An Oscillatory Hierarchy Controlling Neuronal Excitability and Stimulus Processing in the Auditory Cortex

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Lakatos, Peter, Ankoor S. Shah, Kevin H. Knuth, Istvan Ulbert, George Karmos, and Charles E. Schroeder. An oscillatory hierarchy controlling neuronal excitability and stimulus processing in the auditory cortex. J Neurophysiol 94: 1904–1911, 2005. We predict a more extensive systematic dependence of spontaneous EEG (Freeman and Rogers 2002; Vanhatalo et al. 2004). We propose that the hierarchy controls baseline excitability and thus stimulus-related responses in a neuronal ensemble. We propose that the hierarchical organization of ambient oscillatory activity allows auditory cortex to structure its temporal activity pattern so as to optimize the processing of rhythmic inputs.

INTRODUCTION

The EEG has been widely used for over 75 yr as a measure of human brain function (see e.g., Berger 1929). However, because of the dynamic complexity of the EEG, our understanding of its control and functional significance remains rudimentary. Recent studies have begun to link specific brain operations to specific components of the EEG, including gamma (Bertrand and Tallon-Baudry 2000; Engel et al. 2001; Fries et al. 2001b; Singer and Gray 1995), “theta” (Buzsaki and Draguhn 2004; Chrobak et al. 2000; Kahana et al. 2001), and “alpha” (Makeig et al. 2004; Worden et al. 2000). This study exploits and extends these findings.

Our study focused on three issues. The first issue is the intrinsic oscillatory “structure” of the EEG. There is an apparent relationship between theta oscillatory phase and gamma oscillatory amplitude in hippocampus (Bragin et al. 1995; Buzsaki et al. 2003; Csicsvari et al. 2003) and entorhinal cortex (Chrobak and Buzsaki 1998; Cunningham et al. 2003). Human recordings also show that periodic high-frequency oscillations are nested in lower frequencies and appear rhythmically in the spontaneous EEG (Freeman and Rogers 2002; Vanhatalo et al. 2004). We predict a more extensive systematic dependence of higher frequency oscillatory amplitude on lower frequency phase than that outlined by these studies. The second issue is the relationship between the EEG and neuronal excitability. EEG oscillations are hypothesized to reflect cyclical variation in the excitability of neuronal ensembles (Bishop 1933), but this idea has been explored mainly with regard to the low (<1 Hz) frequency oscillations (Contreras et al. 1996; Sanchez-Vives and McCormick 2000; Steriade et al. 1993). The final issue is the relationship between the EEG and neuronal excitability. While ongoing activity apparently can influence sensory responses (Arieli et al. 1996; Fiser et al. 2004; Fries et al. 2001a; Kruglikov and Schiff 2003; Massimini et al. 2003; Polich 1997; Truccolo et al. 2002), the extent of these effects and mechanisms by which they are controlled are poorly understood.

We analyzed both the spontaneous and stimulus-driven oscillatory activity present in cortical laminar profiles of field potentials and concomitant multiunit activity (MUA). These profiles were recorded with linear array multielectrodes during penetrations through primary auditory cortex (A1) in awake rhesus monkeys. Rather than analyzing the laminar field potential distribution itself, we analyzed its second spatial derivative, the current source density (CSD) profile, because this approach eliminates effects of volume conduction and allows more direct interpretation of field potential oscillations in terms of the underlying synaptic activity in the local neuronal ensemble (Nicholson 1973; Schroeder et al. 1995). We introduce a novel hypothesis about the “hierarchical” organization of EEG oscillations, the “oscillatory hierarchy hypothesis.” This hypothesis states that the amplitude of the oscillations at each characteristic frequency (gamma, theta, etc.) is modulated by the oscillatory phase of a local lower frequency oscillation. We present evidence that the phase of these spontaneous oscillations controls the excitability of the local cortical neuronal ensembles and thus strongly influences stimulus processing. Because the oscillatory hierarchy can entrain to repetitive stimulation, ambient activity can adapt so as to optimize the processing of rhythmic components present in many natural acoustic stimuli.

