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Anticipation Increases Tactile Stimulus Processing in the Ipsilateral Primary Somatosensory Cortex

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Stimulus anticipation improves perception. To account for this improvement, we investigated how stimulus processing is altered by anticipation. In contrast to a large body of previous work, we employed a demanding perceptual task and investigated sensory responses that occur beyond early evoked activity in contralateral primary sensory areas: Stimulus-induced modulations of neural oscillations. For this, we recorded magnetoencephalography in 19 humans while they performed a cued tactile identification task involving the identification of either a proximal or a distal stimulation on the fingertips. We varied the cue–target interval between 0 and 1000 ms such that tactile targets occurred at various degrees of anticipation. This allowed us to investigate the influence of anticipation on stimulus processing in a parametric fashion. We observed that anticipation increases the stimulus-induced response (suppression of beta-band oscillations) originating from the ipsilateral primary somatosensory cortex. This occurs in the period in which the tactile memory trace is analyzed and is correlated with the anticipation-induced improvement in tactile perception. We propose that this ipsilateral response indicates distributed processing across bilateral primary sensory cortices, of which the extent increases with anticipation. This constitutes a new and potentially important mechanism contributing to perception and its improvement following anticipation.

Keywords: attentional orienting, distributed sensory processing, magnetoencephalography, sensorimotor beta-oscillations, sensory memory maintenance, spatial attention

Introduction

Anticipating a stimulus improves its perception (Posner et al. 1980; Carrasco 2011). What are the neurophysiological mechanisms underlying this improvement? To answer this question, both pre- and poststimulus neural activities must be considered. Concerning prestimulus activity, it is now well established that anticipation of a behaviorally relevant stimulus involves a modulation of neural activity in the stimulus-receiving contralateral sensory cortex, as indexed by preparatory increases in the spike rate (e.g. Luck et al. 1997) and regional blood flow (e.g. Kastner et al. 1999) and decreases in neuronal oscillations in the alpha- and beta-bands (e.g. Thut et al. 2006; van Ede et al. 2011). These phenomena are associated with improved perception (e.g. Ress et al. 2000; Thut et al. 2006; Jones et al. 2010; van Ede, Köster, et al. 2012) and are therefore relevant for understanding how anticipation improves perception.

The improvement in perception by anticipatory processes must occur through altered sensory processing of the anticipated sensory information. To explain perceptual improvement by anticipation, it is thus important to also consider this poststimulus sensory processing phase. However, despite

a large body of prior investigations (e.g. Mangun and Hillyard 1991; Miniussi et al. 1999), this literature has been limited by 3 important aspects. First, this literature has focused almost exclusively on evoked neural activity reflecting early processing stages in brain areas contralateral to sensory input. Secondly, this has typically been investigated in the context of simple perceptual tasks such as detection. Particularly in the context of more demanding perceptual tasks, such as identification, relevant sensory processing is likely not confined to these early and contralateral processing stages. Therefore, to obtain a comprehensive understanding of perception and its improvement with anticipation, it is essential to also investigate sensory processing beyond these early stages. Finally, the relation between neural modulations and perceptual improvement has often remained unaddressed.

Here, we investigated how anticipation alters neural activity beyond early processing stages by employing a demanding perceptual identification task and by focusing on stimulus-induced modulations of oscillatory neural activity. This type of sensory response typically persists up to a second after a transient stimulus and occurs in both contra- and ipsilateral sensory cortices (e.g. Chatrian et al. 1959; Cheyne et al. 2003; see also Results). Crucially, because this type of response does not require precise locking in time of the underlying events, it is also sensitive to perceptually relevant cognitive processes whose onset times likely vary between trials.

One particular process that is important in more demanding perceptual tasks is the online maintenance of relevant sensory information. Such maintenance allows further processing of the sensory information after it has physically disappeared. Interestingly, recent literature suggests that primary sensory cortices are important for such maintenance. For example, visual information kept in working memory can be decoded from patterns of functional magnetic resonance imaging activity in the primary visual cortex (V1; Harrison and Tong 2009; Serences et al. 2009; Sneve et al. 2012). Likewise, cueing of visual (Sergent et al. 2011) and somatosensory (Spitzer and Blankenburg 2011) stimuli after their disappearance modulates, respectively, V1 and primary somatosensory cortex (S1) activities. Moreover, this type of process is also reflected in induced modulations of oscillatory neural activity (Spitzer and Blankenburg 2011).

Because previous studies on anticipation have focused mainly on early sensory processing stages in simple perceptual tasks, it has remained unclear whether and how the above-sketched memory-dependent processing stage (i.e. the process of maintaining and/or manipulating information in sensory memory after stimulus disappearance) is affected by anticipation. To address this, we employed a demanding tactile identification task in which brief (20 ms) tactile stimuli required substantial further processing (reaction times were in

the order of 1 s)—thus relying on memory-dependent perceptual processing. As a manipulation of anticipation, we presented these stimuli at variable intervals after a symbolic attentional cue. Because anticipatory processes build up over time, this manipulation of the cue–target interval allowed us to investigate stimulus processing as a function of degree of anticipation.

In our data, anticipation modulates an induced response (i.e., suppression of beta-band oscillations) that occurs in the period in which the sensory memory trace is analyzed: 300–600 ms poststimulus. Moreover, this modulation is correlated with the anticipation-induced improvement in tactile accuracy. Strikingly, however, this response originates from the ipsilateral S1. We propose that primary sensory cortex is involved in identifying the fine detail of a sensory memory trace, and that this can occur even when the sensory information is not received via direct afferent pathways. The increased ipsilateral response might reflect an increase in the distribution of sensory processing across bilateral primary sensory cortices, which may contribute to the perceptual improvement following stimulus anticipation.

Materials and Methods

Materials and methods of this experiment were reported previously (van Ede, de Lange, et al. 2012). We here reiterate those elements that are essential for understanding the results of the present study and describe in more detail those methods that are specific to the currently presented analyses.

Participants

Nineteen humans (13 males, age: $M=28$, $SD=6$) participated in the experiment. Two participants were excluded from the analyses because of chance-level performance. The experiment was conducted in accordance with guidelines of the local ethical committee (Committee on Research Involving Human Subjects, Region Arnhem-Nijmegen, The Netherlands).

Design, Task, and Procedure

Participants performed a cued somatosensory identification task. Figure 1 depicts the trial sequence. In each trial, a binaural auditory cue (25 ms duration) indicated with 75% validity on which side (i.e. which hand) the to-be-identified tactile target would occur. The side was indicated by the type of auditory stimulus (white noise or 750 Hz pure tone) and was counterbalanced across participants. Tactile targets consisted of a stimulation of either the upper (distal) or the lower (proximal) part of all fingertips of a single hand. Targets were delivered using a custom-built Braille device housing 5 Braille cells (Metec,

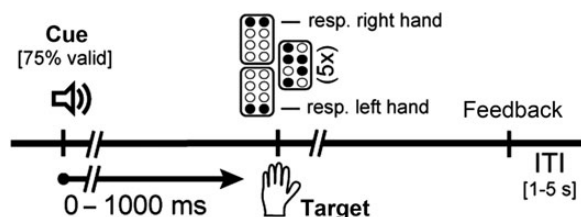


Figure 1. Task. 75% valid symbolic auditory cue indicates whether a tactile stimulus will occur on the left or the right hand. Between 0 and 1000 ms after this cue, the tactile target stimulus is presented to the upper or lower part of all fingertips (using the upper or lower pins of the Braille cells; see black dots) of either hand. This is followed by 5 masks without spatial structure. Participant’s task is to identify the target (upper or lower) and to respond with the right (upper target) or the left (lower target) hand. Following a response, participants receive feedback. The intertrial interval (ITI) is drawn from a truncated negative exponential distribution with values between 1 and 5 s.

