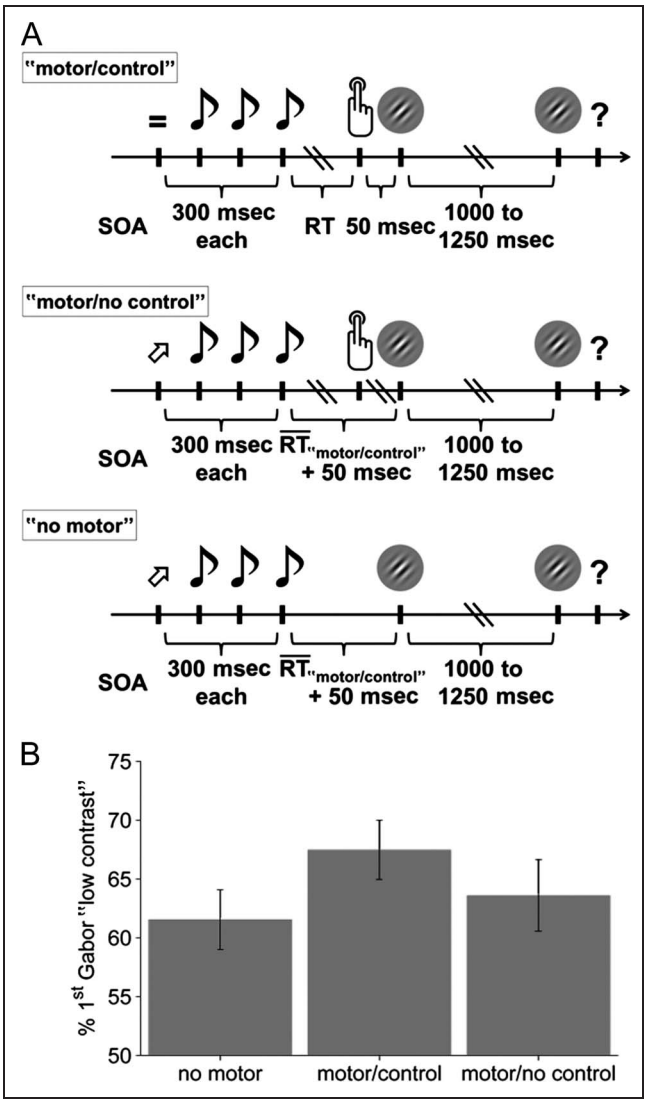

Figure 1. Task and behavioral results. (A) Timing of events in "motor/control" (top), "motor/no control" (middle), and "no motor" (bottom) conditions. Each trial started with the presentation of a visual cue, which signaled the button/orientation mapping in the "motor/control" condition and the orientation of upcoming Gabor patches in "motor/no control" and "no motor" conditions. This visual cue was followed by an auditory cue, which signaled the start of the response window in "motor/control" and "motor/no control" conditions and predicted the onset time of the first Gabor in "motor/no control" and "no motor" conditions. Two Gabor patches were presented sequentially in each trial. Participants had to indicate which of the two was of higher contrast at the end of each trial (first or second Gabor patch). The crucial experimental manipulation was a variation of the extent to which participants' button presses determined the onset and orientation of the first Gabor patch. Note that trial events and average timing of these events was balanced across "motor/control" and "motor/no control" conditions. (B) Percentage of trials in which the first Gabor patch was reported as the lower-contrast stimulus in the three conditions (mean ± *SEM*).

On each trial, participants chose between two alternative buttons, operated by their right index and middle fingers. They were instructed to choose the two buttons approximately equally often across each block (of 26 trials). They received feedback on the relative frequencies of their choices at the end of each block. On each trial, this choice determined the orientation of the two subsequently presented Gabor patches, which could be 45° clockwise or 45° counterclockwise. The mapping of the two buttons onto the two orientation varied from trial to trial, so that, for example, pressing the index finger button led to Gabor patches oriented 45° clockwise in some trials and to stimuli of the opposite orientation in others. On any given trial, the current valid mapping was signaled by a visual cue from the beginning of each trial onwards (presented centrally, visual angle 1°, white). This cue could either be an equal sign, meaning that the index finger button would lead to Gabor patches oriented 45° counterclockwise and the middle finger button to Gabor patches oriented 45° clockwise, or a bidirectional arrow, which signaled the opposite mapping. Our motivation to avoid a constant mapping was to keep consistency of the mapping across trials similarly low as in the "motor/no control" condition, where this mapping was incidental (see "motor/no control" condition below). Note that, because of the visual cue, participants could fully predict the orientation of Gabor patches from their button choice on each trial. Participants were familiarized with both mappings and the meaning of the visual cues in a practice session before the main experiment.[2]

In addition to controlling stimulus orientation, participants also determined the onset time of the first Gabor, which was programmed to be 50 msec after the button press (measured time between button press and Gabor onset: 59 ± 5 msec [mean ± *SD*]; this slight deviation from the programmed value results from the jitter of response timing with respect to vertical refreshes of the screen [at 60 Hz]). To be able to achieve, on average, comparable timings of trial events in the "motor/control" condition as in the "motor/no control" condition (where Gabor onset was independent of RT, see below), participants had to press the button that triggered Gabor presentation within a short time window. This time window was signaled by an auditory cue, which consisted of three consecutive sine wave tones (700 Hz, 700 Hz, and 1000 Hz; duration = 75 msec; SOA = 300 msec, binaurally presented over headphones; the first tone was played 300 msec after the onset of the visual cue). Participants had to press a button within 500 msec after the end of the third tone of this auditory cue in order for the Gabor patches to be presented. Earlier or later button presses produced an error sound and no Gabor presentation.

Lastly, to emphasize the dependency of stimulus presentation on button presses, we introduced infrequent no-go trials, which were signaled by a color change of the fixation cross from white to red at the beginning of the trial (replacing the visual cue present in all other trials). No Gabor and no error sound were presented upon successfully withholding any right-hand button press in these trials. There were two no-go trials in each block (of 26 trials).

In summary, participants could control and predict the presentation, timing, and orientation of Gabor patches by their button presses in the "motor/control" condition.

"Motor/No Control" Condition

Similar to the "motor/control" condition, the "motor/no control" condition (Figure 1A, middle) required participants to press one of the two right-hand buttons following the auditory cue. However, RT and choice of button had no influence on the timing and orientation of the first Gabor patch, as its orientation was predetermined pseudorandomly and its onset was time-locked to the auditory cue.