METHODS

Surgery

Four male macaques (Macaca mulatta), weighing 5–7 kg, were prepared for chronic awake electrophysiological recording. All pro-
Electrophysiological recording

Recordings were made in an electrically shielded, sound-attenuated chamber with SONEX ProSPEC Composite sound absorbing foam. Laminar profiles of field potentials (EEG) and concomitant action potentials (multunit activity or MUA) were recorded during 25 experiments in four awake rhesus monkeys (number of experiments in the individual monkeys: 5, 8, 6, 6), using a linear array multi-contact electrode (24 contacts, 100-μm intercontact spacing) positioned to sample from all the layers simultaneously (Schroeder et al. 2001) (Fig. 1A). Signals were impedance matched with a preamplifier (10× gain, band-pass DC 10 kHz) situated on the electrode, and after further amplification (500×), the signal was split into field potential (0.1–500 Hz) and MUA (300–5,000 Hz) range by analogue filtering. Field potentials were sampled at 2 kHz/16-bit precision, MUA was sampled at 20 kHz/12-bit precision. Additional zero phase shift digital filtering (300–5,000 Hz) and rectification was applied to the MUA data, and finally it was integrated down to 1 kHz (sampled at 2 kHz) to extract the continuous estimate of cell firing. One-dimensional CSD profiles were calculated (Fig. 2A) from the spatially smoothed (Hamming window) local field potential profiles using a three-point formula for estimation of the second spatial derivative of voltage (Nicholson and Freeman 1975; Ulbert et al. 2001). CSD analysis provides an index of the location, direction, and density of transmembrane current flow, the first-order neuronal response to synaptic input (Schroeder et al. 1998).

Experimental design

In each of the 25 experiments, the laminar CSD profile evoked by binaural Gaussian noise bursts was used to position the multielectrode array to straddle the auditory cortex from the pial surface to the white matter (Schroeder et al. 2001). Once the position was refined, it was left stable for the duration of recording. In this study, the subjects were conditioned to sit quietly, but were not required to attend or respond to the stimuli. During recording, they were monitored continuously using EEG recording and infrared video and were kept in an alert state by interacting with them. At the beginning of each experimental session, after refining the electrode position in the neocortex, we determined the best frequency off-line, we recorded 2 min of spontaneous activity followed by an ~2-min stimulus train (n = 150) consisting of the best frequency pure tone with an interstimulus interval (ISI) of 767 ms. We repeated this if there was any sign of the monkey moving during the recording and only analyzed artifact-free recordings.

Data analysis

Data were analyzed off-line using Matlab (Mathworks, Natick, MA). After selectively averaging the CSD and MUA responses to a series of seven randomly presented pure tones and Gaussian noise, we functionally assigned each recording site to AI versus belt auditory cortex, as determined by preimplant MRI (Schroeder et al. 1998). Further, to determine the location and density of transmembrane current flow, the second spatial derivative of voltage (Nicholson and Freeman 1975; Ulbert et al. 2001). CSD analysis provides an index of the location, direction, and density of transmembrane current flow, the first-order neuronal response to synaptic input (Schroeder et al. 1998).

FIG. 1. Laminar amplitude profile of spontaneous oscillation currents in area AI. A: for each experiment, field potentials were recorded with a linear array multi-contact electrode positioned to sample from all cortical layers. Laminar boundaries were determined based on functional criteria. B: a characteristic 1.5-s long spontaneous intracortical field potential recording. We applied 1-dimensional current source density (CSD) analysis to the field potentials obtained with the linear array multielectrode to estimate the locally generated intracortical synaptic currents. C: color-coded CSD map of the spontaneous activity segment shown in B; CSD was derived from the field potential profile using a 2nd derivative approximation. Current sinks (red) represent net inward transmembrane current flow in the local neuronal ensemble; sources (blue) represents outward currents. D: laminar amplitude profiles of the spontaneous oscillation currents (delta, theta, and gamma) gained from wavelet analysis of the spontaneous CSD segment (C) show 2 maxima in the supragranular layers (S1 and S2): 1 in the granular (G1) and 1 in the infragranular layer (I1). These 4 peaks were reliably identifiable in all recordings. Sites were not in adjacent electrode channels. E: graphs show pooled data (all 25 experiments in 4 subjects) for the amplitude of spontaneous delta, theta, and gamma band oscillations obtained from the 2 supragranular (S1, S2), granular (G1), and infragranular electrodes (I1). Error bars represent SE. Amplitude of delta and theta oscillations in the infragranular layer were significantly greater than in granular and infragranular layers. Gamma amplitude was significantly smaller in the infragranular than in the supragranular and granular layers.
cortex (Fu et al. 2004; Schroeder et al. 2001; Steinschneider et al. 1995). In this study, only recordings obtained from area A1 were used. Our data set consisted of two parts: 1) 2-min spontaneous recordings and 2) event-related responses evoked by a stimulus train \( (n = 150, \text{ISI} = 767 \text{ ms}) \) of best frequency pure tones (also recorded continuously and epoched off-line from -2,000 to 2,000 ms to avoid edge effects of the wavelet transformation). For the analysis of spontaneous and event-related oscillations, instantaneous power and phase were extracted by wavelet decomposition (Morlet wavelet) on 84 scales from 1 to 101.2 Hz (Fig. 2B). After applying the wavelet transformation, we calculated the square root of the power values to estimate the amplitude of the local oscillation currents (Fig. 2A). To compute oscillatory amplitude or MUA amplitude as a function of oscillatory phase, we sorted the phase values obtained from the wavelet transformation for the entire 2-min recording from \(-\pi\) to \(\pi\) radians (Fig. 2D, bottom trace). We applied the permutation vector obtained from sorting the phases to the oscillatory amplitude values and the MUA that was recorded concomitant. Blue trace in the middle and red trace on top show rearranged oscillatory amplitude and MUA values.
means we rearranged the oscillatory amplitude and MUA time series using the data gained from sorting the phase values from $-\pi$ to $\pi$ radians. If, for example, the fifth data point is closest to $-\pi$ in the original phase series, in the rearranged phase series, it will be the first, and after applying the permutation vector gained from rearranging the phase values, the fifth point of the oscillatory amplitude time series and concomitant MUA will also be the first in the rearranged series. After rearranging, the data were down-sampled to 360 data points.