Stuttgart, Germany) that can be individually adjusted. Before starting, each fingertip was positioned over one such Braille cell. For upper (lower) targets, we transiently raised the upper (lower) 2 Braille pins for each fingertip (Fig. 1). For both hands, tactile stimulation by the upper (lower) pins required a right (left) hand button press. Because of this, (anticipated) target side was uncorrelated with the required response side. More specifically, left- and right-hand button presses were required equally often for targets on either hand. This independence between target and response sides was also reflected in the behavioral responses: Side-congruent responses (same target- and response sides) did not occur more frequently ($t_{(16)}=0.314$; $P=0.758$) and were not faster ($t_{(16)}=0.777$; $P=0.449$) than side-incongruent responses. This implies that neural activity that lateralized according to the (anticipated) target side cannot be due to response preparation and/or execution. To increase difficulty, targets were followed, on the same hand, by 5 masks that contained no spatial structure. Target and masks together lasted 270 ms (20 ms stimulations, 50 ms interstimulus intervals). Responses were self-paced. Following responses, tactile feedback was presented after 300 ms. A correct (incorrect) response was followed by a single (double) 20-ms tap to both hands. The interval between feedback and the next stimulus was drawn from a truncated negative exponential distribution (range: 1–5 s). Because this distribution has a nearly flat hazard rate, the onset of the next cue could not be predicted on the basis of elapsed time since the last cue.

The crucial manipulation in this study is the manipulation of the degree of anticipation. This was realized by varying the interval between the auditory cue and the tactile target. Per trial, this interval was randomly drawn from a uniform distribution with values between 0 and 1000 ms.

Before recording, subjects received instructions and practiced the task for about 10 min. In 2 recording sessions of approximately 1 h, we collected about 1500 trials. Each session contained between 9, 10, or 11 blocks (depending on the subject’s motivation) of 75 trials. Left and right cued trials were randomly intermixed. Intervals between blocks were self-paced by the participants.

Recording and Extraction of Neural Data of Interest

Recordings and analyses of neural data were highly similar to previous reports from our lab (e.g. van Ede et al. 2011). Data were collected using a 275 axial gradiometers magnetoencephalography (MEG) system (CTF MEG TM Systems, Inc., Port Coquitlam, Canada) and analyzed using FieldTrip (Oostenveld et al. 2011). From the axial gradiometer signal, we calculated the planar gradient (Bastiaansen and Knosche 2000), which is maximal above the neuronal sources.

Per participant, 10 channels above both left and right S1 were selected after contrasting all left- and right-hand stimulations with respect to beta-band (13–30 Hz) amplitude in the 150- to 400-ms post-target window. Because beta-band amplitude is more suppressed across contralateral than ipsilateral S1 in this time window (e.g. Chatrian et al. 1959; Cheyne et al. 2003; van Ede et al. 2010; van Ede et al. 2011), the contrast (left- minus right-hand stimulation) results in negative values for right S1 and positive values for left S1. We thus selected the 10 most negative and 10 most positive channels to represent the right and left S1, respectively. Note that this channel selection is independent of the main analysis (involving the correlation between neural activity and cue–target interval), because it is based on all target stimuli, independent of the cue–target interval.

Oscillatory amplitudes were estimated using Fourier analysis with and without time- and/or frequency resolution. Estimates with frequency resolution were based on a Hanning taper, while estimates without frequency resolution were based on the multitaper method (Percival and Walden 1993). The multitaper method allows for the estimation of a spectral band (i.e. 13–30 Hz; the beta-band). For analyses with time resolution, a 250-ms sliding time window was used that was advanced in 12.5 ms steps.

Investigating Neural Activity As a Function of Degree of Anticipation

The manipulation of the cue–target interval allowed us to investigate target processing as a function of the degree of anticipation. We did

this separately for validly and invalidly cued targets. For our main analysis, we sorted trials into 4 cue–target interval bins (targets following the cue within [0–250], [250–500], [500–750], or [750–1000] ms). After sorting the trials in this way, we used linear regression analyses to reveal which spatial, temporal, and spectral aspects of the data varied with degree of anticipation. Because anticipation increases roughly linearly over the first second after a cue (i.e. across the 4 cue–target interval bins; van Ede, de Lange, et al. 2012), this type of analysis is well suited to reveal which aspects of stimulus processing depend on anticipation.

We initially focused on the target-induced “lateralization” as an index for target processing. We did this for 2 reasons. First, it reduces the spatial dimension to a single value. Secondly, it is unaffected by sensory processing of the auditory cue (which was presented binaurally) as well as motor preparation or execution. With respect to the latter, this holds because motor preparation and execution do not lateralize according to the target side; left- and right-hand targets required as often a left- as a right-hand button press.

We first describe how we quantified the relation between the cue–target interval and the target-induced lateralization indices (steps 1–2; Fig. 2A). Thereafter, we describe how we statistically evaluated this relation (steps 3–5; Fig. 2B). Together, this analysis involved 5 steps that are also depicted in Figure 2. In step 1, we separated trials by their cue–target interval (placing them into 1 of the 4 consecutive cue–target interval bins), and for each bin we calculated the normalized difference in amplitude between contra- and ipsilateral tactile stimuli ($(\text{contra} - \text{ipsi}) / (\text{contra} + \text{ipsi})$). We did this in a time- and frequency-resolved manner. Importantly, these lateralization indices were calculated on target-centered data (with time 0 defining target onset). This analysis thus resulted in 4 sets of target-induced modulations, with the only difference between the sets being the interval between the preceding cue and the target (see Fig. 2A for these data of a representative participant). To evaluate where in time- and frequency neural activities depended on cue–target interval, we estimated the linear regression coefficient describing the relation between the modulation indices and the cue–target interval (step 2). We did this separately for each time–frequency point.

Because we did not have an hypothesis about where in time and frequency neural activities might vary with degree of anticipation, we used a statistical test that was time- and frequency uninformed. More precisely, we blindly scanned the full time–frequency space for a statistically significant dependence of amplitude lateralization on the cue–target interval. For this, we used a cluster-based permutation test (Maris and Oostenveld 2007). Importantly, this statistical analysis controls the false-alarm rate when facing multiple comparisons, as in our case, where the correlation with the cue–target interval is evaluated for multiple time and frequency samples. The details of this analysis are described and depicted (Fig. 2B) in steps 3–5.

In step 3, we obtained our cluster statistics of interest by (1) evaluating, per time–frequency point, the regression coefficients from step 2 at the group level using a 1-sample *t*-test, (2) clustering the *t*-values of neighboring time and frequency samples in case these exceeded the threshold corresponding to a univariate *t*-test at the 0.05 level (2-tailed), and (3) summing the *t*-values per cluster (called “cluster-level statistics”). Using a permutation approach, we then evaluated these cluster-level statistics against the null hypothesis of statistical independence between cue–target interval and oscillatory amplitude lateralization (steps 4 and 5). In step 4, the order of the 4 cue–target interval bins was permuted (at the single-subject level), and steps 2 and 3 were repeated. This was done 1000 times. The idea here is that, by randomly permuting the order of the cue–target interval bins, all systematic variations with the cue–target interval will be removed. In other words, all clusters observed after random permutation must be due to chance. The distribution that is obtained by randomly permuting the order of the cue–target interval bins (each time keeping the maximum cluster-level statistic) is a distribution under the null hypothesis of statistical independence between cue–target interval and oscillatory amplitude lateralization. In step 5, we assessed the significance of the time–frequency clusters observed in the original data by evaluating them under the permutation distribution of the maximum cluster statistic ($\alpha = 0.05$, 2-tailed). Because this involves a single

distribution by means of which the full time–frequency space is evaluated, this analysis bypasses the multiple comparison problem.

