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Localized high gamma motor oscillations respond to perceived biological motion

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Abstract

Power changes in the beta frequency range (17–25 Hz) in the human motor and premotor areas during action observation have been associated with the mirror neuron system (MNS) and have been studied extensively. These changes mimic motor activity during actual motion execution, albeit reduced in strength. Recent non-invasive (EEG/MEG) and invasive studies (ECoG) have shown that during actual motion, beta power changes are accompanied by highly localized changes in the high gamma (HG) band (70–100 Hz). In this study we investigate, using 27 channel EEG in combination with a generic head model and a cortical mapping algorithm, whether such high gamma changes are also present during motion observation. Subjects were presented with a 2.7 s video of a moving hand, contrasted with a video of moving scenery of equal length. Our results show non-lateralized beta band decrease in power in response to the moving hand vs. the response to the moving scenery. We also find significant increase in high gamma power. However, unlike the beta band response, increases in this band are lateralized, with a preference for the hemisphere of the dominant hand.

Keywords

EEG; high gamma; motor system; motion observation

Introduction

Observation of biological motion has been widely interpreted as invoking the mirror neuron system (MNS) and there has been considerable interest in the phenomenon. The existence of the MNS in humans is controversial (Dinstein et al., 2008) and its proposed association with cognitive functions such as action understanding is not well established (Hickok, 2009). Nevertheless, research using a range of neuroimaging methods demonstrates the existence of a cortical execution and observation matching system related to action and perception. A large number of non-invasive studies of cortical activity have been carried out using action observation paradigms in varying modalities, i.e. functional magnetic resonance imaging (fMRI) (Kilner et al., 2009b), magnetoencephalography (MEG) (Hari et al., 1998, Nishitani and Hari, 2000) and electroencephalography (EEG) (Babiloni et al., 2002). Due to the high temporal resolution of MEG/EEG, these modalities allow to study not only the spatial, but also the temporal aspects of the MNS response on a millisecond scale. Specifically induced power changes in the mu/alpha (9–12 Hz) and beta (17–25 Hz) frequency band have been

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studied in the past (Babiloni et al., 2002, Hari et al., 1998) and have been found to be similar, albeit reduced in strength, to changes during movement execution. Invasive studies of movement execution, using electrocorticography (ECoG) (Crone et al., 1998, Miller et al., 2007), have shown that in addition to power changes in the lower frequency bands, i.e. mu/alpha and beta, there is also a high frequency increase in power at frequencies ranging from 71 – 100 Hz, i.e. the high gamma (HG) band. Other studies (Ball et al., 2008, Cheyne et al., 2008, Darvas et al., 2010) have shown that such HG power increases can also be detected non-invasively with MEG and EEG. Given the similarity between cortical activity during biological motion observation and actual motion execution, we hypothesize that there is, in addition to the well-studied lower frequencies, also a HG component.

In the present study, we demonstrate the existence of a lateralized HG increase during biological motion observation as compared to non-biological motion observation in healthy adult subjects.

Materials and methods

Subjects

Data were recorded from ten healthy adult subjects, eight subjects were right handed, two left handed (8 male, 2 female, age range 23–56, mean 34). Subjects gave their informed consent according to the protocol approved by the internal review board (IRB) of the University of Washington.

Task

During data acquisition, subjects were presented alternatively with a video of a moving hand (see still image sequence, Figure 1, top row), where the thumb is sequentially touching the other fingers or moving scenery (Figure 1, bottom row). Subjects were instructed to passively observe the video playback. The stimulus presentation was initiated with a beep and a fixation cross in the center of a blank screen. After a 2 s pre-stimulus period, the video was played back at 30 frames per second (fps), synchronized with the data acquisition system. A total of 81 frames, equal to 2.7s duration, were displayed. Between videos, there was a random interval of 3 – 5 s. Videos of the hand (i.e. the *movement* condition) and the scenery (i.e. *contrast* condition), were played back in random order. The scenery as contrast provides a natural moving object which is sufficiently far removed from the human anatomy and would not have an immediate association with human motion. The whole experiment consisted of 4 sessions, each with 25 presentations of movement and contrast for a total of 100 trials per movement and contrast. There were short breaks between each session.

Recording

Data was recorded from 27 electrodes, using an extended 10–20 system, where 8 additional electrode positions over motor and frontal areas were used. Ground was located on the midline position between CZ and FZ. Data was sampled from DC to 1200 Hz with an anti aliasing filter at 600 Hz, using two GugerTec (GugerTec, Graz, Austria) EEG amplifiers. We used a 3D localizer (Patriot, Polhemus, Colchester, VT) to determine the electrode positions for each subject as well as the positions of three anatomical landmarks, Nasion and the left and right pre-auricular points. The channel configuration, superimposed on the smoothed cortical surface, is shown in Figure 2.

Data preprocessing

Head modeling

We used the anatomical landmarks and 10–20 electrode positions for each subject to compute a realistically shaped head model by warping a generic head model, the Montreal brain phantom (Collins et al., 1998), to match individual positions (Darvas et al., 2006). We used this warped anatomical model to construct a boundary element model (BEM) for each subject.