Nevertheless, both Gabor orientation and onset were predictable from the visual and auditory cues in this condition: Orientation was cued from the beginning of each trial onwards by arrows pointing "northwest" (for Gabor orientation of 45° counterclockwise) or "northeast" (for Gabor orientation of 45° clockwise) (presented centrally, visual angle 1°, white).[3] Gabor onset followed the last tone of the auditory cue after the mean RT in the preceding "motor/control" block plus 50 msec (the programmed action–outcome delay in "motor/control" blocks).

Participants were explicitly instructed to choose button presses independently of orientation cues and succeeded in doing so (button choices corresponded to the cued direction in 49.8% of trials across participants, $t(9) = .4$, $p = .68$ [dependent samples t test on the number of trials in which button choice corresponded to the cued vs. the opposite orientation]). Similar to the "motor/control" condition, participants were also instructed to press the left and right buttons approximately equally often across each block. They received feedback on the relative frequencies of these button choices at the end of each block.

As in the "motor/control" condition, "motor/no control" blocks also contained infrequent no-go trials, in which Gabor patches were presented irrespective of whether participants withheld their response or not to further reduced the action–stimulus contingency.

Thus, our design ensured that "motor/control" and "motor/no control" conditions were balanced for motor output, visual input, and, at least to some extent, predictability of stimulus onset and orientation. They differed primarily in participants' control over stimulus onset and orientation.

"No Motor" Condition

For comparability with previous studies and as a localizer condition for our MEG analysis, we included "no motor" blocks (Figure 1A, bottom). These were identical to "motor/no control" blocks, including the information carried by the visual and auditory cues with respect to Gabor orientation and onset, with the exception that participants were instructed to omit right-hand responses altogether throughout the entire blocks.

Experimental Procedure

All three parts of the experiment (the staircase, the practice, and the main task) were completed twice by each participant, on two consecutive days, to increase power of the behavioral data. MEG data were acquired on the second day only. In the main task, blocks of the "motor/control" and "motor/no control" conditions alternated (starting with a "motor/control" block). There was one "no motor" block every four blocks. In total, participants completed 30 blocks on the first day (12 "motor/control," 12 "motor/no control," 6 "no motor") and 20 blocks on the second (8 "motor/control," 8 "motor/no control," 4 "no motor"). Each block consisted of 26 trials and lasted approximately 2–3 min.

MEG Recording and Analysis

MEG data were recorded continuously from 274 axial gradiometers (one SQUID was defunct) and 35 reference channels of a CTF Omega system, Port Coquitlam, BC, Canada at a sampling frequency of 1200 Hz. Head position was measured via three coils at the nasion and the tragi. Analysis used FieldTrip (http://fieldtrip.fcdonders.nl/; Oostenveld, Fries, Maris, & Schoffelen, 2011) and SPM8 (www.fil.ion.ucl.ac.uk/spm; Litvak et al., 2011). Procedures for preprocessing and artifact treatment were similar to previous work (Bauer, Kennett, & Driver, 2012; Bauer, Kluge, et al., 2012). In short, MEG data were epoched and inspected visually for amplitude jumps, muscle artifacts, and eye blink artifacts using standard options in FieldTrip ("ft_rejectvisual") based on a threshold for the maximum amplitude variance across channels in each trial. Remaining artifacts arising from eye movements were removed using PCA. Artifact rejection was done blind to condition. Line noise was removed from 5-sec periods around each trial using a narrow-band notch filter.

For sensor-level analysis, the artifact-free data were interpolated to a common sensor array template across participants to correct for interindividual variations in head positions. This spatial interpolation method, based on a minimum-norm projection of the data as an intermediate step, is a standard function in FieldTrip ("ft_megrealign") described in detail in previous work (Bauer, Kennett, et al., 2012). Planar gradients were then calculated using a nearest-neighbor method (Bastiaansen & Knösche,

2000) to better estimate the spatial distribution of cortical generators of the MEG field at the sensor level (Bauer, Kennett, et al., 2012).

Source reconstruction was based on individual T1 weighted MRI warped to the standard MNI brain. The first steps of forward modeling, including coregistration of MEG data and individual MRI, segmentation, calculation of a cortical source model with 8196 grid points (down-sampled to 823 grid points using reducepath.m in MatLab after computation of the lead fields), and computation of a single-shell volume conduction model, were performed using SPM8 (Litvak et al., 2011). These source- and volume-conduction models were entered into the lead field computation in FieldTrip. We used linearly constrained minimum variance beamforming to project the sensor data onto the cortical grid, based on the covariance matrix of the low-pass filtered data (<40 Hz), as described in detail in previous work (Bauer, Kluge, et al., 2012). These filters were identical across conditions. For filter computation, orientation was fixed to the direction of the spatial component with largest projected power separately for each grid point (a standard option in FieldTrip). The regularization parameter λ was set to 0 (the default).

Spectral analysis of the time courses at the sensor level and source level for each channel and grid point were performed between 2.5 and 40 Hz in steps of 2.5 Hz. A Fourier-transformed Hanning taper of 400-msec window length was multiplied with the Fourier-transformed data segments sampled every 100 msec between 500 msec before and after stimulus onset. For sensor-level data, vertical and horizontal components of the planar gradient were combined after spectral analysis. Power was calculated as the square of the magnitude of the complex-valued Fourier spectra. For low-frequency analyses, data were analyzed separately before and after removal of phase-locked ("evoked") responses from the spectrum (we show data after the removal of phase-locked components throughout the Results section). Phase-locked responses were removed from the complex-valued Fourier spectra before computation of power by subtracting the average of the Fourier spectra across trials from single-trial data for each individual, as described in previous work (Bauer, Kluge, et al., 2012).

Statistical analysis of sensor- and source-level power was based on a nonparametric randomization test (Maris & Oostenveld, 2007) implemented in FieldTrip, which corrects for multiple comparisons across channels/grid points and/or across time and frequency bins, depending on the settings. All results in our study are corrected for multiple comparisons across all sensors/grid points. In addition, our first analysis of power differences between "motor/control" and "motor/no control" conditions corrects for multiple comparisons across all time bins between 500 msec before stimulus onset and 500 msec after stimulus onset to define the time interval of interest for subsequent tests. Because we had a clear, physiologically motivated hypothesis regarding the frequency window of interest—the alpha frequency band—we focused on 7.5–12.5 Hz.