Significant clusters were used as masks for the time and frequency-resolved plots of the correlation (Pearson’s *r*) between oscillatory amplitude lateralization and cue–target interval (Fig. 3A,C). Thus, the masked clusters in Figure 3A,C represent clustered time–frequency samples of which the lateralization index scales linearly with cue–target interval, more so then can be explained by chance. For masking, we applied an opacity mask ($\alpha = 0.25$ in Matlab) to all nonsignificant time–frequency points.

Our statistical analysis revealed a highly significant cluster at 300–600 ms posttarget, in the 12- to 28-Hz band (Fig. 3A). Having established this statistically significant cluster of interest (in a time- and frequency-uninformed way), we further investigated this effect in 3 ways. First, we mapped its spatial topography. For every channel, we calculated the correlation between the degree of anticipation and the data in this time–frequency window. This was done separately for the left- and right-hand targets (Fig. 3A, topographies) that were followed by left- and right-hand responses (Fig. 4). Secondly, we separately mapped contralateral and ipsilateral time-resolved 12–28 Hz amplitude for stimuli occurring after short (0–250 ms), middle (375–625 ms), or long (750–1000 ms) intervals after the cue. Time was expressed relative to target onset. Amplitude was expressed as a percentage change from a –1500 to –1125 ms pretarget (and therefore pre-cue) baseline (Fig. 3B,D). Note that these 2 analyses were done solely for descriptive purposes. Thirdly, we investigated the correlation across participants between this neural effect and tactile identification accuracy (Fig. 5). We did this as a function of cue–target interval. For this, we analyzed both variables (proportion correct responses and ipsilateral beta-amplitude 300–600 ms posttarget) with time resolution using a 250-ms sliding time window that was advanced in 12.5 ms steps across cue–target intervals. For normalization purposes, we expressed these variables as a percentage change from the average of 2 neutral conditions (target without, or simultaneously with a cue).

Results

We employed a cued somatosensory identification task in which tactile stimuli were delivered to either the left or the right hand. In this task, stimuli required substantial further processing: On average, participants required 931 ± 77 ms (mean ± 1 SEM) to obtain an accuracy level of $70 \pm 1.5\%$ correct.

Because we had presented stimuli between 0 and 1000 ms after a symbolic attentional cue, and because anticipatory processes build up over the first second after a cue (see van Ede, de Lange, et al. 2012), we could use linear regression analysis to reveal what aspects of stimulus processing varied systematically with the degree of anticipation. For this, we separated 4 sets of trials based on their cue–target intervals (i.e. stimuli occurring at [0–250], [250–500], [500–750], and [750–1000] ms after the cue; see also Materials and Methods and Fig. 2), and evaluated the strength of the linear relation across these 4 sets.

Because we did not have an hypothesis about where in time and frequency neural activities might depend on anticipation, we scanned the full time–frequency space using a cluster-based permutation approach (Maris and Oostenveld 2007; see also Materials and Methods and Fig. 2). Outcomes of this approach (i.e. the region in time–frequency space in which neural activity varied significantly across the 4 consecutive cue–target interval bins) are depicted in Figure 3.

Anticipatory Processes Build up Over Time

Before describing the influence of anticipation on posttarget processing, which is the main objective of this study, we briefly highlight the neural processes involved in the anticipat-

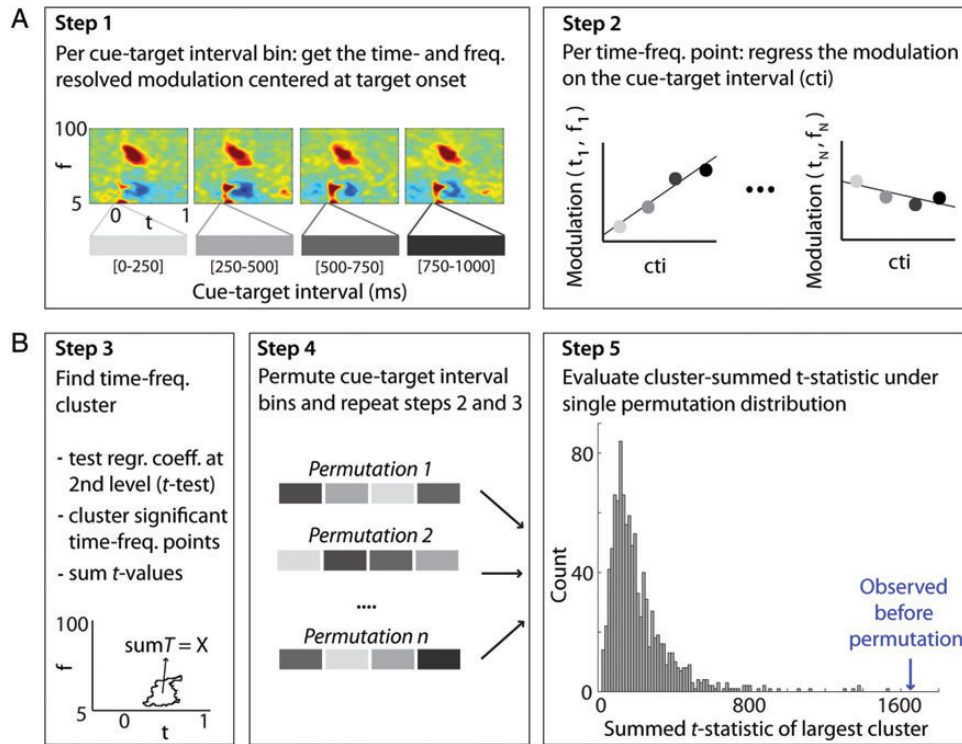


Figure 2. Schematic of our analysis: Evaluation of where in time and frequency neural activities depend on the degree of anticipation. (A) Quantification of where in time and frequency neural activities depend on the degree of anticipation (operationalized by the cue–target interval). We binned trials into 4 sets based on their cue–target interval and for each set calculated the time- and frequency-resolved lateralized modulation indices that were centered on target onset (step 1). Note that the time axis for these modulation indices (with time 0 corresponding to target onset) is different from that of the cue–target interval (where 0 corresponds to a target that occurred 0 ms after the cue). The figure shows that these modulation indices for each of the 4 consecutive cue–target interval bins for a representative participant. In step 2, we evaluated where in time and frequency neural activities depended on the cue–target interval by estimating the linear regression coefficient describing the relation between the modulation indices and the cue–target interval. We did this separately for each time–frequency point. (B) Statistical evaluation. Because there are as many regression coefficients as there are time–frequency points, we face a large multiple comparisons problem. To deal with this problem, we used a cluster-based permutation approach (Maris and Oostenveld 2007), of which the essential steps are depicted in steps 3–5. In step 3, we obtained our cluster statistic of interest by (1) evaluating, for each time–frequency point, the regression coefficient from step 2 at the second level (i.e. across participants), (2) clustering together neighboring time–frequency points that exceeded the statistical threshold, and (3) summing their t -values. In step 4, we permuted cue–target interval bins (at the single-subject level) and repeated steps 2 and 3. This was repeated 1000 times. Each time we only kept the maximum cluster statistic. In step 5, the summed t -statistics of the clusters observed before permutation were evaluated under the permutation distribution of these maximum cluster statistics obtained in step 4. The histogram in the figure depicts the positive tail of the actual permutation distribution obtained for valid trials and includes the cluster statistic of the largest positive cluster obtained before permutation (i.e. the posttarget beta-band cluster between 300 and 600 ms; also depicted in step 3; see also Fig. 3A). Because none of the cluster-summed t -statistics of the permuted data exceeded that of the original data, the pattern in the original data is unlikely to have resulted by chance ($P < 0.001$ in this 1-tailed test).

ory period itself. For a more elaborate coverage of these results, we refer the reader to our previous study that is based on the data from the same experiment. In that study, we specifically focused on the relation between these anticipatory signals and the perceptual improvement with attentional cueing (van Ede, de Lange, et al. 2012).