Pooled amplitude and frequency values were evaluated statistically by ANOVA. When significant changes were detected with ANOVA, multiple comparisons (Statistical Toolbox of MatLab) were used to determine which pairs of means were significantly different. Phase values were analyzed by circular statistics methods. Pooled phases were compared using the Watson $U^2$ test for circular data. Significant deviation from uniform (random) phase distribution was tested with Rayleigh's uniformity test.

RESULTS

Laminar profile of spontaneous oscillations in area A1 of the auditory cortex

Spontaneous rhythmic activity was observed in all cortical layers, but four locations had higher amplitude oscillations than the surrounding ones (Fig. 1, D and E). Two of these locations were in the supragranular layers ($S_1$ and $S_2$), one in the granular layer ($G_1$), and one in the infragranular layers ($I_1$). For each penetration, these sites were identified empirically by noting the peaks in the spontaneous oscillatory spectrum (Fig. 1D). Across penetrations, mean distance was 324 $\mu$m between the $S_1$ and $S_2$ ($SD = 88$), 488 $\mu$m between the $S_2$ and $G_1$ ($SD = 112$), and 364 $\mu$m ($SD = 86$) between the $G_1$ and $I_1$ electrodes. Subsequent analyses focused on these sites.

The spontaneous CSD in each site contained three spectral peaks in the delta, theta, and gamma frequency ranges; this is shown using the amplitude spectrum of the $S_2$ site (Fig. 3). As shown in Fig. 1E, amplitudes of theta and delta oscillations in the supragranular layers were significantly greater ($P < 0.05$) than in granular and infragranular layers. There was no significant difference in gamma amplitude in the supragranular and granular layers, but it was significantly smaller ($P < 0.05$) in the infragranular layers than in more superficial sites. The mean frequency of oscillation within each band did not differ significantly across layers.

Hierarchical organization of spontaneous oscillations

In the spontaneous activity, theta and gamma frequency oscillation currents showed large periodic amplitude fluctuations, and these had systematic patterns: gamma oscillation current density fluctuated at theta frequency, whereas the theta oscillation current density fluctuated at delta frequency. Furthermore, the highest amplitude theta oscillations occurred at a specific phase of the delta oscillation (the rising +positive going part of the oscillation on electrode $S_2$) and the same relationship held between the gamma oscillation amplitude and the theta oscillatory phase. We named the phase of the lower frequency oscillation where the largest amplitude higher frequency fluctuations occur the “bursting phase” of the wave. This is shown using activity from the $S_2$ site (Fig. 4A, top 2 traces). These effects occurred in all layers (the oscillatory amplitude difference between bursting and opposite phase was significant for all frequencies, Wilcoxon signed rank, $P < 0.01$), but were greatest in supragranular sites, as the top graph of Fig. 4B shows. We noted a periodic 0.05- to 0.2-Hz fluctuation in the amplitude of the delta waves, but technical constraints (data were digitized after passing the analog signal through a high-pass filter with a corner frequency of 0.1 Hz) precluded quantitative analysis here.