Nonparametric randomization tests have been widely used in previous studies on MEG time–frequency data (e.g., Van Dijk, van der Werf, Mazaheri, Medendorp, & Jensen, 2010; Van Dijk, Schoffelen, Oostenveld, & Jensen, 2008). The method is explained in detail at http://fieldtrip.fcdonders.nl/tutorial/cluster_permutation_freq/. In short, clusters comprising adjacent sensors, time bins, and frequency bins that exceed a threshold of the t statistic for a given contrast are defined ($p < .05$, dependent samples t tests across participants). A cluster-level statistic is derived for each cluster in the observed data by summing t values across its elements. The null hypothesis is rejected if this cluster-level statistic exceeds a critical value, which is determined by the distribution of the maximum cluster-level statistic after repeatedly permuting the observed data, that is, after randomly reassigning the data to the two conditions within participants and determining the maximum cluster-level statistic in this permuted data. p values are defined as the proportion of randomizations for which the maximum cluster-level statistic exceeds the cluster-level statistic in the observed data.

The majority of previous studies on sensory attenuation compared stimuli that were contingent on an action with identical stimuli in the absence of a preceding motor response (but see Cardoso-Leite et al., 2010; Bays et al., 2005). Here, we were primarily interested in differences between "motor/control" and "motor/no control" conditions, both of which required participants to perform a motor response before stimulus onset. Sensory attenuation is known to decrease over the course of a few hundred milliseconds following the motor action (Bays et al., 2005). Because of this decrease, we expected to find differential effects of "control" between "motor/control" and "motor/no control" conditions on power around the onset of the first versus the second Gabor patch. Thus, we first analyzed effects of "control" on the difference between the power spectral densities of the two stimuli. Note that we subsequently analyzed data for each of the two stimuli separately. No-go trials and trials without a button press within the response time window were excluded from analysis for all conditions.

RESULTS

Behavior

We focused on the percentage of trials in which the first Gabor patch was reported as the stimulus of lower contrast across the three objective contrast levels and across the 2 days of the experiment. We predicted a decrease of the perceived contrast of the first Gabor patch in the "motor/control" condition because of sensory attenuation and therefore an increase in the percentage of trials in which the first stimulus was reported lower in contrast

when compared with the "motor/no control" and "no motor" conditions.

As predicted, participants reported the first Gabor patch as the stimulus of lower contrast more frequently in the "motor/control" condition compared with the "motor/no control" and "no motor" conditions. The proportion of trials in which the first Gabor was reported as the low-contrast stimulus differed significantly between conditions (one-way repeated-measures ANOVA, factor Condition ["motor/control," "motor/no control," "no motor"]: $F(2, 18) = 5.69, p = .012$). Post hoc dependent samples t tests showed that participants reported the first stimulus as the low-contrast one significantly more often in the "motor/control" condition than in the "motor/no control" ($t(9) = 3.4, p = .008$) and in the "no motor" ($t(9) = 2.96, p = .016$) conditions (Figure 1B). There was no significant difference between the "motor/no control" and the "no motor" conditions ($t(9) = 1, p = .34$).[4]

In addition, we determined sensitivity (d') and response bias (criterion) for each condition. The bias to report the first Gabor patch as the low-contrast stimulus differed significantly between conditions (one-way repeated-measures ANOVA, factor Condition: $F(2, 18) = 4.29$, $p = .03$; criterion in the three conditions, mean ± SD: -0.35 ± 0.26 ["no motor"], -0.54 ± 0.25 ["motor/control"], -0.44 ± 0.32 ["motor/no control"]). Post hoc dependent samples t tests showed that this bias tended to be stronger in the "motor/control" than "motor/no control" condition ($t(9) = 2.12, p = .064$) and was significantly stronger in the "motor/control" than in the "no motor" condition ($t(9) = 2.57, p = .03$; note that this analysis was based on the subset of trials in which the contrasts of the two stimuli were not identical, reducing the signal-to-noise ratio). There was no difference between "motor/no control" and "no motor" conditions ($t(9) = 1.27, p > .2$). Sensitivity (d') did not differ significantly between conditions (one-way repeated-measures ANOVA, factor Condition: $F(2, 18) = .74, p > .4$; d' in the three conditions, mean ± SD: 1.4 ± 0.58 ["no motor"], 1.34 ± 0.42 ["motor/control"], 1.47 ± 0.42 ["motor/no control"]).

In summary, our behavioral results are consistent with sensory attenuation of the first Gabor patch in the "motor/control" conditions when compared with the "no motor" condition as well as to the "motor/no control" conditions. When participants controlled stimulus onset and orientation, they were more likely to report the first Gabor patch as the lower-contrast stimulus.

MEG

Here, we focused on alpha-power (7.5–12.5 Hz) in a time window between 500 msec before and 500 msec after Gabor onset. We focused on differences between the "motor/control" condition and the "motor/no control" condition, as these were balanced for motor output (as well as stimulus predictability). We predicted enhanced alpha-power over right occipital cortex in a prestimulus time window in the "motor/control" condition as compared with the "motor/no control." Responses that were phase-locked to stimulus onset ("evoked") did not contribute to the low-frequency spectra reported here as they were removed for both sensor- and source-level analyses (see Methods for details). Virtually the same results were obtained when looking at the total power without removal of transient phase-locked responses.

Sensor-level Analysis

Sensory attenuation is known to decrease over the course of a few hundred milliseconds after a motor action (Bays et al., 2005). Therefore, we first examined differential effects of stimulus control on the power spectral densities of the two stimuli (i.e., on the difference $\text{power}_{(\text{first Gabor})} - \text{power}_{(\text{second Gabor})}$), effectively testing for a "control" × "Gabor" interaction. We used this approach to determine channels that showed a significant differential modulation by "control" for the two stimuli, equivalent to the effect of stimulus control on our perceptual metric of sensory attenuation in the two alternative forced-choice task.

When testing for this interaction, correcting for multiple comparisons across all channels and time bins between 500 msec before and after stimulus onset, we found a significant positive cluster between 7.5 and 12.5 Hz during the 300 msec before stimulus onset in right occipital and parietal sensors (contralateral to the visual hemifield in which the Gabor patches were presented; $p = .044$, cluster-based correction for multiple comparisons across all channels and across all time bins between −500 msec and +500 msec; Figure 2A).[5]

This effect of stimulus control on the power difference was predominantly due to higher alpha-band power in the "motor/control" condition compared with the "motor/no control" condition in the prestimulus period corresponding to the first Gabor patch as opposed to the second: Power between 7.5 and 12.5 Hz was significantly higher in the "motor/control" condition than in the "motor/no control" conditions in right occipital and parietal sensors during the 300 msec before onset of the first Gabor patch ($p = .034$, cluster-based correction for multiple comparisons across all channels; power spectrum in Figure 2B, top, averaged across the channels highlighted in Figure 2A, top). The same contrast for the prestimulus period of the second Gabor patch did not yield any significant cluster (power spectrum in Figure 2B, bottom, averaged across the channels highlighted in Figure 2A, top). When comparing the "motor/control" condition and the "no motor" condition, we found a very similar cluster of right occipital sensors, which showed significantly higher alpha-power (7.5–12.5 Hz) up to 400 msec before the onset of the first Gabor patch ($p = .036$, corrected for multiple comparisons across all sensors). We found no significant difference between the "motor/no control" condition and the "no motor" condition