First, we consider data locked to validly cued stimuli (i.e. when the tactile target occurred on the hand that was indicated by the cue). Figure 3A shows the time- and frequency-resolved linear correlation between the cue–target interval (as an operationalization of the degree of anticipation) and oscillatory amplitude lateralization (S1 contralateral minus ipsilateral to the target), masked by statistical significance (see Materials and Methods, as well as Fig. 2 for the rationale behind this analysis).

Before target onset, there is a strong negative correlation between cue–target interval and alpha- and beta-band amplitude lateralization (cluster- $P < 0.001$). This reflects a well-established anticipatory phenomenon that develops within the first second after the cue (e.g. Worden et al. 2000; Thut et al. 2006; Jones et al. 2010; van Ede et al. 2011; van Ede, de Lange,

et al. 2012) and is constituted by a stronger contralateral suppression, at least in our data (van Ede et al. 2011; van Ede, de Lange, et al. 2012; van Ede, Köster, et al., 2012). This is also visible from the blue lines in Figure 3B depicting ipsi- and contralateral baseline-corrected beta-amplitude for trials in which the cue preceded the target between 750 and 1000 ms. Strikingly, in contrast to previous observations (e.g. Haegens et al. 2012), we did not observe an increase in the amplitude ipsilateral to the anticipated target. In fact, in ipsilateral channels, beta-amplitude also decreased during anticipation. While an elaborate discussion of this observation is beyond the scope of the current study, we would like to point to 2 possible explanations for this apparent discrepancy. First, in our experiment, no distracters were anticipated on the uncued hand, an issue that is further discussed in van Ede et al. (2011). Secondly, a potential increase in ipsilateral amplitude might be overshadowed by a potential bilateral effect of motor preparation. Importantly, note that the possibility of such bilateral motor preparation would not be a confound for our main results, because these are based on lateralized modulations (see Materials and Methods for details).

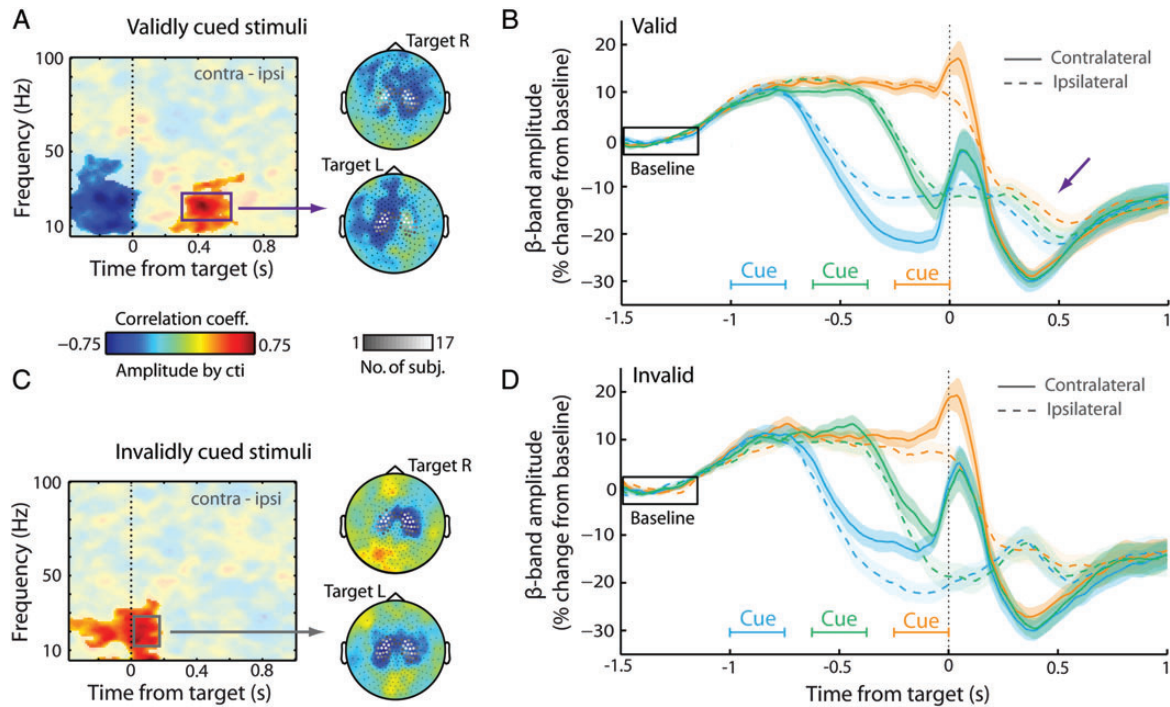


Figure 3. Anticipation increases the ipsilateral beta-band response to a unilateral tactile target. (A) Left: time- and frequency-resolved correlation between cue-target interval (cti) and oscillatory amplitude lateralization (contralateral minus ipsilateral S1), masked by significant clusters. Right: topographies of the correlation for the indicated cluster, separately for the left- and right-hand targets. Highlighted channels represent selected channels above left and right S1 (see Materials and Methods for the selection procedure). Channel colors show the number of subjects for which a given channel was selected (we adopted this strategy from Siegel et al. 2007). (B) Time-resolved contralateral and ipsilateral beta-band amplitudes plotted for stimuli that occurred at short (0–250 ms; orange), middle (375–625 ms; green), or long (750–1000 ms; blue) intervals after the symbolic cue. Color patches represent ± 1 SEM. Purple arrow indicates the phenomenon of interest. (C and D) Same as A and B, for invalidly cued stimuli.

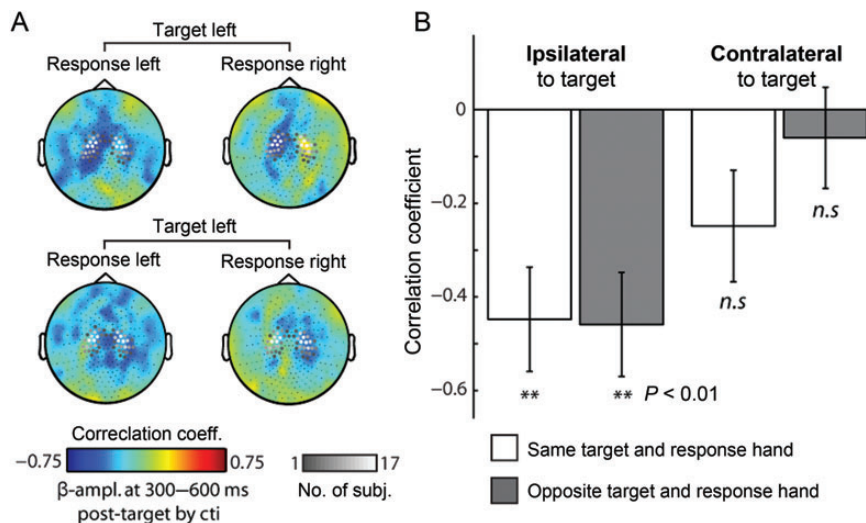


Figure 4. The anticipation-dependent beta-band response occurs ipsilateral to the target, irrespective of the side of the motor response. (A) Topographies of the correlation between the cue-target interval (cti) and beta-band amplitude at 300–600 ms posttarget, separated by target and response hand. Highlighted channels represent selected channels above left and right S1 (see Materials and Methods for the selection procedure). Channel colors show the number of subjects for which a given channel was selected (we adopted this strategy from Siegel et al. 2007). (B) Bar chart showing the average correlation coefficient between beta-amplitude at 300–600 ms posttarget and cue-target interval separately for the selected channels above ipsi- and contralateral S1 relative to the target side. Data were separated for trials in which the response (a button press with the thumb) was made on the same or the opposite side as the target. Error bars represent ± 1 SEM.