The BEM computation was carried out with the BrainStorm software package (www.neuroimage.usc.edu/Brainstorm). The BEM consisted of three layers, skin, outer and inner skull. The bioelectric forward problem was solved with this BEM, using the generic cortex, which was tessellated into 10001 surface nodes as cortical source space. The resulting 27×10001 forward field matrix, which maps the electrical potentials of all unit dipolar sources on the cortex to all 27 electrodes, was used in subsequent inverse computations mapping the electrical potential changes, as measured on the scalp, to the cortex for each subject (Darvas et al., 2004). While this surface landmark based warp does not produce an exact representation of each individual's head geometry, it is sufficient to identify activity mapped to the correct functional anatomical locations such as primary motor areas. An advantage of the generic head model is that activity for all subjects is mapped into the same source space and hence allows us to perform a group analysis on cortical activity maps.

Data preprocessing

The data was segmented into 3 s long trials, with $t=0$ s set on the video onset, with a 1 s long baseline and a 2 s long post stimulus period. Although inter trial intervals were longer we did not include earlier times, to avoid contaminating the baseline with activity resulting from the beep at $t=-2$ s. We also did not include the end of the video at 2.7 s, since subjects typically produced large blink artifacts when the video stopped playing. A 60 Hz notch filter was applied to the raw data set. We visually inspected each trial of the EEG data for eye and movement artifacts. Specifically, we rejected trials with large power increases in the 1–4 Hz band in frontal electrodes, typically for eye-blink artifacts and trials, which show a spatially widely distributed increase in the 71–100 Hz range, which would indicate facial or neck muscle artifacts. Trials were rejected if artifacts were present in any channel between 1 s prior to video onset and 2 s after. While no separate electrooculogram was recorded, the 1–4 Hz component in the frontal electrodes, specifically FP1 and FP2, provides a reliable indication of eye movement artifacts. Overall, the segmentation procedure resulted in between 42 and 98 trials per subject. We down sampled the EEG data to 400 Hz to facilitate the subsequent signal analysis. All EEG data were also re-referenced to a common average reference (CAR), after artifact rejection, to reduce common mode noise.

Signal analysis

Inverse solution—We used a weighted linear minimum norm least squares (MNLS) method to map data from the EEG sensor space to the generic cortical surface. In this approach, the forward field matrix was weighted by the regularized noise covariance matrix of the EEG data in order to reduce the impact of noisy channels on the cortical mapping (Dale and Sereno, 1993). The noise covariance was computed from all trials over a time segment from -800 ms to -100 ms. Source orientations in our approach were constrained to be perpendicular to the cortical surface. We have used a similar method to successfully localize specific high gamma frequency changes during a movement execution paradigm (Darvas et al., 2010), using 27 channel EEG. Since the inverse mapping combines information from all EEG channels, in addition to the anatomical information from the

headmodel, effects of a consistent cortical HG source can be amplified in individual cortical source locations. While the HG signal might be too weak in the channel domain (e.g. in channels C3/C4 over the motor areas), the linear inverse mapping forms a weighted average for each source location, which can move the SNR of this source above the noise threshold.

Detection of band power changes and statistical testing—In order to map the power changes over time in a specific band, we band-pass filtered and Hilbert transformed the channel-domain data in that band for each trial, then computed the linear inverse cortical map for this trial. Since the inverse transform is linear, the filtering and Hilbert transform steps can be applied before the mapping, which is computationally efficient. For the resulting map of analytic signals in a given band, we take the square of the absolute value to convert the map into a single trial map of time varying band power. We then average these maps across trials in the source domain.

For this study, we consider two bands, which are involved during active motion, the beta band (17–25 Hz) and the HG band, which we divide into five 5 Hz sub-bands. This subdivision is necessary, as previous non-invasive studies of HG activity (Cheyne et al., 2008, Darvas et al., 2010) have found that individual HG frequencies typically fall into narrow bands, which can vary across individuals. The bands used in our analysis are shown in Table 1 below:

The nature of the beta-rhythm changes and the HG changes during actual movement execution is quite different. While the former is spatially broadly distributed, sustained over longer periods of time and typically observable with EEG with a robust signal-to-noise ratio (SNR), the latter is more short lived in time, spatially focal and has a low SNR in the EEG signal (Darvas et al., 2010, Miller et al., 2007). Therefore we use different approaches to analyze the Beta and HG power changes during the *movement* and *contrast* conditions.