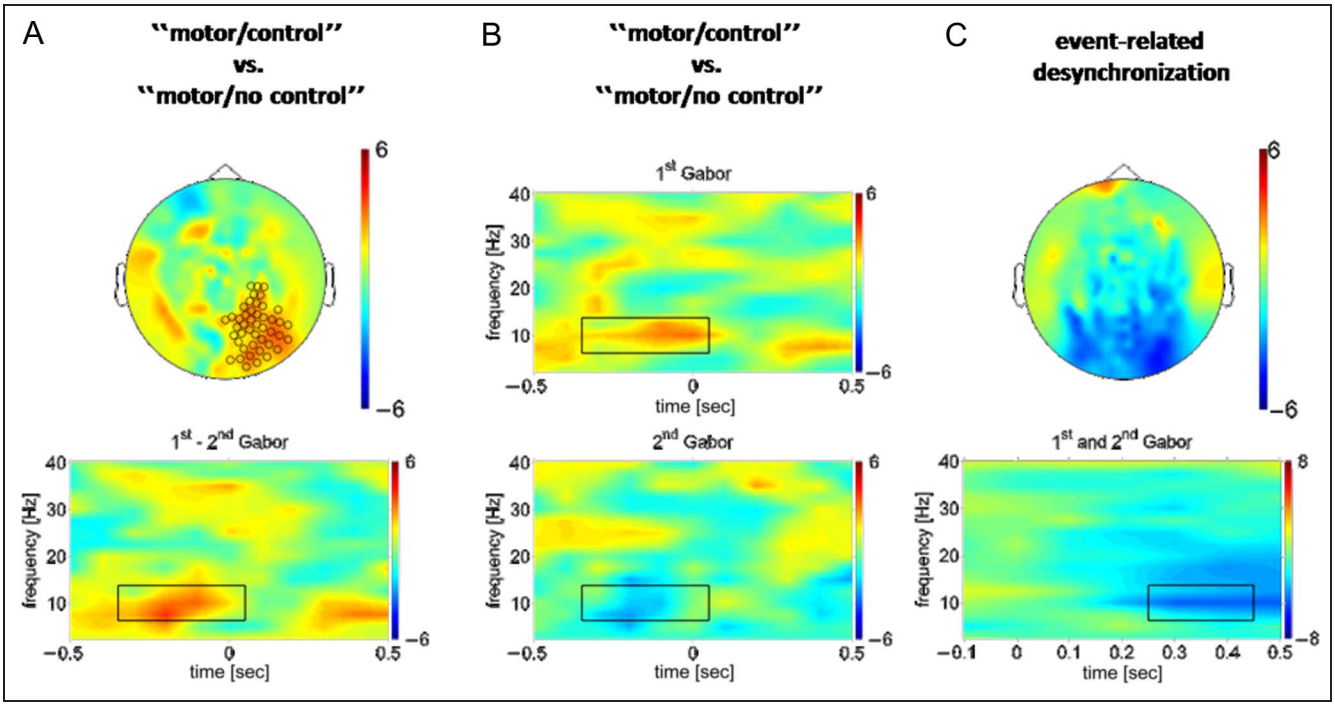

Figure 2. Effects of stimulus control and of Gabor patch presentation on peristimulus alpha-oscillations at the sensor level. Color codes for t values in all panels. (A) Sensor-level planar gradient topography (top) and time–frequency representation (bottom) of the effect of stimulus control ("motor/control" vs. "motor/no control") on the difference in alpha-power (7.5–12.5 Hz) between the first and the second Gabor patch (first − second). Markers in the sensor topography indicate channels that showed significantly higher alpha-power in the "motor/control" condition than in the "motor/no control" condition within the prestimulus time–frequency window depicted in the bottom panel (box). Data were averaged across these channels for the time–frequency representation. (B) Time–frequency representations of the effect of stimulus control ("motor/control" vs. "motor/no control") on the peristimulus low-frequency power spectrum, averaged across the channels highlighted in (A), top, around the onset of the first (top) and second (bottom) Gabor patch. (C) Topography and time–frequency representation of the effect of Gabor patch presentation on alpha-power (event-related alpha-desynchronization). The topography (top) is based on power averaged across the window depicted in the time–frequency representation (bottom), which in turn shows t values for power averaged across significant channels.

in this time and frequency window (smallest cluster-level p value: $p = .28$).

The topography of this prestimulus cluster in the alpha-band (Figure 2A, top) overlapped with the spatial distribution of the well-known stimulus-induced response in the same frequency band (Figure 2C, t values, poststimulus [300–400 msec] vs. baseline [−200 msec] across both Gabor patches for the "no motor" condition as a localizer). Note that the reconstructed planar gradient used here represents local cortical activity underneath corresponding sensors (Bastiaansen & Knösche, 2000). The prestimulus modulation of alpha-band power by stimulus control thus involved similar cortical regions as the early event-related alpha-desynchronization, which is one of the strongest neural signatures of afferent stimulation of sensory cortex (Pfurtscheller & Aranibar, 1977).

Source-level Analysis

We next determined the cortical source of the effect of stimulus control on prestimulus alpha-power and compared it to the topography of the stimulus-induced decrease in alpha-band power. As expected, stimulus control significantly modulated the source-level alpha-power difference between the first and second Gabor in a prestimulus time interval (around −200 msec). This prestimulus effect of stimulus control survived cluster-based whole-brain correction in a region including the calcarine sulcus and ventral occipital cortex of the right hemisphere (contralateral to the hemifield in which the Gabor patches were presented; Figure 3A, top: medial view, bottom: lateral view; $p = .016$, cluster-based correction for multiple comparisons across all cortical grid points).