Interestingly, on validly cued trials (Fig. 3A), at target onset ($t=0$), this correlation vanishes, implying a discontinuation of this anticipatory brain state. In contrast, on invalidly cued trials (Fig. 3C,D), the anticipatory brain state (now represented by a positive cluster, because the subject prepares

for the opposite hand; cluster- $P < 0.001$) does continue beyond target onset. This occurs contralateral to the expected target (thus ipsilateral to the actual target; see topographies) and likely reflects the fact that it requires approximately 200 ms to reorient attention.

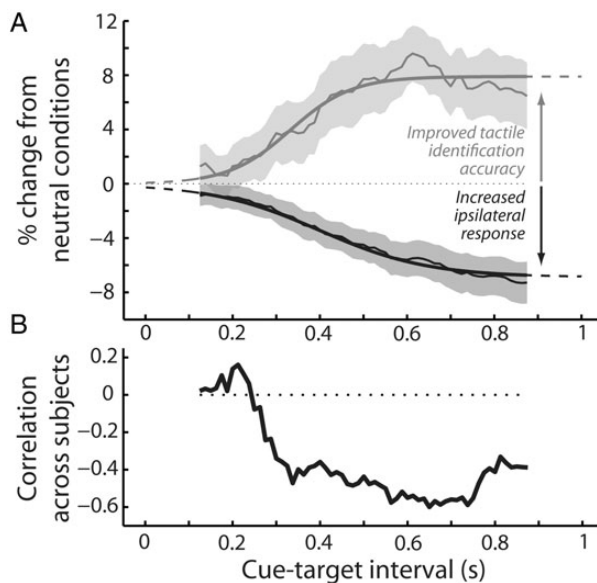


Figure 5. The increased ipsilateral response is associated with the anticipation-induced improvement in tactile perception. (A) Upper panel: Tactile identification accuracy (gray) and ipsilateral beta-band amplitude at 300–600 ms posttarget (black), as a function of the cue–target interval. Data are expressed relative to the average of 2 neutral conditions (target without, or simultaneously with a cue). Colored patches represent ± 1 SEM. (B) Correlation across subjects between the variables in the upper panel. Only data from validly cued trials are shown.

These results thus confirm that anticipation builds up over the first second after the cue (as we had previously reported: see van Ede, de Lange, et al. 2012). As such, these results validate the use of the cue–target interval as an operationalization of the degree of anticipation. In the following, we will use this fact to investigate what aspects of target processing vary with this degree of anticipation, which is the main objective of this study.

Anticipation Increases the Ipsilateral Beta-Band Response to a Unilateral Tactile Target

In addition to the anticipatory cluster discussed above, we also observed a strong correlation between the degree of anticipation and neural activity at 300–600 ms posttarget in the low frequencies, with a peak in the classical beta-band (13–30 Hz; Fig. 3A; cluster- $P < 0.005$). To evaluate whether this correlation originated from the ipsi- or the contralateral hemisphere, we correlated the cue–target interval and oscillatory beta-band amplitude in the 300- to 600-ms interval separately for left- and right-hand tactile targets (Fig. 3A, topographies). Clearly, beta-band amplitude in this time window correlated negatively with the cue–target interval over the sensorimotor cortex ipsilateral to the stimulated hand. Thus, a higher degree of anticipation is associated with lower amplitude in channels above the sensorimotor cortex ipsilateral to the stimulated hand.

This observation is further explored by zooming in on the time-resolved beta-band amplitude for stimuli occurring at 3 different degrees of anticipation (Fig. 3B): After short (0–250 ms; orange), middle (375–625 ms; green), or long (750–1000 ms; blue) cue–target intervals (note that the amount of bins is arbitrary, and that we depict 3 bins solely for visualization purposes). Ignoring the amplitude increase around target onset (which is a consequence of the evoked response), there

are 2 clear target-induced responses. First, unilateral tactile stimulation strongly suppresses contralateral beta-band oscillations between 150 and 400 ms posttarget. The strength of this suppression is independent of the degree of anticipation (i.e. identical for the 3 traces) and can therefore be considered a “mandatory” response. Secondly, unilateral tactile stimulation also suppresses ipsilateral beta-band oscillations (see also Chatrian et al. 1959; Cheyne et al. 2003). This occurs around 300–600 ms posttarget, slightly delayed relative to the contralateral response. Crucially, in contrast to the contralateral response, this ipsilateral response does depend on the degree of anticipation: A higher degree is associated with a stronger suppression (Fig. 3B, purple arrow). Anticipation thus increases the ipsilateral hemisphere’s response to a unilateral tactile target. This is a robust effect, because it is this effect that was revealed as a significant cluster ($P < 0.005$) by our time- and frequency-uninformed statistical analysis.

We did not observe any posttarget consequence of anticipation in invalidly cued trials (Fig. 3C,D). Importantly, this cannot be explained by the lower number of invalid than valid trials: Using this number of valid trials, we observed qualitatively the same results as in Figure 3A.

Importantly, the anticipation-dependent ipsilateral response in valid trials cannot be simply explained by a continuation of the anticipatory brain state. In fact, if this were the case, then the strongest effect should be observed in invalid trials directly following target onset, because in this window the anticipatory suppression is larger in the ipsi- than the contralateral hemisphere (Fig. 3D). Instead, the effect only occurs in valid trials and is initiated only at the time when the ipsilateral response is initiated, which is 300 ms posttarget (see also Chatrian et al. 1959; Cheyne et al. 2003). Thus, rather than a passive consequence of the prestimulus state, the effect involves the modulation of an existing stimulus-induced response.

The Increased Ipsilateral Response Is Related to Perception

It is important to rule out motor preparation and/or execution as possible explanations for the anticipation-dependent ipsilateral response. In this respect, we must first note that, in our experiment, target and response sides were uncorrelated (see Materials and Methods). Because our main statistical analysis was based on neural lateralization relative to the side of the target, the effect cannot be due to response preparation and/or execution. We could also show this empirically. We calculated, for all channels, the correlation between cue–target interval and beta-amplitude at 300–600 ms posttarget. We did this separately for trials separated by target and by response side. As can be seen in Figure 4A, the negative correlation with the cue–target interval remains ipsilateral to the target side, irrespective of the subsequent response side. For example, following a left tactile target, this correlation occurs in channels above the left (ipsilateral) sensorimotor cortex, irrespective of whether the subsequent response followed with the left or right hand. This pattern is also evident from Figure 4B depicting these correlations for selected channels above ipsi- and contralateral S1 (see panel A for the location of these channels and Materials and Methods for details of our selection procedure). Thus, the correlation occurs ipsilateral to the target, irrespective of whether the subsequent response was given with the same or the opposite hand. These observations, combined

with the fact that the response is clearly lateralized and observed in channels above the primary sensorimotor cortex, suggest that the modulations originates from ipsilateral S1.

The Increased Ipsilateral Response Is Associated with Perceptual Improvement

Our data also provide evidence for the perceptual relevance of this posttarget ipsilateral modulation. Figure 5A shows the dependence of this response on the cue–target interval. Strikingly, the ipsilateral response depends on the cue–target interval in much the same way as the tactile identification accuracy (Fig. 5A). For example, after a cue, both the ipsilateral response and tactile identification accuracy increase between 250 and 600 ms. Note that, as we showed previously (van Ede, de Lange, et al. 2012), the effect of anticipation on reaction time follows a different time course.