For each subject, we computed the mean time varying map of beta band power changes across all trials for both conditions from –1s prior to stimulus onset to 2s post stimulus onset. We then normalized each cortical node of the map by applying a Z-transform (Bar et al., 2006) using a baseline period from –800 ms to –100 ms, resulting in a *Z-score map* of cortical activity. We do not make use of the full baseline of 2 s in order to ensure that the actual baseline period used is free of artifacts, i.e. the visual evoked potential at 0 ms or positions shifts and movements of the subject between stimulus presentations. Limiting the baseline to –100 ms before stimulus onset also ensures that stimulus locked events at 0 ms are not propagated into the baseline through the wavelet analysis. The resulting Z-score maps of beta band changes were used to form a grand average map of the beta power. We use a signed rank test across subjects to determine significant changes of beta power in the grand average. Since beta power changes occur over a spatially broad area, we defined two broad regions of interest (ROI) in the left (comprised of 165 voxels) and right (159 voxels) motor area (see Figure 2). In a realistic cortical geometry, these ROIs would cover more than just the primary motor areas. However, here, due to the inherent spatial imprecision of the inverse EEG mapping, a specific distinction of functional areas cannot be made based on geometry alone and by covering a large area, we allow for a reasonable spatial variation when searching for common effects across all subjects. For each subject and condition, we integrate the normalized time series over the whole ROI to form a single left motor ROI or right motor ROI time series of activation, representative of left or right motor activity for each subject and condition. These time series again are summarized in a grand average across all subjects to describe the average beta band response to the different stimuli for the left and right motor areas.

We use the onset of the beta power suppression for the *movement* condition at 300 ms post stimulus onset to guide our search for HG power changes. During actual movement, HG increases typically occur shortly (Ball et al., 2008, Cheyne et al., 2008, Darvas et al., 2010) after the beta suppression onset; hence we limit the feasible time range for HG power increases to the interval of 300–1000 ms post stimulus onset. Unlike beta power changes, HG changes are spatially focal (Darvas et al., 2010, Miller et al., 2007), broad spatial averaging over ROIs is therefore very likely to obscure any HG changes. In order to capture significant changes in HG power between the conditions in a small subset of cortical nodes in individual subjects, we use a non-parametric permutation test (Nichols and Holmes, 2002, Pantazis et al., 2005). With this test we identify cortical nodes that have increases in HG power during the *movement* in a 300 – 1000 ms interval over the *contrast* condition in each subject. Such tests have been used in cases where the distribution of the data is only known empirically (Darvas et al., 2009). Unlike beta power, which changes slowly over time and is stable over large spatial ROIs, HG power changes are transient, and our primary goal is to establish a significant difference over a period of non-stationarity, i.e. during the presumed stimulus induced changes of HG power from baseline. Once identified by the permutation test, we use individual sites, rather than integrating over a large generic ROI, to extract HG power time series to go into a grand average. It is important to note, that the permutation test itself, due to the large number of comparisons (across voxels in the ROI, frequency bands and time points considered) would have to have a very large number of permutations to yield a statistically significant result, after correcting for all comparisons. However our main purpose for this test here is to identify voxels and frequencies as candidates for the group statistic.

The permutation tests works as follows:

For each subject, we select an equal number of trials from each condition. While initially we have 100 presentations of both, *movement* and *contrast* stimulus, due to artifact rejection, we end up with a different number of trials for these conditions. For the permutation test, we choose the minimum number of available trials for both conditions. For all ten subjects an average of 67 trials per condition going into the permutation test, with a minimum of 42 trials and a maximum of 98 trials. From the combined set of both conditions we draw, without replacement, a number of trials equal to the number of trials per condition that went into the set. For example, for 50 trials for the *movement* condition and 50 trials for the *contrast* condition, we form a new set of 100 trials from both conditions, from which we draw randomly, without replacement, 50 trials. We then compute an average map of the HG power changes in a given HG band for this permuted set. This permutation average represents a sample from the Null-hypothesis, under which both conditions, *contrast* and *movement*, can be exchanged without having an effect on the HG power. We repeat this process 1000 times for each HG band and subject. For each cortical node of the map and each time sample, we compare this permutation mean to the original HG power map, specifically testing whether the original mean for the *movement* condition is larger than the permutation sample. This yields an empirical *p*-value that the HG power in a given band, for a given location at a given time in the *movement* condition is higher than for the *contrast* condition.

We use the resulting *p*-value maps for each frequency band, cortical location and time sample to threshold Z-score maps computed for each HG band. Under each condition, *contrast* or *movement*, there could be changes in HG power, relative to the respective pre-stimulus rest period, i.e. the baseline. However here we are interested in whether biological motion leads to increase in HG power over the *contrast* condition as well as over the rest-period. We therefore limit our search for potential sites of HG changes to the most significant sites, i.e. where HG power in the *movement* condition was consistently greater

than under the *contrast* condition irrespective of the baseline condition. After applying this threshold, we select those nodes in the left and right motor ROIs and those HG bands which have the maximum Z-score across ROI and frequency bands. In other words, we show the strongest HG increase relative to the pre-stimulus baseline for the movement condition. By using only the maximum values across ROI, frequency and time interval, we eliminate the multiple comparison problem across time, frequency and space, as this reduces the large number of possible time series per subject to choose from to just one. We then use the Z-score time series for these particular bands and cortical locations to compute a grand average of the HG power changes and to compare conditions. In order to test for selection bias, we apply the same procedure for the alternative hypothesis that the *contrast* condition yields an increase over the *movement* condition.