When comparing the source of the effect of stimulus control on prestimulus alpha-power with the cortical topography of the event-related alpha-desynchronization, we found highly overlapping regions: Alpha-power was significantly suppressed by stimulus presentation in a region comprising the calcarine sulcus, parieto-occipital sulcus, cuneus, ventral occipital cortex, and middle and inferior occipital gyri of the right hemisphere (Figure 3B; poststimulus [300–400 msec] vs. baseline [−200 msec] across both Gabor patches for the "no motor" condition as a localizer, $p < .001$, cluster-based correction for multiple comparisons across all cortical grid points). Thus, stimulus control modulated prestimulus alpha-power in

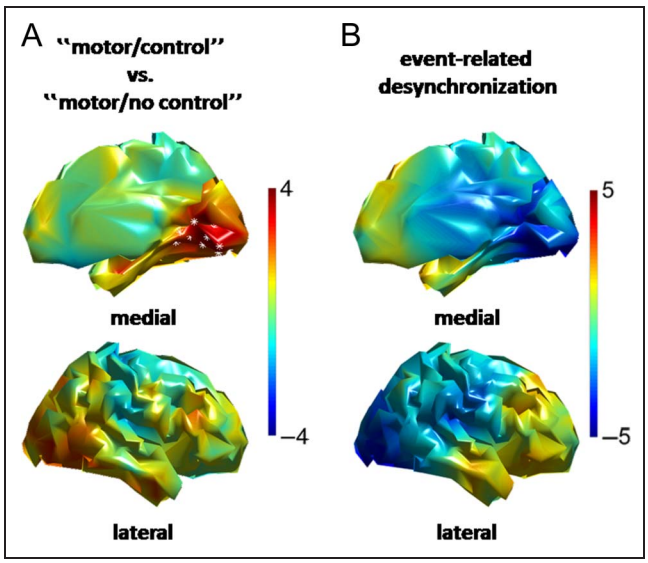

Figure 3. Main effects of stimulus control and of Gabor patch presentation on alpha-oscillations at the source level. Color codes for *t* values in all panels. (A) Right-hemisphere topography of the main effect of stimulus control on the prestimulus (−200 msec) difference in alpha-power (10 Hz) between the first and the second stimulus. White asterisks indicate significant grid points. Top, medial view; bottom, lateral view. (B) Right-hemisphere topography of the event-related alpha-desynchronization (300–400 msec poststimulus vs. baseline [−200 msec]). Top, medial view; bottom, lateral view.

regions that were subsequently engaged in stimulus-driven processing as reflected by alpha-desynchronization.

DISCUSSION

We demonstrate that causal control over both onset and identity of visual stimuli results in enhanced anticipatory alpha-power in visual cortex, an enhancement we link to a psychophysical measure of sensory attenuation. We suggest that this prestimulus effect of causal control on alpha-power reflects an inhibitory signal of motor-induced anticipation in visual cortex that reduces its excitability to afferent input, thereby leading to sensory attenuation.

Specificity to Stimulus Control

Our study was motivated by the fact that a majority of previous studies on sensory attenuation confound voluntary causal control over a sensory outcome with execution of a motor response and/or stimulus predictability (Hughes et al., 2012). These studies compare stimuli that are contingent on motor actions (typically labeled "motor-to-effect" conditions) to stimuli that are not preceded by motor responses (typically labeled "effect only" conditions). By comparing stimuli that were either controlled by a preceding action or independent of this action, we avoided this confound. Stimulus onset and orientation were determined by the timing and the choice of preceding button presses in the "motor/control" condition and independent of an irrelevant, but otherwise identical motor response in the "motor/no control" condition.

Hughes et al. (2012) recently emphasized the importance of dissociating the relative contributions of four components of causal control to sensory attenuation, namely the contributions of stimulus onset predictability, predictability of stimulus identity, control over stimulus onset and control over stimulus identity. Here, we manipulated causal control while keeping temporal and identity predictability comparable across conditions. Because of the cues at the beginning of each trial, the identity (orientation) of the stimulus was equally predictable across conditions. Stimulus onset was fully predictable by the timing of button presses in "motor/control" condition and became temporally predictable within the first few trials of each "motor/no control" block because of the fixed time lag between the auditory cue and stimulus onset. Together with temporal control, we manipulated the "outcome density" (Vallée-Tourangeau, Murphy, & Baker, 2005), namely the probability of stimulus presentation in the absence of action (in no-go trials).

It is difficult to control for both stimulus predictability and motor output at the same time. In particular, equal subjective predictability of the onset of a stimulus in "motor/control" and "motor/no control" conditions is challenging when the temporally predictive cue and the to-be-predicted stimulus are separated by a motor action that is irrelevant to stimulus timing. Despite any potential insufficiency of temporal cueing in "motor/no control" conditions in our study, our main physiological finding —enhanced anticipatory alpha-power in "motor/control" conditions—is the opposite of what the previous literature predicts with respect to low-frequency oscillations under conditions of increased temporal expectancy. Specifically, in analogy to the well-established anticipatory modulation of low-frequency oscillations in sensory cortex during spatial attention (e.g., Thut, Nietzel, Brandt, & Pascual-Leone, 2006), Rohenkohl and Nobre (2011) reported stronger occipital alpha-desynchronization when comparing conditions of high versus low temporal expectancy of visual stimuli. An equivalent result—temporal specificity of the typical prestimulus desynchronization of low-frequency oscillations during attention—has been reported in the somatosensory modality (Van Ede, de Lange, Jensen, & Maris, 2011). If temporal predictability and, thereby, temporal expectancy were indeed higher in the "motor/control" condition than in the "motor/no control" condition, we would have expected the opposite to what we found, namely decreased anticipatory alpha-power. Higher predictability and, as a result, greater expectancy of the stimulus in "motor/control" versus "motor/no control" conditions would therefore only strengthen our physiological finding of enhanced anticipatory alpha-power.

Bias as a Measure of Sensory Attenuation

We used bias in a contrast discrimination task as a measure of sensory attenuation, similar to many previous studies of sensory attenuation, which report shifts in the PSE in discrimination tasks (e.g., Desantis et al., 2012; Weiss et al., 2011; Haggard & Whitford, 2004). A change in bias (or PSE) due to sensory attenuation is in line with theoretical predictions of an influential computational model of motor control (Wolpert & Miall, 1996) that is often referred to explain sensory attenuation. This model predicts that a close match between predicted and observed action consequences results in signal cancellation (Blakemore, Wolpert, & Frith, 1998). Signal cancellation is distinct from a change of the sensory gain function, which has been considered an alternative mechanism underlying sensory attenuation (Brown, Adams, Parees, Edwards, & Friston, 2013). The effects of signal cancellation and of a change in gain on the two main signal detection theory measures, sensitivity and bias, depend on the task design. In a detection paradigm, both signal cancellation and gain reduction predict a decrease in sensitivity. However, in discrimination paradigms, like the one used here, mere cancellation of a sensory signal leads to a perceptual bias without change in sensitivity. In contrast, a change in sensory gain affects both signal amplitude and variance (signal-to-noise ratio) and, thereby, affects discriminability from the reference stimulus, that is, discrimination sensitivity. Indeed, absence of a sensitivity effect in our discrimination task speaks against any major confounding effect of our manipulation of stimulus control on attention. An attentional effect would typically alter the signal-to-noise ratio and, thereby, the sensory gain function (Hillyard, Vogel, & Luck, 1998).