More direct evidence for the perceptual relevance of the increased ipsilateral response is shown by its correlation with the improvement in perceptual accuracy. For each cue–target interval bin depicted in Figure 5A, we correlated the anticipation-induced change in ipsilateral response with that in tactile identification accuracy across our 17 participants (change was calculated from the average of 2 baseline conditions; see Materials and Methods). Strikingly, participants with a stronger increase in the ipsilateral response also benefit more from anticipation in terms of perceptual accuracy (for data averaged across all cue–target intervals: $r = -0.499$, $P = 0.042$). Following the cue, this correlation occurs from 250 ms onwards (Fig. 5B), which is consistent with the time it takes before anticipation affects neural responses and behavior (van Ede, de Lange, et al. 2012). Thus, when anticipatory processes come into play, the more they increase the ipsilateral response, the larger the corresponding improvement in perceptual accuracy.

Discussion

We investigated how anticipation affects stimulus processing and how this improves perception. We observed that anticipation of a unilateral tactile stimulus increases the ipsilateral S1 response (suppression of beta-band oscillations at 300–600 ms poststimulus) to this stimulus, and that this increased response is associated with the improvement in perception. Three implications stand out. First, anticipation also affects relevant sensory processing beyond early, contralateral stages. Secondly, ipsilateral S1 likely plays a more active role in tactile perception than is commonly believed. Thirdly, our data suggest a new mechanism contributing to perception and its improvement with anticipation: Distributed sensory processing across bilateral sensory cortices. In the following, we will discuss these points in more detail.

Anticipation and Late Sensory Processing Stages

In the context of demanding perceptual tasks, behaviorally relevant sensory processing is likely not confined to the early and contralateral processing stages on which most previous investigations have focused. Specifically, previous studies mainly focused on evoked responses, reflecting processes that are precisely locked in time (e.g. neural activity propagating through fixed feedforward and feedback anatomical connections). In sensory cortex, such responses typically occur within the first

200- to 300-ms poststimulus. In our tactile identification task, subjects required on average 931 ms to identify the target. This indicates that the memory trace of the transiently presented target was analyzed for at least 600–700 ms before a response was planned. An important question thus becomes whether such late-stage stimulus processing is also influenced by anticipation, and if so, in which brain regions? Scanning spatial, temporal, and spectral dimensions of oscillatory neural activity, we observe that anticipation can also affect sensory processing of 300–600 ms poststimulus (see Wyart and Tallon-Baudry 2008) for another example of such a “late effect” in the visual modality). Moreover, to our surprise, this occurred in the primary sensory cortex ipsilateral to the stimulus.

The involvement of the primary sensory cortex in later stages of sensory processing fits well with the recent literature. In fact, a number of studies provide evidence for the active role of primary sensory cortices in the maintenance of sensory information after stimulus disappearance (Harrison and Tong 2009; Serences et al. 2009; Sergent et al. 2011; Sneve et al. 2012). In a demanding perceptual task like ours, in which target stimuli are only presented briefly, such maintenance likely plays a central role in perception. Interestingly, in a recent electrophysiological study investigating such sensory maintenance, Spitzer and Blankenburg (2011) also observed a suppression of induced neural oscillations in S1. Here, we show that this type of response is modulated by anticipation. Considering the ipsilateral nature of this modulation in our data, it is interesting to note that the utilization of the primary sensory cortex for sensory maintenance need not be restricted to the sensory cortex in which sensory information was initially received. Indeed, even perceptual imagery condition without sensory input can engage primary sensory cortices (Kosslyn et al. 1995). Possibly via the same mechanism, the nonrecipient ipsilateral primary sensory cortex might also be utilized for perception. To corroborate this interpretation, experiments are required in which both anticipation and sensory maintenance are explicitly manipulated. For example, if the stimulus must be maintained for several seconds, would the anticipation-dependent ipsilateral response maintain over this period as well?

In the following, we first review other available evidence, suggesting that ipsilateral S1 can contribute to the perception of unilateral tactile stimuli. We then further elaborate on possible mechanisms via which this increased ipsilateral response might mediate perceptual improvement.

Ipsilateral S1 and Tactile Perception

We suggested above that ipsilateral primary sensory cortex might be utilized for perception via purely top-down pathways (i.e. pathways that are also employed by perceptual imagery). In line with this, computational modeling has suggested that S1 beta oscillations might depend critically on inputs to the supragranular (feedback receiving) layers of cortex (Jones et al. 2009). In addition, the utilization of ipsilateral S1 might proceed via bottom-up pathways existing within the somatosensory system. In this system, contralateral S1 is the primary cortical recipient of tactile information. This likely explains the stronger as well as earlier poststimulus beta-band response over contralateral (when compared with ipsilateral) S1 (Fig. 3B,D). At the same time, there is evidence suggesting that ipsilateral S1 also gains access to some of this information.

Ipsilateral responses to unilateral tactile stimuli have been observed not only in beta-band oscillations (Chatrian et al. 1959; Cheyne et al. 2003; current study, Fig. 3B,D), but also in regional cerebral blood flow (Hlushchuk and Hari 2006; Lipton et al. 2006; Tommerdahl et al. 2006), dendritic current flow (Lipton et al. 2006), and action potentials (Iwamura et al. 1994; Wiest et al. 2005; Lipton et al. 2006). However, it is currently not clear whether these different types of responses are related to the same underlying principles. For example, while some studies point to inhibition of ipsilateral processing (Hlushchuk and Hari 2006; Lipton et al. 2006; but see Wiest et al. 2005), our data most likely reflect the opposite. This is because inhibition has been associated with an increase in beta-band amplitude (Jensen et al. 2005; Pogosyan et al. 2009). One important difference between our study and the aforementioned ones is that our tactile stimulus required substantial further processing.

Ipsilateral S1 responses likely involve pathways that pass via contralateral S1, in particular Brodmann area 2 (BA2). In BA2, Iwamura et al. (1994) observed numerous cells with bilateral receptive fields for hands and digits. Ablation and inactivation of contralateral S1 abolished bilateral receptive fields, implying that the ipsilateral responses are transmitted via contralateral S1. One likely pathway involves a callosal connection between left and right BA2 (Iwamura et al. 1994). Alternatively, contralateral S1 might project to ipsilateral S1 indirectly via secondary somatosensory cortex (S2) or the thalamus (Blankenburg et al. 2008). Noteworthy, a pathway between left and right S1 has behavioral advantages, because tactile information is often coregistered between both S1 cortices, as a result of bimanual exploration of tactile objects. Because this feature might be central to both somatosensation and action, so might be the ability to distribute processing across the hemispheres. Indeed, analogous to our observations in the tactile domain, ipsilateral primary motor cortex has been implicated in motor processing (Crone et al. 1998; Donchin et al. 1998; Mehring et al. 2003).

Several lines of evidence suggest that these responses may also contribute to perception. First, processing of sensory information in contralateral S1 is modulated by ipsilateral sensory stimulation (Schnitzler et al. 1995; Wiest et al. 2005). This points to the integration of sensory information between bilateral S1 and, thus, argues for a functional role of ipsilateral S1 in perception. On a behavioral level, Harris et al. (2001) showed that the memory trace of a unilateral tactile stimulus is maintained with specificity for the finger on which the to-be-remembered stimulus occurred. Crucially, this specificity of the tactile memory trace also applied to the corresponding fingers on the other hand. Because this finger specificity implies working memory maintenance within somatotopically organized brain regions (S1 and/or S2), the intermanual transfer implies maintenance in ipsilateral S1 and/or S2. Moreover, similar to the present report (Fig. 5B), such working memory performance has been reported to correlate positively with the degree of ipsilateral beta-band suppression in the retention interval (Li Hegner et al. 2007). These observations are thus consistent with a scenario in which the improvement in tactile accuracy following anticipation is mediated by an increased ipsilateral response, as reported in this paper. We next turn to possible mechanisms via which the increased ipsilateral response might mediate the corresponding improvement in perception.