Results

Beta band changes

A summary of the beta band changes is shown in Figure 3. The time course of the beta band power changes relative to base line for both hemispheres, left and right, show a significant higher suppression of beta power during the *movement* condition compared to the *contrast* condition, regardless of handedness. Initial tests for a lateralization of the beta power changes did not show any significant difference between hemispheres across either right handed or left handed subjects, hence we ignored handedness in the analysis and present results averaged across all subjects. The integrated beta activity over the whole period from 300 ms to 2000 ms shows a clear distinction for both hemispheres between conditions, ($p < 0.05$) using a signed rank test, but within the *movement* condition there is no significant difference between the hemispheres in the amount of beta suppression relative to baseline. The time courses of the beta band power, as shown in the top row of Figure 3 also show that there is also slight beta suppression, when observing *any* video presentation of motion, but it is considerably weaker.

The grand average spatial map of changes in the beta band, integrated from the onset of the beta band suppression at 300 ms post stimulus to 2000 ms is shown in Figure 4. Since beta power changes occur for both conditions, the changes shown in this figure are the difference between the *movement* and *contrast* condition to highlight changes to observed movement. Only nodes on the cortical surface which show a significant ($p > .05$) difference across subjects, using a signed rank test, are shown. Spatially, there is a slight lateralization effect towards the left hemisphere. Only the spatial extent of the right hemisphere differences was reduced, significant differences are observed in fewer right hemisphere nodes. Left and right hemisphere differences were equivalent in strength

High gamma band changes

The results of the permutation test are shown in Figure 5. For each subject, a normalized cortical map is shown at the peak of the HG power during the *movement* condition in a specific HG band. The bands and peak times for each subject are listed in Table 2. A threshold for each node in each map has been set at $p < 0.001$, according to the permutation test described in the previous section. Thus only HG power changes over baseline during observation of the moving hand are shown, that are significantly higher for a particular cortical location and band than the corresponding power changes during observation of the moving scenery. Due to the conservative threshold, these areas are sometimes reduced to small patches containing a single node and its neighbors. The most significant HG changes for the right handed subjects occur in the left motor area, while for the left handed subjects HG activity in the right motor areas is significantly increased. It is noteworthy that the peak HG activity shown in Figure 5 shows only small areas of active cortex, which has two

implications: 1) Typically muscle artifacts, after mapping to the cortex, produce large increases in HG power over a wide area, i.e. the activity shown here is likely of genuine cortical origin. 2) By choosing a specific ROI, i.e. in the motor areas, we might spatially bias our findings. However we observe that activity is strongest and most significantly increased in only these regions, thus justifying our choice of ROI.

The grand average normalized power spectra for the contra and ipsi-lateral sites for both conditions and a baseline period (before onset of the video presentation) are shown in Figure 6. While the power spectra, which integrate activity over a 700 ms period, cannot show subtle transient changes in HG power, they can serve as an indicator of overall data quality. Specifically, these spectra do not show a broad band increase in high (>30 Hz) frequencies during stimulus presentation, which would indicate motor artifacts. In order to form the grand average across subjects, individual spectra were normalized to their total power.

The locations of the strongest HG increase during *movement* are summarized in Figure 5, which shows a lateralization with respect to handedness, of the HG power increase during the hand motion observation. Similar to the beta power changes, we summarize the HG power changes into a single time series per subject and condition. However, here, instead of integrating over the whole ROI, we select only the time course of these peak locations. For comparison, we also select the peak activity for the contralateral side, ignoring the results from the permutation test. The grand average for both conditions and hemispheres is shown in the top row of Figure 7. Due to the choice of the peak locations for the *movement* condition, individual time series for *movement* will generally show a greater increase in HG power over the *contrast* condition. However there is also a clear increase in HG power for the *movement* condition in the grand average, i.e. there is a relatively close alignment of the peak HG activity across all subjects. This is also evident from the peak times in Table 2, which cluster relatively tight around a mean at 638 ms post stimulus onset with standard deviation of 173 ms. The integrated HG power increase over baseline are shown in the bottom row of Figure 7, where the time series for each subject are integrated over an interval of 1.5 times s.d. around the mean peak time, i.e. from 378 ms – 898 ms. Using a signed rank test across subjects, we find that overall, HG power during hand movement observation is significantly ($p < 0.006$) increased over HG activity during observation of non-human originated motion for the contralateral side, i.e. in relation to handedness. We also find, that the HG power is significantly increased over the baseline period for the *movement* condition (one sided sign rank test, $p < 0.02$), while for the *contrast* condition a significant change from the baseline cannot be shown at $p < 0.05$. Furthermore, there is significant difference ($p < 0.048$) between contra and ipsilateral motor areas for the *movement* condition, while there is no significant increase in HG power on the ipsilateral side over the contrast condition. Likewise, this hemisphere does also not significantly increase HG power over the *movement* baseline period.