In signal detection theory, bias has traditionally been regarded as a measure of decision-related rather than purely sensory processes. However, an interpretation of bias as a purely decisional parameter, indexing only late response stages, has been questioned on the grounds of recent evidence that bias ("criterion") can in fact reflect the baseline activity of signal-selective units at an early, sensory stage (Nobre, Summerfield, & Wyart, 2012). Our manipulation of stimulus control leads to a change of bias together with a physiologically plausible MEG signal in the relevant sensory cortex. This co-occurrence suggests that our behavioral result reflects a change in sensory processing rather than a purely decisional effect.

Sensory Attenuation and Anticipatory Alpha-oscillations

The original definition of sensory attenuation, based on the observation that detection of somatosensory stimuli is reduced when the stimulated limb is engaged in active movement (Chapman, Bushnell, Miron, Duncan, & Lund, 1987), has recently been extended to include other sensory modalities and abstract action–outcome associations in psychophysical and electrophysiological studies (Gentsch & Schütz-Bosbach, 2011; Weiss et al., 2011; Cardoso-Leite et al., 2010; Martikainen et al., 2005). The lack of data on neurophysiological mechanisms, particularly in humans, which may contribute to sensory attenuation, has meant that similarities in physiology and function of these sensory phenomena, as implied by the common term, have remained speculative.

A traditional theoretical framework used to explain sensory attenuation across sensory modalities and motor programs is based on motor-induced sensory predictions (Waszak et al., 2012; Schütz-Bosbach & Prinz, 2007; Wolpert et al., 1995). Although a number of studies have examined the functional anatomy of putative efference copy pathways upstream of primary sensory processing by focusing on cortical motor areas (Voss et al., 2009; Haggard & Whitford, 2004), the FEF (Ostendorf et al., 2012), or on the thalamus (Ostendorf, Liebermann, & Ploner, 2010; Bellebaum, Hoffmann, Koch, Schwarz, & Daum, 2006; Bellebaum, Daum, Koch, Schwarz, & Hoffmann, 2005), direct neural evidence of anticipatory sensory activity as a result of such motor-induced predictions is sparse (Chen et al., 2011; Kühn et al., 2010) and rarely studied in relation to sensory attenuation. We note that Chen et al. recently reported enhanced phase-locking in the gamma-frequency band between Broca's area and auditory cortex 50 msec before speech onset using ECoG and a paradigm that compared vocalizing and listening. This enhanced gamma-phase synchrony correlated interindividually with attenuation of the auditory N100 in response to self-produced speech when compared with replay (Chen et al., 2011).

Studying visual action consequences, we show a prestimulus effect of voluntary causation on neural oscillations in sensory cortex that differs from that reported by Chen et al. for speech. Of the two Gabor patches that followed the motor response in our task, the first was preceded by enhanced alpha-amplitude by up to 300 msec when its onset and orientation were fully determined by the action, whereas the amplitude of alpha-oscillations was slightly (and not significantly) reduced during the prestimulus interval of the second, temporally uncontrollable and later stimulus. We thus observe an anticipatory effect in sensory cortex that starts earlier than reported by Chen et al., affects low-frequency oscillations rather than gamma-frequencies, and depends on the temporal control of the stimulus by the action and/or the time lag between the two.

Occipital alpha-oscillations have been implicated in top–down control of visual perception (Von Stein, Chiang, & König, 2000) across a variety of cognitive tasks (Bauer, Kluge, et al., 2012; Jensen & Mazaheri, 2010). An inhibitory role of alpha-oscillations is widely accepted (Lange, Oostenveld, & Fries, 2013; Klimesch, Sauseng, & Hanslmayr, 2007) and has given rise to proposals of generic functions of sensory alpha-oscillations, namely gating (Jensen & Mazaheri, 2010) and prioritizing (Jensen et al., 2012) of

sensory processing. Prestimulus occipital alpha-oscillations have been causally linked to performance on visual detection tasks (Romei, Gross, & Thut, 2010; Van Dijk et al., 2008; Hanslmayr et al., 2007), with impaired target detection following higher alpha-amplitudes. In light of this inhibitory role of (prestimulus) alpha-oscillations in the top–down control of visual perception, our finding of alpha-amplitude enhancement before self-generated visual stimuli is a plausible correlate of motor-induced sensory anticipation such as corollary discharge. The fact that alpha-amplitude enhancement precedes both stimulus onset and motor execution does not speak against this proposal. The origin of efference copies has been linked to motor processing upstream of primary motor cortex in humans (Voss et al., 2009; Haggard & Whitford, 2004), and anticipatory sensory activity might conceivably precede the execution of the motor command.

To the best of our knowledge, this is the first study that establishes empirical links between a psychophysical measure of sensory attenuation and physiological evidence of anticipatory sensory modulation during action. Our findings extend on previous studies, which have shown changes in stimulus-evoked responses alone. We highlight frequency-specific effects of voluntary causal control, specifically on alpha-oscillations, a frequency band mechanistically linked to top–down inhibitory control of perception. Thus, our results link sensory attenuation, motor-induced predictions, and alpha-oscillations in sensory cortex.

Acknowledgments

This work was supported by the Wellcome Trust (Ray Dolan Senior Investigator Award 098362/Z/12/Z). M. P. S. is receiving a scholarship from the German Research Foundation (Deutsche Forschungsgemeinschaft DFG, STE 2091/1-1). The Wellcome Trust Centre for Neuroimaging was supported by core funding from the Wellcome Trust (091593/Z/10/Z). We thank David Bradbury for his help with data acquisition.

Reprint requests should be sent to Max-Philipp Stenner, Wellcome Trust Centre for Neuroimaging, 12 Queen Square, WC1N 3BG, UK, or via e-mail: max-philipp.stenner@med.ovgu.de.