Improved Perception Through Distributed Processing

Ample evidence exists that oscillatory neural activity is related to information processing in underlying neural populations. In fact, these oscillations interact with local spiking activity (e.g. Fries et al. 2001; Haegens et al. 2011). Directly relevant to our observations, the suppression of beta-band oscillations in sensorimotor cortex has been associated with an increase in cortical excitability (Pogosyan et al. 2009; Maki and Ilmoniemi 2010) as well as an improvement in tactile detection performance (Palva et al. 2005; Jones et al. 2010; van Ede, Köster, et al. 2012). Because both contra- and ipsilateral S1 show this type of response, we propose that the increase in the ipsilateral response with anticipation reflects an increase in the distribution of sensory processing across contra- and ipsilateral S1.

It is commonly believed that anticipation improves perception through selective focusing of attention, involving amplification of relevant signals and/or suppression of irrelevant signals (Carrasco 2011). Our task also involved selective attention: Subjects anticipated a stimulus on one of their hands, allowing selective spatial attention. Therefore, our results point to an additional mechanism via which this type of attention might improve perception: The extent to which sensory processing is distributed across contra- and ipsilateral sensory cortices. In line with this hypothesis, we show that the degree to which the ipsilateral response increases (in essence, the degree to which the response becomes more bilateral or distributed) predicts the amount of perceptual improvement with spatial attention.

What type of sensory processing might benefit such distribution across the hemispheres within the context of our tactile identification task? An important notion is that performance in this task relies on the matching of the incoming sensory stimulus to memory templates of the possible targets. In our set-up, these targets differed in a fine spatial aspect (stimulation of the upper versus the lower part of the fingertips), and therefore only S1 might have the appropriate neural circuitry for storing their templates and matching the incoming sensory information. Moreover, throughout our experiment, identical targets occurred on both the left and the right hands, and therefore these templates might have been formed within both the left and right S1. In this scenario, optimal matching of the incoming sensory information might involve both contra- and ipsilateral templates. Thus, the increased ipsilateral S1 response might reflect an increase in the extent to which the unilaterally presented sensory information is maintained and matched to the ipsilaterally stored template (in addition to the contralateral one). Alternatively, such template matching might occur downstream of S1. In this case, functional roles of the ipsilateral S1 response might involve: (1) Solely holding online the sensory memory trace as long as up- or downstream areas need to read this out, or (2) elaborate processing of the sensory information preceding such read out.

Why is the influence of anticipation only visible in the ipsi-, but not in the contralateral beta-band response? We can only speculate about this unexpected result. An important observation is that the stronger contralateral suppression of beta-band oscillations reaches a plateau, independently of the degree of anticipation (Fig. 3B). This plateau might reflect saturation in the processing capacity of contralateral S1, which might be the reason for utilizing ipsilateral S1 for additional processing. Because a higher degree of anticipation involves

more strongly suppressed contralateral beta-band oscillations prior to the target, the additional contralateral suppression following the target is more easily accomplished when the subject is more prepared (compare blue and orange solid lines, Fig. 3B) and this may allow additional engagement of ipsilateral S1.

We have put forward the hypothesis that ipsilateral S1 assists in the processing of the tactile memory trace, and that anticipation increases this process to improve perception. However, contrary to this hypothesis, previous investigations into the neural correlates of tactile working memory maintenance did not reveal involvement of S1 (reviewed in Romo and Salinas 2003). At least 3 factors might explain this discrepancy. First, the work by Romo and Salinas (2003) focused on local neural spiking activity, while we focused on a large-scale population aggregate of postsynaptic potentials. Indeed, this latter type of signal has been associated with tactile working memory operations (Spitzer and Blankenburg 2011). Secondly, in addition to online maintenance, our identification task required manipulation of the sensory memory trace (e.g. matching of the sensory memory trace to the stored templates). These additional operations may engage additional neural populations. Thirdly, in contrast to our human subjects, the monkeys in the experiments by Romo and colleagues were highly overtrained. This is relevant because neural dynamics can change with practice (e.g. Wan et al. 2011). Despite these arguments, it is important to keep in mind that our results do not necessarily imply that there are also anticipation-dependent modulations of the spiking activity in ipsilateral S1. For example, work by Das and colleagues (Sirotnin and Das 2009; Cardoso et al. 2012) has revealed anticipation-related modulations in hemodynamic signals that are poorly related to local spiking activity. It is currently not clear to what extent such a dissociation may also hold for the beta-band oscillations in the MEG, the neural signal studied here. For example, these oscillations might reflect rhythmic fluctuations of subthreshold dendritic currents (Jones et al. 2009). Thus, at present, we cannot exclude that such modulations of spiking activity occur elsewhere, such as in contralateral S1 or bilateral S2, and produce synchronized subthreshold activity in connected cortical areas (i.e. ipsilateral S1). The ipsilateral response might thus reflect the consequence of stimulus processing that takes place elsewhere. However, this account would predict that the influence of anticipation should also be observed in channels above contralateral S1/bilateral S2. This was not the case (Fig. 3A,B).

Conclusion

We conclude that anticipation can also affect memory-dependent perceptual processing in the primary sensory cortex, and that ipsilateral S1 likely plays a more active role in tactile perception than is commonly thought. Increasing the extent to which sensory processing is distributed across bilateral primary sensory cortices might constitute an important mechanism contributing to the improvement in perception following stimulus anticipation.

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Notes

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References

- Bastiaansen MC, Knosche TR. 2000. Tangential derivative mapping of axial MEG applied to event-related desynchronization research. *Clin Neurophysiol.* 111:1300–1305.
- Blankenburg F, Ruff CC, Bestmann S, Bjoertomt O, Eshel N, Josephs O, Weiskopf N, Driver J. 2008. Interhemispheric effect of parietal TMS on somatosensory response confirmed directly with concurrent TMS-fMRI. *J Neurosci.* 28:13202–13208.
- Cardoso MM, Sirotnin YB, Lima B, Glushenkova E, Das A. 2012. The neuroimaging signal is a linear sum of neurally distinct stimulus- and task-related components. *Nat Neurosci.* 15:1298–1306.
- Carrasco M. 2011. Visual attention: the past 25 years. *Vision Res.* 51:1484–1525.
- Chatrian GE, Petersen MC, Lazarte JA. 1959. The blocking of the rolandic wicket rhythm and some central changes related to movement. *Electroencephalogr Clin Neurophysiol.* 11:497–510.
- Cheyne D, Gaetz W, Garnero L, Lachaux JP, Ducorps A, Schwartz D, Varela FJ. 2003. Neuromagnetic imaging of cortical oscillations accompanying tactile stimulation. *Brain Res.* 17:599–611.
- Crone NE, Miglioretti DL, Gordon B, Sieracki JM, Wilson MT, Uematsu S, Lesser RP. 1998. Functional mapping of human sensorimotor cortex with electrocorticographic spectral analysis. I. Alpha and beta event-related desynchronization. *Brain.* 121:2271–2299.
- Donchin O, Gribova A, Steinberg O, Bergman H, Vaadia E. 1998. Primary motor cortex is involved in bimanual coordination. *Nature.* 395:274–278.
- Fries P, Reynolds JH, Rorie AE, Desimone R. 2001. Modulation of oscillatory neuronal synchronization by selective visual attention. *Science.* 291:1560–1563.
- Haegens S, Luther L, Jensen O. 2012. Somatosensory anticipatory alpha activity increases to suppress distracting input. *J Cogn Neurosci.* 24:677–685.
- Haegens S, Nacher V, Luna R, Romo R, Jensen O. 2011. Alpha-oscillations in the monkey sensorimotor network influence discrimination performance by rhythmic inhibition of neuronal spiking. *Proc Natl Acad Sci USA.* 108:19377–19382.
- Harris JA, Harris IM, Diamond ME. 2001. The topography of tactile working memory. *J Neurosci.* 21:8262–8269.
- Harrison SA, Tong F. 2009. Decoding reveals the contents of visual working memory in early visual areas. *Nature.* 458:632–635.
- Hlushchuk Y, Hari R. 2006. Transient suppression of ipsilateral primary somatosensory cortex during tactile finger stimulation. *J Neurosci.* 26:5819–5824.
- Iwamura Y, Iriki A, Tanaka M. 1994. Bilateral hand representation in the postcentral somatosensory cortex. *Nature.* 369:554–556.
- Jensen O, Goel P, Kopell N, Pohja M, Hari R, Ermentrout B. 2005. On the human sensorimotor-cortex beta rhythm: sources and modeling. *NeuroImage.* 26:347–355.
- Jones SR, Kerr CE, Wan Q, Pritchett DL, Hamalainen M, Moore CI. 2010. Cued spatial attention drives functionally relevant modulation of the mu rhythm in primary somatosensory cortex. *J Neurosci.* 30:13760–13765.
- Jones SR, Pritchett DL, Sikora MA, Stufflebeam SM, Hamalainen M, Moore CI. 2009. Quantitative analysis and biophysically realistic neural modeling of the MEG mu rhythm: rhythmogenesis and modulation of sensory-evoked responses. *J Neurophysiol.* 102:3554–3572.
- Kastner S, Pinsk MA, De Weerd P, Desimone R, Ungerleider LG. 1999. Increased activity in human visual cortex during directed attention in the absence of visual stimulation. *Neuron.* 22:751–761.
- Kosslyn SM, Thompson WL, Kim IJ, Alpert NM. 1995. Topographical representations of mental images in primary visual cortex. *Nature.* 378:496–498.
- Li Hegner Y, Lutzenberger W, Leiberg S, Braun C. 2007. The involvement of ipsilateral temporoparietal cortex in tactile pattern working