Testing for the alternative hypothesis

In addition to the group analysis for voxels, where we test our hypothesis that HG increases during observation of biological motion, we also test the alternative hypothesis, i.e. that HG increases during observation of non-biological motion. The results for this analysis are shown in Figure 8. The alternative hypothesis yields no significant increase over the original hypothesis. The p-value for the integrated power (over the original time interval, 378 ms to 898 ms) of the original HG change during biological motion over the HG power for non-biological motion is at $p < 0.049$. Similarly, we find no significant increase in HG power in the ipsilateral motor area under the alternative hypothesis. Since for both the original hypothesis (movement observation increases HG in the contralateral motor area) and alternative hypothesis (observation of non-biological motion increases HG in the contralateral motor area), we control for the maximum increase over baseline for all

frequencies, time points and locations, the resulting p -value for rejecting the alternative hypothesis does not have to be corrected for comparisons across these dimensions.

Alternative causes of HG increase in the motor areas

It is difficult to produce a contrast condition to biological motion that is sufficiently similar to the original stimulus, yet appears sufficiently different in perception to be useful as a contrast stimulus. Hence, our contrast stimulus is not matched well in over all speed of motion, shape or contrast. For purely optical differences in the stimuli, one would expect differences in the visual cortex. Also, one could speculate that the different stimuli produce differences in eye motion, which in turn could cause the observed differences of HG activity in the motor areas rather than the hypothesized genuine motor response to observed motion. As shown in Figure 5, activity in most subjects (with the exception of subject 1) is concentrated in the motor areas. Since these areas are sufficiently distant from the primary visual areas, cross-talk effects due to the use a linear minimum norm inverse from visual areas to our ROIs are unlikely, especially in the absence of visual cortex activity at the same time.

An increase in eye-movements during the *movement* condition over the *contrast* should lead to an increase of activity in frontal areas, which are most sensitive to the frontal electrodes, i.e. Fp1 and Fp2 ((Ball et al., 2009), which in turn pick up eye movements. While eye movements have power mostly in the 1–4 Hz band, it has been shown that saccades can have a significant impact on higher frequencies (Jerbi et al., 2009). In order to exclude a stimulus specific effect of eye movements we analyze the HG component, using the same bands as in our analysis of the motor ROIs, of the frontal channels Fp1 and Fp2 for each condition. We also integrate the HG power over the same interval (378 ms to 898 ms). The results are shown in Figure 9. There is no significant increase in HG power between *contrast* and *movement* condition. While both conditions produce a robust evoked potential (EP) in Fp1 and Fp2, correlated to an eye movement artifact, at video on- and offset, these EPs have no significant component in the HG bands that were used. In the figure, we show the EP of the sum of Fp1 and Fp2, as the EP is very similar for both channels. There is also no notable difference between the EPs in the interval that was used in our previous analysis to integrate HG power in the ROIs. When computing the correlation across subjects between HG power (for the *movement* condition) in the contralateral motor ROI, where we see a HG effect, and the HG power in Fp1 and Fp2, we find no significant correlation ($p < 0.542$ for Fp1 and $p < 0.421$). Likewise, we find no significant correlation for the HG power in the *contrast* condition (under the alternative hypothesis, where we pick the maximum HG power in the motor ROI) and HG power changes in Fp1 and Fp2 ($p < 0.089$ for Fp1 and $p < 0.742$ for Fp2).

From this we conclude that there is no measurable effect of eye movements on the HG power between conditions.

Discussion

The observed beta response is consistent with the existing findings on action observation using non-invasive methods (Babiloni et al., 2002, Hari et al., 1998, Jarvelainen et al., 2004). While action observation has been shown to activate a complex cortical network involving more than the motor areas, our interest in this study was primarily focused on the motor area, as other studies involving non-invasive electrophysiological measurements of cortical activity, i.e. MEG and EEG, have reported on activity in these regions for the lower part of the human EEG/MEG frequency spectrum.

Regardless of handedness, there is a stronger suppression in beta power during hand movement observation than during observation of movement of objects in both hemispheres.

As recent studies suggest (Kilner et al., 2009a), a cause for lateralization of the beta suppression can be the attended visual hemifield rather than the handedness of the observed hand. In our presentation of the stimulus, we used a video of a right hand, however, the presentation was at the center of the screen and we show only the hand without the rest of the body. Therefore both hemifields were stimulated equally thus leading to a bilateral attenuation of beta power, consistent with current observations (Aziz-Zadeh et al., 2006, Babiloni et al., 2002, Iacoboni and Dapretto, 2006). An inherent problem with studies of movement observation is that it is difficult to distinguish between responses induced by motor imagery versus execution-observation responses (Hari et al., 1998, Jarvelainen et al., 2004). The bilateral pattern of beta suppression we find however supports the assumption of execution-observation matching system activity rather than imagery, since the latter leads to a lateralized beta response (Pfurtscheller and Neuper, 1997, Pfurtscheller et al., 1998). Similarly, actual movement would also lead to lateralized beta suppression. While we did not explicitly control for involuntary hand movements, contamination by such movements is low (Babiloni et al., 2002, Jarvelainen et al., 2004) and movements strong enough to produce a detectable HG component in the EEG usually are accompanied by a strong EMG artifact after the initial HG response (Darvas et al., 2010). Our methods included visual rejection of all data for EMG artifacts, which (in contrast to real HG responses) are typified by broad spatial and temporal patterns and consequently obvious in the HG analysis procedures.