Notes

1. Here, we used a weighted up/down staircase procedure (3 up, 1 down; Kaernbach, 1991) to adjust the difference in contrast of two sequentially presented Gabor patches. In four interleaved staircases, the contrast of the test stimulus approached the pedestal contrast (0.25 Michelson contrast) exponentially from above and below, separately for each of two Gabor patch orientations (45° clockwise or counterclockwise). The order of the test stimulus and the pedestal varied from trial to trial. Participants had to indicate whether the first or the second Gabor patch was the high-contrast stimulus. On a given trial, the contrast of the test stimulus was computed by $(0.25 \pm 0.1 \times 1.06^{\text{exponent}})$. Starting from zero, the exponent was varied by a 3 up, 1 down manipulation, that is, the exponent of the current staircase was decreased by 1 after a correct response and increased by 3 after an incorrect response. The staircase procedure was stopped after a total of 200 trials, and discrimination thresholds were determined as the average of the contrast levels of test stimuli at all reversals (from decreasing to increasing contrast differences and vice versa). The trial structure during the staircase procedure was identical to the "no motor" condition in the main experiment.
2. According to Cardoso-Leite et al. (2010), variations of action–stimulus mapping with respect to previous learning can modulate the degree of sensory attenuation. Following up on this observation, we trained participants predominantly on one of the two mappings during practice (index finger, 45° counterclockwise; middle finger, 45° clockwise) and, to avoid extinction, presented the predominantly learnt mapping slightly more often during the main task (in 62.5% of trials). However, since we found no significant perceptual effect of mapping, we collapsed all "motor/control" trials across both mappings (but note that we report control analyses that treat the two mappings separately).
3. The information carried by the visual cues in "motor/control" and "motor/no control" blocks was therefore different. In "motor/control" blocks, cues signaled the button/orientation mapping, whereas cues in "motor/no control" blocks predicted stimulus orientation independently of the following button choice. This difference in the information carried by the cues reflects the idea that sensory predictions that arise from motor actions are physiologically distinct from associative sensory predictions that arise from external cues (Hughes et al., 2012; Wolpert & Flanagan, 2001).
4. The button-Gabor orientation mapping had no significant effect in the "motor/control" and the "motor/no control" conditions as assessed by dependent samples t tests.
5. We observed highly overlapping clusters of right occipital and parietal sensors that showed significantly enhanced prestimulus (−300 to −100 msec) power between 7.5 and 12.5 Hz when testing "motor/control" trials with the two button stimulus orientation mappings separately against the "motor/no control" condition ($p = .008$ and $p = .024$; cluster-based correction for multiple comparisons across all channels).

REFERENCES

Alink, A., Schwiedrzik, C. M., Kohler, A., Singer, W., & Muckli, L. (2010). Stimulus predictability reduces responses in primary visual cortex. *The Journal of Neuroscience, 30,* 2960–2966.

Aliu, S. O., Houde, J. F., & Nagarajan, S. S. (2009). Motor-induced suppression of the auditory cortex. *Journal of Cognitive Neuroscience, 21,* 791–802.

Baess, P., Widmann, A., Roye, A., Schröger, E., & Jacobsen, T. (2009). Attenuated human auditory middle latency response and evoked 40-Hz response to self-initiated sounds. *The European Journal of Neuroscience, 29,* 1514–1521.

Bastiaansen, M. C., & Knösche, T. R. (2000). Tangential derivative mapping of axial MEG applied to event-related desynchronization research. *Clinical Neurophysiology, 111,* 1300–1305.

Bauer, M., Kennett, S., & Driver, J. (2012). Attentional selection of location and modality in vision and touch modulates low-frequency activity in associated sensory cortices. *Journal of Neurophysiology, 107,* 2342–2351.

Bauer, M., Kluge, C., Bach, D., Bradbury, D., Heinze, H. J., Dolan, R. J., et al. (2012). Cholinergic enhancement of visual attention and neural oscillations in the human brain. *Current Biology, 22,* 397–402.

Bays, P. M., Wolpert, D. M., & Flanagan, J. R. (2005). Perception of the consequences of self-action is temporally tuned and event driven. *Current Biology, 15,* 1125–1128.