- memory as reflected in beta event-related desynchronization. *NeuroImage*. 37:1362–1370.
- Lipton ML, Fu KM, Branch CA, Schroeder CE. 2006. Ipsilateral hand input to area 3b revealed by converging hemodynamic and electrophysiological analyses in macaque monkeys. *J Neurosci*. 26:180–185.
- Luck SJ, Chelazzi L, Hillyard SA, Desimone R. 1997. Neural mechanisms of spatial selective attention in areas V1, V2, and V4 of macaque visual cortex. *J Neurophysiol*. 77:24–42.
- Maki H, Ilmoniemi RJ. 2010. EEG oscillations and magnetically evoked motor potentials reflect motor system excitability in overlapping neuronal populations. *Clin Neurophysiol*. 121:492–501.
- Mangun GR, Hillyard SA. 1991. Modulations of sensory-evoked brain potentials indicate changes in perceptual processing during visual-spatial priming. *J Exp Psychol Hum Percept Perform*. 17:1057–1074.
- Maris E, Oostenveld R. 2007. Nonparametric statistical testing of EEG- and MEG-data. *J Neurosci Methods*. 164:177–190.
- Mehring C, Rickert J, Vaadia E, Cardoso de Oliveira S, Aertsen A, Rotter S. 2003. Inference of hand movements from local field potentials in monkey motor cortex. *Nat Neurosci*. 6:1253–1254.
- Miniussi C, Wilding EL, Coull JT, Nobre AC. 1999. Orienting attention in time. Modulation of brain potentials. *Brain*. 122:1507–1518.
- Oostenveld R, Fries P, Maris E, Schoffelen JM. 2011. FieldTrip: open source software for advanced analysis of MEG, EEG, and invasive electrophysiological data. *Comput Intell Neurosci*. 2011:156869.
- Palva S, Linkenkaer-Hansen K, Naatanen R, Palva JM. 2005. Early neural correlates of conscious somatosensory perception. *J Neurosci*. 25:5248–5258.
- Percival DB, Walden AT. 1993. Spectral analysis for physical applications: multitaper and conventional univariate techniques. Cambridge: Cambridge University Press.
- Pogosyan A, Gaynor LD, Eusebio A, Brown P. 2009. Boosting cortical activity at beta-band frequencies slows movement in humans. *Curr Biol*. 19:1637–1641.
- Posner MI, Snyder CR, Davidson BJ. 1980. Attention and the detection of signals. *J Exp Psychol*. 109:160–174.
- Ress D, Backus BT, Heeger DJ. 2000. Activity in primary visual cortex predicts performance in a visual detection task. *Nat Neurosci*. 3:940–945.
- Romo R, Salinas E. 2003. Flutter discrimination: neural codes, perception, memory and decision making. *Nat Rev*. 4:203–218.
- Schnitzler A, Salmelin R, Salenius S, Jousmaki V, Hari R. 1995. Tactile information from the human hand reaches the ipsilateral primary somatosensory cortex. *Neurosci Lett*. 200:25–28.
- Serences JT, Ester EF, Vogel EK, Awh E. 2009. Stimulus-specific delay activity in human primary visual cortex. *Psychol Sci*. 20:207–214.
- Sergent C, Ruff CC, Barbot A, Driver J, Rees G. 2011. Top-down modulation of human early visual cortex after stimulus offset supports successful postcued report. *J Cogn Neurosci*. 23:1921–1934.
- Siegel M, Donner TH, Oostenveld R, Fries P, Engel AK. 2007. High-frequency activity in human visual cortex is modulated by visual motion strength. *Cereb Cortex*. 17:732–741.
- Sirotin YB, Das A. 2009. Anticipatory haemodynamic signals in sensory cortex not predicted by local neuronal activity. *Nature*. 457:475–479.
- Sneve MH, Alnaes D, Endestad T, Greenlee MW, Magnussen S. 2012. Visual short-term memory: activity supporting encoding and maintenance in retinotopic visual cortex. *NeuroImage*. 63:166–178.
- Spitzer B, Blankenburg F. 2011. Stimulus-dependent EEG activity reflects internal updating of tactile working memory in humans. *Proc Natl Acad Sci USA*. 108:8444–8449.
- Thut G, Nietzel A, Brandt SA, Pascual-Leone A. 2006. Alpha-band electroencephalographic activity over occipital cortex indexes visuospatial attention bias and predicts visual target detection. *J Neurosci*. 26:9494–9502.
- Tommerdahl M, Simons SB, Chiu JS, Favorov O, Whitsel BL. 2006. Ipsilateral input modifies the primary somatosensory cortex response to contralateral skin flutter. *J Neurosci*. 26:5970–5977.
- 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. *J Neurosci*. 31:2016–2024.
- van Ede F, de Lange FP, Maris E. 2012. Attentional cues affect accuracy and reaction time via different cognitive and neural processes. *J Neurosci*. 32:10408–10412.
- van Ede F, Jensen O, Maris E. 2010. Tactile expectation modulates pre-stimulus beta-band oscillations in human sensorimotor cortex. *NeuroImage*. 51:867–876.
- van Ede F, Köster M, Maris E. 2012. Beyond establishing involvement: quantifying the contribution of anticipatory alpha- and beta-band suppression to perceptual improvement with attention. *J Neurophysiol*. 108:2352–2362.
- Wan Q, Kerr C, Pritchett D, Hamalainen M, Moore C, Jones S. 2011. Dynamics of dynamics within a single data acquisition session: variation in neocortical alpha oscillations in human MEG. *PLoS One*. 6:e24941.
- Wiest MC, Bentley N, Nicoletti MA. 2005. Heterogeneous integration of bilateral whisker signals by neurons in primary somatosensory cortex of awake rats. *J Neurophysiol*. 93:2966–2973.
- Worden MS, Foxe JJ, Wang N, Simpson GV. 2000. Anticipatory biasing of visuospatial attention indexed by retinotopically specific α -band electroencephalography increases over occipital cortex. *J Neurosci*. 20:RC63:1–6.
- Wyart V, Tallon-Baudry C. 2008. Neural dissociation between visual awareness and spatial attention. *J Neurosci*. 28:2667–2679.