Our main reason for including the beta analysis into the present study was to identify the earliest onset of movement observation induced activity in the motor areas. This onset was then subsequently used to define the onset for the interval to search for HG activity.

Our study shows, that in addition to the beta response, there is also a HG component to observation of biological motion, complementing the overall congruence of the MNS with actual motion execution (Ball et al., 2008, Cheyne et al., 2008, Crone et al., 1998, Darvas et al., 2010). Unlike the beta band response, we find that the HG increase in power in the motor areas following observation of biological motion is lateralized, where for all eight right handed subjects the effect was dominant in the left hemisphere and for the two left handed subjects in the right hemisphere. Lateralization has also been observed (Nishitani and Hari, 2000), with a clear left hemispheric dominance of the MNS for functionally right-handed subjects. While the present study lacks the necessary number of left handed subjects to make a definite statement about the handedness effect on lateralization, the fact that both left handed subjects showed HG changes in the contralateral hemisphere, i.e. in the right motor area, indicates the possibility (at $p \leq .25$ chance level) that handedness might affect the localization of HG.

While high gamma oscillations appear to be ubiquitous and have been observed in wide range of paradigms and cortical regions, their exact role, other than being “a general index of cortical processing” (Crone et al., 2011), remains unclear. Independent of their specific function however, the “index” function of these high frequency rhythms might be of some utility, either as biomarker, i.e. to distinguish pathological conditions from normal function or as control signal for brain-computer interface applications.

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Literature

- Aziz-Zadeh L, Koski L, Zaidel E, Mazziotta J, Iacoboni M. Lateralization of the human mirror neuron system. *J Neurosci*. 2006; 26:2964–70. [PubMed: 16540574]
- Babiloni C, Babiloni F, Carducci F, Cincotti F, Coccozza G, Del Percio C, Moretti DV, Rossini PM. Human cortical electroencephalography (EEG) rhythms during the observation of simple aimless movements: a high-resolution EEG study. *Neuro Image*. 2002; 17:559–72. [PubMed: 12377134]
- Ball T, Demandt E, Mutschler I, Neitzel E, Mehring C, Vogt K, Aertsen A, Schulze-Bonhage A. Movement related activity in the high gamma range of the human EEG. *Neuro Image*. 2008; 41:302–10. [PubMed: 18424182]
- Ball T, Kern M, Mutschler I, Aertsen A, Schulze-Bonhage A. Signal quality of simultaneously recorded invasive and non-invasive EEG. *Neuro Image*. 2009; 46:708–16. [PubMed: 19264143]
- Bar M, Kassam KS, Ghuman AS, Boshyan J, Schmidt AM, Dale AM, Hamalainen MS, Marinkovic K, Schacter DL, Rosen BR, Halgren E. Top-down facilitation of visual recognition. *Proceedings of the National Academy of Sciences of the United States of America*. 2006; 103:449–54. [PubMed: 16407167]
- Cheyne D, Bells S, Ferrari P, Gaetz W, Bostan AC. Self-paced movements induce high-frequency gamma oscillations in primary motor cortex. *Neuro Image*. 2008; 42:332–42. [PubMed: 18511304]
- Collins DL, Zijdenbos AP, Kollokian V, Sled JG, Kabani NJ, Holmes CJ, Evans AC. Design and construction of a realistic digital brain phantom. *IEEE Trans Med Imaging*. 1998; 17:463–8. [PubMed: 9735909]
- Crone NE, Korzeniewska A, Franaszczuk PJ. Cortical gamma responses: searching high and low. *Int J Psychophysiol*. 2011; 79:9–15. [PubMed: 21081143]
- Crone NE, Miglioretti DL, Gordon B, Lesser RP. Functional mapping of human sensorimotor cortex with electrocorticographic spectral analysis. II. Event-related synchronization in the gamma band. *Brain*. 1998; 121 (Pt 12):2301–15. [PubMed: 9874481]
- Dale AM, Sereno MI. Improved Localization of Cortical Activity by Combining EEG and MEG with MRI Cortical Surface Reconstruction: A Linear Approach. *Journal of Cognitive Neuroscience*. 1993; 5:162–76.
- Darvas F, Ermer JJ, Mosher JC, Leahy RM. Generic head models for atlas-based EEG source analysis. *Hum Brain Mapp*. 2006; 27:129–43. [PubMed: 16037984]
- Darvas F, Miller KJ, Rao RP, Ojemann JG. Nonlinear phase-phase cross-frequency coupling mediates communication between distant sites in human neocortex. *J Neurosci*. 2009; 29:426–35. [PubMed: 19144842]
- Darvas F, Pantazis D, Kucukaltun-Yildirim E, Leahy RM. Mapping human brain function with MEG and EEG: methods and validation. *Neuroimage*. 2004; 23 (Suppl 1):S289–99. [PubMed: 15501098]
- Darvas F, Scherer R, Ojemann JG, Rao RP, Miller KJ, Sorensen LB. High gamma mapping using EEG. *Neuro Image*. 2010; 49:930–8. [PubMed: 19715762]
- Dinstein I, Thomas C, Behrmann M, Heeger DJ. A mirror up to nature. *Curr Biol*. 2008; 18:R13–8. [PubMed: 18177704]
- Hari R, Forss N, Avikainen S, Kirveskari E, Salenius S, Rizzolatti G. Activation of human primary motor cortex during action observation: a neuromagnetic study. *Proc Natl Acad Sci U S A*. 1998; 95:15061–5. [PubMed: 9844015]
- Hickok G. Eight problems for the mirror neuron theory of action understanding in monkeys and humans. *J Cogn Neurosci*. 2009; 21:1229–43. [PubMed: 19199415]
- Iacoboni M, Dapretto M. The mirror neuron system and the consequences of its dysfunction. *Nat Rev Neurosci*. 2006; 7:942–51. [PubMed: 17115076]
- Jarvelainen J, Schurmann M, Hari R. Activation of the human primary motor cortex during observation of tool use. *Neuro Image*. 2004; 23:187–92. [PubMed: 15325365]
- Jerbi K, Freyermuth S, Dalal S, Kahane P, Bertrand O, Berthoz A, Lachaux JP. Saccade related gamma-band activity in intracerebral EEG: dissociating neural from ocular muscle activity. *Brain Topogr*. 2009; 22:18–23. [PubMed: 19234780]