Bellebaum, C., Daum, I., Koch, B., Schwarz, M., & Hoffmann, K.-P. (2005). The role of the human thalamus in processing corollary discharge. *Brain, 128,* 1139–1154.
Bellebaum, C., Hoffmann, K.-P., Koch, B., Schwarz, M., & Daum, I. (2006). Altered processing of corollary discharge in thalamic lesion patients. *The European Journal of Neuroscience, 24,* 2375–2388.
Blakemore, S. J., Wolpert, D. M., & Frith, C. D. (1998). Central cancellation of self-produced tickle sensation. *Nature Neuroscience, 1,* 635–640.
Brown, H., Adams, R. A., Parees, I., Edwards, M., & Friston, K. (2013). Active inference, sensory attenuation and illusions. *Cognitive Processing, 14,* 411–427.
Cardoso-Leite, P., Mamassian, P., Schütz-Bosbach, S., & Waszak, F. (2010). A new look at sensory attenuation. Action-effect anticipation affects sensitivity, not response bias. *Psychological Science, 21,* 1740–1745.
Chapman, C. E., Bushnell, M. C., Miron, D., Duncan, G. H., & Lund, J. P. (1987). Sensory perception during movement in man. *Experimental Brain Research, 86,* 516–524.
Chen, C.-M. A., Mathalon, D. H., Roach, B. J., Cavus, I., Spencer, D. D., & Ford, J. M. (2011). The corollary discharge in humans is related to synchronous neural oscillations. *Journal of Cognitive Neuroscience, 23,* 2892–2904.
Desantis, A., Weiss, C., Schütz-Bosbach, S., & Waszak, F. (2012). Believing and perceiving: Authorship belief modulates sensory attenuation. *PLoS One, 7,* e37959.
Gentsch, A., & Schütz-Bosbach, S. (2011). I did it: Unconscious expectation of sensory consequences modulates the experience of self-agency and its functional signature. *Journal of Cognitive Neuroscience, 23,* 3817–3828.
Haggard, P., & Whitford, B. (2004). Supplementary motor area provides an efferent signal for sensory suppression. *Brain Research. Cognitive Brain Research, 19,* 52–58.
Hanslmayr, S., Aslan, A., Staudigl, T., Klimesch, W., Herrmann, C. S., & Bäuml, K.-H. (2007). Prestimulus oscillations predict visual perception performance between and within subjects. *Neuroimage, 37,* 1465–1473.
Hesse, M. D., Nishitani, N., Fink, G. R., Jousmäki, V., & Hari, R. (2010). Attenuation of somatosensory responses to self-produced tactile stimulation. *Cerebral Cortex, 20,* 425–432.
Hillyard, S. A., Vogel, E. K., & Luck, S. J. (1998). Sensory gain control (amplification) as a mechanism of selective attention: Electrophysiological and neuroimaging evidence. *Philosophical Transactions of the Royal Society of London, Series B, Biological Sciences, 353,* 1257–1270.
Hughes, G., Desantis, A., & Waszak, F. (2012). Mechanisms of intentional binding and sensory attenuation: The role of temporal prediction, temporal control, identity prediction, and motor prediction. *Psychological Bulletin, 139,* 133–151.
Hughes, G., & Waszak, F. (2011). ERP correlates of action effect prediction and visual sensory attenuation in voluntary action. *Neuroimage, 56,* 1632–1640.
Jensen, O., Bonnefond, M., & VanRullen, R. (2012). An oscillatory mechanism for prioritizing salient unattended stimuli. *Trends in Cognitive Sciences, 16,* 200–206.
Jensen, O., & Mazaheri, A. (2010). Shaping functional architecture by oscillatory alpha activity: Gating by inhibition. *Frontiers in Human Neuroscience, 4,* 186.
Kaernbach, C. (1991). Simple adaptive testing with the weighted up-down method. *Perception & Psychophysics, 49,* 227–229.
Klimesch, W., Sauseng, P., & Hanslmayr, S. (2007). EEG alpha oscillations: The inhibition-timing hypothesis. *Brain Research Reviews, 53,* 63–88.
Kühn, S., Seurinck, R., Fias, W., & Waszak, F. (2010). The internal anticipation of sensory action effects: When action induces FFA and PPA activity. *Frontiers in Human Neuroscience, 4,* 54.
Lally, N., & Frendo, B. (2011). Sensory cancellation of self-movement facilitates visual motion detection. *Journal of Vision, 11,* 1–9.
Lange, J., Oostenveld, R., & Fries, P. (2013). Reduced occipital alpha power indexes enhanced excitability rather than improved visual perception. *The Journal of Neuroscience, 33,* 3212–3220.
Litvak, V., Mattout, J., Kiebel, S., Phillips, C., Henson, R., Kilner, J., et al. (2011). EEG and MEG data analysis in SPM8. *Computational Intelligence and Neuroscience, 2011,* 1–32.
Maris, E., & Oostenveld, R. (2007). Nonparametric statistical testing of EEG- and MEG-data. *Journal of Neuroscience Methods, 164,* 177–190.
Martikainen, M. H., Kaneko, K., & Hari, R. (2005). Suppressed responses to self-triggered sounds in the human auditory cortex. *Cerebral Cortex, 15,* 299–302.
Nobre, A. C., Summerfield, C., & Wyart, V. (2012). Dissociable prior influences of signal probability and relevance on visual contrast sensitivity. *Proceedings of the National Academy of Sciences, U.S.A., 109,* 6354.
Oostenveld, R., Fries, P., Maris, E., & Schoffelen, J.-M. (2011). FieldTrip: Open source software for advanced analysis of MEG, EEG, and invasive electrophysiological data. *Computational Intelligence and Neuroscience, 2011,* 3593–3598.
Ostendorf, F., Kilias, J., & Ploner, C. J. (2012). Theta-burst stimulation over human frontal cortex distorts perceptual stability across eye movements. *Cerebral Cortex, 22,* 800–810.
Ostendorf, F., Liebermann, D., & Ploner, C. J. (2010). Human thalamus contributes to perceptual stability across eye movements. *Proceedings of the National Academy of Sciences, U.S.A., 107,* 1229–1234.
Pfurtscheller, G., & Aranibar, A. (1977). Event-related cortical desynchronization detected by power measurements of scalp EEG. *Electroencephalography and Clinical Neurophysiology, 42,* 817–826.
Poulet, J. F. A., & Hedwig, B. (2007). New insights into corollary discharges mediated by identified neural pathways. *Trends in Neurosciences, 30,* 14–21.
Rohenkohl, G., & Nobre, A. C. (2011). Alpha oscillations related to anticipatory attention follow temporal expectations. *The Journal of Neuroscience, 31,* 14076–14084.
Romei, V., Gross, J., & Thut, G. (2010). On the role of prestimulus alpha rhythms over occipito-parietal areas in visual input regulation: Correlation or causation? *The Journal of Neuroscience, 30,* 8692–8697.
Schütz-Bosbach, S., & Prinz, W. (2007). Perceptual resonance: Action-induced modulation of perception. *Trends in Cognitive Sciences, 11,* 349–355.
Sommer, M. A., & Wurtz, R. H. (2006). Influence of the thalamus on spatial visual processing in frontal cortex. *Nature, 444,* 374–377.
Sperry, R. W. (1950). Neural basis of the spontaneous optokinetic response produced by visual inversion. *Journal of Comparative and Physiological Psychology, 43,* 482–489.
Thut, G., Nietzel, A., Brandt, S. A., & Pascual-Leone, A. (2006). Alpha-band electroencephalographic activity over occipital cortex indexes visuospatial attention bias and predicts visual target detection. *The Journal of Neuroscience, 26,* 9494–9502.
Vallée-Tourangeau, F., Murphy, R. A., & Baker, A. G. (2005). Contiguity and the outcome density bias in action–outcome contingency judgements. *The Quarterly Journal of*

Experimental Psychology: Series B, Comparative and Physiological Psychology, 58, 177–192.

Van Dijk, H., Schoffelen, J.-M., Oostenveld, R., & Jensen, O. (2008). Prestimulus oscillatory activity in the alpha band predicts visual discrimination ability. *The Journal of Neuroscience, 28,* 1816–1823.

Van Dijk, H., van der Werf, J., Mazaheri, A., Medendorp, W. P., & Jensen, O. (2010). Modulations in oscillatory activity with amplitude asymmetry can produce cognitively relevant event-related responses. *Proceedings of the National Academy of Sciences, U.S.A., 107,* 900–905.

Van Ede, F., de Lange, F., Jensen, O., & Maris, E. (2011). Orienting attention to an upcoming tactile event involves a spatially and temporally specific modulation of sensorimotor alpha- and beta-band oscillations. *The Journal of Neuroscience, 31,* 2016–2024.

Von Stein, A., Chiang, C., & König, P. (2000). Top–down processing mediated by interareal synchronization. *Proceedings of the National Academy of Sciences, U.S.A., 97,* 14748–14753.

Voss, M., Ingram, J. N., Haggard, P., & Wolpert, D. M. (2009). Sensorimotor attenuation by central motor command signals in the absence of movement. *Nature Neuroscience, 9,* 26–27.

Waszak, F., Cardoso-Leite, P., & Hughes, G. (2012). Action effect anticipation: Neurophysiological basis and functional consequences. *Neuroscience and Biobehavioral Reviews, 36,* 943–959.

Weiss, C., Herwig, A., & Schütz-Bosbach, S. (2011). The self in action effects: Selective attenuation of self-generated sounds. *Cognition, 121,* 207–218.

Wolpert, D. M., & Flanagan, J. R. (2001). Motor prediction. *Current Biology: CB, 11,* R729–R732.

Wolpert, D. M., Ghahramani, Z., & Jordan, M. I. (1995). An internal model for sensorimotor integration. *Science, 269,* 1880–1882.

Wolpert, D. M., & Miall, R. C. (1996). Forward models for physiological motor control. *Neural Networks, 9,* 1265–1279.