We also examined the relationship between the phase of delta, theta, and gamma frequency oscillation currents and MUA (Fig. 4A, bottom 3 traces). There was a clear phase related modulation of the MUA in all the layers (the MUA difference between the phase with maximal MUA, the firing phase, and opposite phase was significant for all frequencies, Wilcoxon signed rank, $P < 0.01$), with the modulation being greatest in the granular layer (Fig. 4B, bottom graph). In the case of delta and theta oscillations, the firing phase appeared to lag slightly behind the bursting phase, but there was no significant difference between the two angular means (Watson $U^2$, $P > 0.05$).

Entrainment of delta oscillation to stimulus presentation

The event-related CSD at the peak of the initial transient cortical response had three clear amplitude maxima (Fig. 5A), similarly to the spontaneous spectrogram (Fig. 3). While there were no significant differences between spontaneous and stimulus-driven conditions in the frequencies of theta and gamma oscillations, the mean frequency of delta oscillations was significantly different in the two conditions (ANOVA, $P < 0.05$). Strikingly, the peak frequency of stimulus-related delta oscillation was a near perfect match to the stimulus rate; in 22 of 25 experiments, the peak frequency in the delta band during stimulation was 1.33 Hz (the rate auditory stimuli were delivered at). Delta oscillation was different from the other two types in two more aspects: whereas stimulus-related theta and gamma oscillations were significantly larger ($P < 0.05$) than the spontaneous oscillatory amplitudes, the amplitude of delta oscillation was significantly smaller ($P < 0.05$). Also while
there was a clear pre- to poststimulus amplitude increase in the theta and gamma range, the amplitude of the delta oscillation showed no stimulus-related change (Fig. 5B). While the amplitude and frequency of spontaneous and stimulus related delta oscillations were significantly different, there was no difference in their laminar distribution (Fig. 6). This argues against the general possibility that spontaneous and entrained delta oscillations are generated by different neural elements.

FIG. 4. Oscillatory phase, oscillatory amplitude, and MUA relationships of spontaneous oscillation currents. A: top 2 traces show delta phase-theta amplitude and theta phase-gamma amplitude relationship in an individual experiment. Three cycles are shown for illustration purposes. Oscillatory amplitude is clearly modulated by lower frequency oscillatory phase. Bursting phase of the lower frequency oscillation is the phase with the maximal higher frequency oscillation amplitude. For example, in the case of delta oscillation, it is the phase where the largest amplitude theta oscillations occur. Overlaid box and whisker plot show pooled data from all 25 experiments in 4 subjects (box has lines at lower quartile, median, and upper quartile values, whereas whiskers show extent of data). Bottom 3 traces show modulation of concomitant MUA by the phase of different frequency spontaneous oscillations in an individual experiment (same as above). Firing phase is the phase of the spontaneous oscillation currents during which cells are most likely to generate action potentials (largest MUA). Overlaid box and whisker plots show pooled data for bursting and firing phases for all sites (S1, S2, G1, and I1).

FIG. 5. Frequency and spectral amplitude of event-related oscillations. A: gray trace shows wavelet amplitude spectrum of stimulus-related CSD at the lower supragranular site (S2) 30 ms poststimulus in the same experiment for which the spontaneous spectrum was shown in Fig. 3. Box and whisker plots show pooled data (all 25 experiments in 4 subjects) for amplitude maxima and frequency of event-related oscillation currents in delta, theta, and gamma bands. Outliers are marked by a red cross. B: pooled (n = 25) single trial wavelet amplitude data show time-course of event-related oscillation amplitudes. Note that, whereas theta and gamma oscillation currents show stimulus-related increase, there is no change in amplitude of the ongoing delta oscillation. Apparent prestimulus oscillatory amplitude increase in theta and gamma bands is a side effect of temporal smearing caused by wavelet transformation.
These results indicate that, although there was no stimulus-evoked delta oscillation, the frequency of the ongoing delta waves rapidly adapted or "entrained" to the stimulation frequency. The entrainment is also indicated by the analysis of the prestimulus delta phase. That is, at the first stimulus in a train, delta phase was random (Rayleigh's uniformity test, $P / H_{11022} 0.05, n / H_{11005} 25$), but repetitive rhythmical stimulation resulted in a nonuniform prestimulus delta phase distribution in the rest of the trials (Rayleigh's uniformity test, $P / H_{11021} 0.05, n / H_{11005} 149$). This result was characteristic of all 25 experiments (Fig. 7).