- Kilner JM, Marchant JL, Frith CD. Relationship between activity in human primary motor cortex during action observation and the mirror neuron system. *PLoS ONE*. 2009a; 4:e4925. [PubMed: 19290052]
- Kilner JM, Neal A, Weiskopf N, Friston KJ, Frith CD. Evidence of mirror neurons in human inferior frontal gyrus. *J Neurosci*. 2009b; 29:10153–9. [PubMed: 19675249]
- Miller KJ, Leuthardt EC, Schalk G, Rao RP, Anderson NR, Moran DW, Miller JW, Ojemann JG. Spectral changes in cortical surface potentials during motor movement. *J Neurosci*. 2007; 27:2424–32. [PubMed: 17329441]
- Nichols TE, Holmes AP. Nonparametric permutation tests for functional neuroimaging: a primer with examples. *Hum Brain Mapp*. 2002; 15:1–25. [PubMed: 11747097]
- Nishitani N, Hari R. Temporal dynamics of cortical representation for action. *Proc Natl Acad Sci U S A*. 2000; 97:913–8. [PubMed: 10639179]
- Pantazis D, Nichols TE, Baillet S, Leahy RM. A comparison of random field theory and permutation methods for the statistical analysis of MEG data. *Neuro Image*. 2005; 25:383–94. [PubMed: 15784416]
- Pfurtscheller G, Neuper C. Motor imagery activates primary sensorimotor area in humans. *Neurosci Lett*. 1997; 239:65–8. [PubMed: 9469657]
- Pfurtscheller G, Neuper C, Schlogl A, Lugger K. Separability of EEG signals recorded during right and left motor imagery using adaptive autoregressive parameters. *IEEE Trans Rehabil Eng*. 1998; 6:316–25. [PubMed: 9749909]

Figure 1. Still images from the presented video sequences. The top row shows images from the movement video, the bottom row from the contrast. The actual videos contain 81 images each.

Figure 2. Left and right motor area region of interest on the generic cortex and electrode configuration. The cortical surface has been smoothed for better visibility.

Figure 3.

Summary of beta power changes. Top row left: Grand average of the normalized (to Z-scores) beta power changes for the left motor ROI. The blue curve shows the power change, relative to the baseline, for the *movement* condition, the black curve shows changes for the *contrast* condition. The shaded areas indicate the standard error across subjects for each condition. Top row center: left and right motor ROIs. Top row right: Grand average of the normalized (to Z-scores) beta power changes for the right motor ROI. The red curve shows the power change, relative to the baseline, for the *movement* condition, the black curve shows changes for the *contrast* condition. Bottom left: Comparison between *movement* and *contrast* of the integrated beta power change from 300 ms – 2000 ms for the grand average of the left motor ROI. The error bars indicate the standard error across subjects. ** indicates $p < 0.01$ significance that overall beta power is lower for the *movement* condition, using a signed rank test. Bottom center: Comparison between the integrated beta power changes for the left and right motor ROI for the *movement* condition. Across subjects, there is no significant difference between hemispheres in beta power changes. Bottom right: Comparison between *movement* and *contrast* of the integrated beta power change of the right motor ROI. * indicates $p < 0.05$ significance that overall beta power is lower for the *movement* condition.

Figure 4.

Grand average map of beta band power changes over all subjects during the *movement* condition. The map shows the spatial mapping of beta band changes, integrated from 300 – 2000 ms post stimulus onset, for all subjects on the smoothed WM surface. A spatial threshold has been set at $p < 0.05$ (uncorrected), using a signed rank test between conditions movement vs. contrast, for each node. i.e. only those regions, where beta is significantly lower in the *movement* condition, as compared to *contrast*, are shown.

Figure 5.

Thresholded Z-score maps of HG power increase over baseline during the *movement* condition. Shown are increases in a particular HG band at the peak of the HG increase. The top two rows show areas of increase for right handed subjects, the bottom row shows the results for the two left handed subjects. For each node of the cortical map, the threshold was set at an uncorrected $p < 0.001$ that HG power during the *movement* condition is greater than HG power during the *contrast* condition. Lower right corner: Location of the strongest HG power changes in the motor ROI during *movement*. Locations for left handed subjects are shown in blue, for right handed subjects in red.

Figure 6.

Normalized grand average power spectra of the contralateral (left panel) and ipsilateral (right panel) sites of HG activities. Power during *movement* (300 – 1000 ms) is shown in blue power during the corresponding period for the *contrast* condition is shown in red. The average baseline power for a matched interval (–800 – – 100 ms) is shown in black. Spectra for each individual subject have been normalized to the total power for each subject.

Figure 7.

Summary of HG power changes. Top row left: Grand average of the normalized (to Z-scores) HG power changes for the *contralateral* motor ROI. The blue curve shows the power change, relative to the baseline, for the *movement* condition, the black curve shows changes for the *contrast* condition. The shaded areas indicate the standard error across subjects for each condition. The red lines indicate the interval over which power changes were integrated for a summary comparison. Top row right: Grand average of the normalized (to Z-scores) HG power changes for the *ipsilateral* motor ROI. The red curve shows the power change, relative to the baseline, for the *movement* condition, the black curve shows changes for the *contrast* condition. Bottom row left: Comparison between *movement* and *contrast* of the integrated HG power change, from 378 ms to 898 ms of the contralateral motor ROI. ** indicates $p < 0.01$ significance that overall HG power is higher for the *movement* condition. Bottom row center: Comparison between the integrated HG power changes for the contra- and ipsilateral motor ROI for the *movement* condition. Indicates $p < 0.05$ significance that HG power for the contralateral side is higher than for the ipsilateral side. Across subjects, there is a significant lateralization at $p < 0.05$. Bottom row right: Comparison between *movement* and *contrast* of the integrated HG power change for the ipsilateral motor ROI. There is no significant change of HG power for the ipsilateral side.

Figure 8.

Summary of the alternative hypothesis test of an increase in HG power during observation of non-biological motion for contralateral M1. Left panel: Grand average of the normalized (to Z-scores) HG power changes for the *contralateral* motor ROI under the alternative hypothesis, that HG power is increased for the *contrast* condition. The blue curve shows the power change, relative to the baseline, for the *movement* condition under the original hypothesis; the black curve shows changes for the *contrast* condition for the alternative hypothesis. The shaded areas indicate the standard error across subjects for each condition. The red lines indicate the interval (from the original hypothesis) over which power changes were integrated for a summary comparison. Right panel: Comparison between *movement* and *contrast* of the integrated HG power change, from 378 ms to 898 ms of the contralateral motor. The p -value for the difference between the alternative hypothesis and the original hypothesis is $p < 0.049$.

Figure 9.

Summary of the HG analysis of the frontal channels (Fp1 and Fp2). Top row: Average HG power changes, relative to baseline, for Fp1 and Fp2 for both conditions, i.e. *contrast* and *movement*. There is no significant change in power between conditions. Also, power changes in these electrodes, are much smaller than those in the respective ROIs (see Figure 7). Bottom row: The grand average evoked potential (EP) for the sum of Fp1 and Fp2, bandpass filtered from 3 to 40 Hz. The blue line and the blue shaded area show the EP and standard error for the *movement* condition, the black line and the gray shaded area show the EP and standard error for the *contrast* condition. The red lines indicate the integration interval for the HG power. The green lines indicate video on- and offset.

Table 1

Frequency bands used for analysis.

Band	Frequency [Hz]
Beta	17–25
HG1	71–76
HG2	77–82
HG3	83–88
HG4	89–94
HG5	95–100

Table 2

Summary of individual subject HG bands and peak times for HG power.

Subject	Handedness	Band	HG peak time [ms]
1	Right	HG3	608
2	Right	HG5	932
3	Right	HG4	637
4	Right	HG4	822
5	Right	HG4	487
6	Right	HG1	712
7	Right	HG1	380
8	Right	HG2	695
9	Left	HG2	697
10	left	HG5	418