**Effect of delta phase on stimulus processing**

Do the above described rhythmical oscillation current and multiunit activity amplitude fluctuations represent excitability changes that have an impact on stimulus processing? To address this question, we analyzed the effect of prestimulus delta phase on the stimulus-related CSD and MUA. The analysis and the results are summarized in Fig. 8. To measure

![Amplitude of spontaneous and event-related delta oscillations](image)

**FIG. 6.** Laminar amplitude profiles of spontaneous and event-related delta oscillation currents. Graphs on left show laminar amplitude profiles of spontaneous and event-related delta oscillation currents recorded in 1 experimental session (same as Fig. 1). Both show 2 maxima in the supragranular layer, 1 in the granular layer, and 1 in the infragranular layer. On the right, bars show pooled data (all 25 experiments in 4 subjects) for amplitude of spontaneous and event-related delta oscillations obtained from the 2 supragranular ($S_1, S_2$), granular ($G_1$), and infragranular electrodes ($I_1$). Error bars represent SE.

![Effect of delta phase on stimulus processing](image)

**FIG. 7.** Entrainment of ongoing delta oscillation during rhythmic acoustic stimulation. Stacked bars on left show prestimulus phase of delta oscillation for the 1st trial of each trial block analyzed (25 trial blocks in 4 subjects). Different shades of gray represent different trial blocks. Phase is measured at time of stimulus presentation (0 ms) on supragranular electrode $S_2$. Distribution is random, meaning that phase of the ongoing delta oscillation is random at the time of presentation of the 1st stimulus. Stacked bars on the right show pooled data for prestimulus delta phase in all succeeding trials (149 in each trial block) recorded in all 25 experiments. As in the previous graph, different shades of gray represent different experimental sessions. Number of trials is normalized to total number of trials ($n = 149$) in each experimental session ($n = 25$). It is obvious from the graph that the prestimulus delta phase in these trials is not random. This means that ongoing delta oscillation is influenced (entrained) by rhythmic acoustic stimulation.

![Delta phase dependence of the event-related response](image)

**FIG. 8.** Delta phase dependence of the event-related response. A: characteristic laminar event-related CSD (left) and MUA (right) profile. Channels with the largest event related oscillations are marked $S_1, S_2$ (supragranular), $G_1$ (granular), and $I_1$ (infragranular). To estimate effect of prestimulus delta phase on the event-related response, we measured rectified event-related CSD and MUA amplitudes for the 10- to 100-ms time interval (colored areas). B: event-related CSD and MUA amplitudes of trials with different delta phase in the same experiment. Event-related response (CSD and MUA) is clearly dependent on the prestimulus delta phase in all cortical layers (sites $S_1, S_2, G_1$, and $I_1$). There is an ideal phase that results in maximal event-related CSD and MUA (red stars), and there is a worst phase, which means that if prestimulus delta is in this phase, the event-related response will be much smaller (blue stars). C: event-related CSD profiles evoked by best frequency tones: left, average of trials with delta phase corresponding to the largest poststimulus activation (ideal phase, red stars in B); right, average of trials with delta phase corresponding to the smallest poststimulus activation (worst phase, blue stars in B). Overlaid traces show MUA for supragranular ($S_1$), granular ($G_1$), and infragranular ($I_1$) electrodes. D: pooled data (all 25 experiments in 4 subjects) showing modulation index (ratio of ideal phase response over worst phase response) for all cortical layers.
the influence of the prestimulus delta phase on the single trial event-related responses, we created two variables for each individual trial. These were the rectified CSD and the MUA amplitudes in the 10- (response onset) to 100-ms (offset of the auditory stimulus) poststimulus interval (Fig. 8A). We found that the amplitude of the evoked response is influenced by the phase of ongoing delta oscillation. This is shown in two ways for a single experiment in Fig. 8. Figure 8B shows the distribution of single trial response amplitudes (CSD, left; MUA, right) as a function of delta phase. Delta phase was systematically related to stimulus-evoked response amplitude in all the cortical layers, with the modulation being largest in the supragranular layers (Fig. 8D). Figure 8C shows the laminar profile of response (CSD with superimposed MUA) to a pure tone, averaged across trials with ideal prestimulus delta phase (left) and worst prestimulus delta phase (right).

The ideal prestimulus phase, which resulted in the largest event related activation ($\phi$ mean = 1.98 rad, $\phi$ dev = 0.88) was counter-phase to the worst phase, which resulted in the smallest event related CSD and MUA ($\phi$ mean = $-1.54$ rad, $\phi$ dev = 1.09). Statistical comparison of the ideal phase to the spontaneous bursting phase across all experiments revealed no significant difference (Watson $U^2$, $P > 0.05$). This fits with the idea that the bursting phase of the spontaneous oscillations represents increased excitability.

Effects were quantified across experiments using a modulation index (amplitude in ideal phase trials/amplitude in worst phase trials; Fig. 8D). Laminar differences in delta phase dependence are noteworthy. The extreme extent of delta phase dependence in the superficial layers indicates that processing in these layers is determined largely by “context,” that is, the instantaneous cortical state (in terms of frequency, amplitude, and phase of ongoing activity) in which inputs arrive.

**DISCUSSION**

An early hypothesis (Bishop 1933) suggested that spontaneous EEG reflects rhythmic variation of cortical excitability. Although the relationship of the EEG to neuronal activity was relatively neglected over the intervening years, recent studies have rekindled interest in this topic. Intracellular recordings in carnivores provided a striking demonstration of neuronal membrane potentials undergoing slow rhythmic shifts between depolarized and hyperpolarized states during slow wave sleep (Sanchez-Vives and McCormick 2000; Steriade et al. 1993). Other recent findings have pointed to an underlying structure to the EEG spectrum. As mentioned above I) in humans, cats, and rats, higher frequency oscillations display amplitude fluctuations with periodicity matching that of lower frequency oscillations (Amzica and Steriade 1998; Freeman and Rogers 2002; Lakatos et al. 2004; Steriade et al. 1996; Vanhatalo et al. 2004); 2) moreover, dependence of gamma oscillation amplitude on theta oscillatory phase is well characterized in rodent hippocampus (Bragin et al. 1995; Buzsaki et al. 2003; Csicsvari et al. 2003) and entorhinal cortex (Chrobak and Buzsaki 1998; Cunningham et al. 2003), and 3) there is gathering evidence that ongoing cortical activity has an effect on sensory processing (Arieli et al. 1996; Fiser et al. 2004; Fries et al. 2001a; Jansen and Brandt 1991; Kisley and Gerstein 1999; Kruglikov and Schff 2003; Massimini et al. 2003; Polich 1997).

This study provides a way to organize these important findings. First, we show that there is a hierarchical structure to the EEG, with amplitude at each oscillatory frequency being modulated by the phase of a lower frequency oscillation. This structure seems to extend from slow waves up through the gamma frequencies, although technical constraints in this study precluded quantitative assessment of the interrelationship of delta and very slow oscillations. Earlier findings from intracellular recordings in vitro suggest that layer 5 pyramidal cells play a key role in organizing and promoting slow oscillations in cortical neurons (Sanchez-Vives and McCormick 2000). The fact that, in our recordings, delta and theta oscillations are by far largest in the supragranular layers suggests that the pyramidal neuron ensembles there are also important in controlled cortical processing. The relationship between these findings remains to be established.

A second key aspect of our findings is that like the slow oscillation, the higher frequency oscillations reflect concerted excitability variations in cortical ensembles. This is reflected in local neuronal firing (MUA), which is clearly related to the phase of delta, theta, and gamma oscillations.

Finally, we confirm that ambient oscillatory activity has significant effects on stimulus processing (i.e., stimulus-related activity), in that, for each band of oscillation, there are both ideal and worst phases, during which stimulus responsiveness is enhanced or suppressed. The facts that spontaneous and event-related oscillations occur in the same frequency bands, are both phasic, and have similar laminar distributions implies that they use the same neural circuitry. However, whether the oscillatory hierarchy present in spontaneous activity is preserved in stimulus-related activity remains an important question for future studies.

Our findings have important implications for cortical processing of natural acoustic stimuli. While stimulus processing clearly is structured by the ambient context (Arieli et al. 1996), the onset of a sound can instantly reset the phase of the ambient delta oscillation, which effectively phase-locks the entire hierarchical structure of oscillatory activity to the stimulus. Thus effects of ambient activity on cortical processing should be more dramatic for more complex rhythmic inputs that are typical of a natural environment. For example, resetting of the ambient oscillatory hierarchy should be enormously useful in processing sounds that occur with a period of 1–4 Hz and have relatively phase-locked, rhythmic components at 4–10 (theta) and 30–50 Hz (gamma). It so happens that for primates, including humans, the temporal structure of numerous biologically relevant stimuli (Singh and Theunissen 2003), especially vocalizations (Shannon et al. 1995; Wang et al. 1995), fit this pattern remarkably well.

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