Synchronization in Monkey Motor Cortex During a Precision Grip Task. II.
Effect of Oscillatory Activity on Corticospinal Output

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Baker, Stuart N., Elizabeth M. Pinches, and Roger N. Lemon. Synchronization in monkey motor cortex during a precision grip task. II. Effect of oscillatory activity on corticospinal output. J Neurophysiol 89: 1941–1953, 2003; 10.1152/jn.00832.2002. Recordings from primary motor cortex (M1) during periods of steady contraction show oscillatory activity; these oscillations are coherent with the activity of contralateral muscles. We investigated synchronization of corticospinal output neurons with the oscillations, which could provide the pathway for their transmission to the spinal motoneurons. One hundred seventy-six antidromically identified pyramidal tract neurons (PTNs) were recorded from M1 in three macaque monkeys trained to perform a precision grip task. Local field potentials (LFP) were simultaneously recorded. All analysis was confined to the hold period of the task, where our previous work has shown that there is the strongest oscillatory activity. Coherence was calculated between LFP and PTN discharge. Significant coherence was seen in three bands, with frequencies of 10–14, 17–31, and 34–44 Hz. Coherence values were low, with the majority of PTN–LFP coherences having a peak lower than 0.05. The phase of coherence was approximately $-\pi/2$ radians for each band (with LFP polarity defined as negative upward), although there was some dispersion of phase across the population of PTNs. Coherence was also calculated between pairs of PTNs that had been simultaneously recorded. Where there was significant coherence, it was also generally smaller than 0.05. The phase of PTN–PTN coherence clustered around zero radians. A computer model was constructed to assist the interpretation of the experimental results. It simulated an integrate-and-fire neuron responding to synaptic inputs. A fraction of the synaptic inputs was synchronized with a simulated LFP; the remainder were uncorrelated with it. The model showed that coherence between the LFP and the output spike train considerably underestimated the fraction of synchronized inputs. Additionally, for a given fraction of synchronized inputs, coherence was smaller for high-compared with low-frequency bins. Cell discharge rate also influenced the spike–LFP coherence; coherence was higher for simulations in which the cell discharged at a faster rate. Thus although levels of PTN–LFP coherence seen experimentally were low, a considerable proportion of the input to the PTN must be synchronized with the global oscillatory activity recorded by the LFP. The low LFP–PTN coherences do however indicate that cortical oscillations are transmitted with only low fidelity in the discharge of a single PTN. Using further computer simulations, it was demonstrated that a small population of PTNs could encode the cortical oscillatory signal effectively, since the action of averaging across the population improves the signal:noise ratio. The oscillations will therefore be effectively transmitted to spinal motoneurons, and this has important consequences for the possible role of oscillations in motor control of the hand.

INTRODUCTION

A number of recent studies have investigated oscillatory activity around 15–30 Hz in the primary motor cortex (M1), both in humans using magnetoencephalography (MEG) (Conway et al. 1995; Salenius et al. 1997) and in monkeys using local field potential recordings (LFP) (Baker et al. 1997; Donoghue et al. 1998; Murthy and Fetz 1996a,b; Sanes and Donoghue 1993; see Baker et al. 1999 and Hari and Salenius 1999 for recent reviews). Such oscillations appear to be strongest during rest or steady contractions but are abolished during movements (Baker et al. 1997; Kilner et al. 1999; Salmelin and Hari 1994; Stancak and Pfurtscheller 1996). The cortical oscillations are synchronized with simultaneously recorded electromyogram (EMG) activity in contralateral hand muscles (Baker et al. 1997; Conway et al. 1995; Kilner et al. 1999; Murthy and Fetz 1992; Salenius et al. 1997).

A number of previous reports have also described synchronization between pairs of neurons, assessed using the cross-correlation histogram (Baker et al. 2001; Hatsopoulos et al. 1998; Smith and Fetz 1989; Vaadia et al. 1995), and single neurons have been shown to be phase locked to field potential oscillations (Baker et al. 1997; Donoghue et al. 1998; Murthy and Fetz 1996b). It is thus natural to assume that this phase locking to a common LFP signal leads to the synchronization between neurons. Murthy and Fetz (1996b) showed explicitly an increase in pairwise neuronal synchrony during epochs of field potential oscillatory activity. Baker et al. (2001) showed a rise in synchrony between pairs of single units during steady holding, when oscillatory activity is strongest.

It has been assumed by previous studies on corticomuscular synchrony that the oscillatory activity in the motor cortex is propagated down to the motoneurones via the synchronous oscillatory discharge of corticospinal output neurons. However, there is no direct evidence that the locking of such pyramidal tract neurons (PTNs) to cortical oscillations is sufficiently strong to give rise to the observed corticomuscular coherence. It is important to know the extent of oscillatory activity in the descending corticospinal command, as it could have a substantial effect on the extent of motoneurone recruitment and hence on the strength of a given contraction (Baker 1997).

We have here investigated the extent of the synchronization of identified PTNs with LFP oscillations using coherence anal-

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analysis in the frequency domain. We show that, although statistically significant coherence is present in three frequency bands, the values of coherence are low. Using a computer model of a neuron responding to synaptic inputs, we show that the low values of coherence are an inevitable consequence of the nonlinear nature of neural spiking. By adjusting our simulation to produce coherence spectra similar to a given experimental recording, we are able to obtain estimates of the fraction of the inputs to the cell at a given frequency that are synchronized with the global cortical oscillations. Finally, we use our model to simulate populations of PTNs and show how cortical oscillations could be transmitted down the corticospinal tract in such a way as to exert significant effects at the motoneuronal level.

**Methods**

The results reported in this paper are based on data recorded from three adult female macaques (monkeys 29, 33, and 35). Full details of the surgical and recording techniques are described in a previous paper (Baker et al. 2001). Briefly, the animals were trained to perform a precision grip task for food reward and were then surgically prepared to record multiple single units and LFP from M1. PTNs were identified from antidromic responses to stimulation of the medullary pyramidal tract. A precision grip task for food reward and were then surgically prepared for single units and LFP from M1. PTNs were identified from antidromic responses to stimulation of the medullary pyramidal tract.

Single unit action potentials and LFP were extracted by different filtering of the same preamplified electrode signal (1–10 and 10–250 Hz, respectively). The electrode was connected to the inverting input of the amplifier, so that LFP records have negative upward phase. A phase of zero between cell and LFP therefore indicates increased firing associated with a negative LFP.

All procedures were carried out under appropriate licenses from the UK Home Office.

**Analysis**

**Coherence calculation**

Coherence analysis of LFP–PTN and PTN–PTN pairs was performed. All coherence calculations used a sampling rate of 500 Hz. LFP had been initially sampled at this rate; unit spike trains were converted to a waveform sampled at 500 Hz by counting spikes in successive 2-ms bins.

Coherence calculations followed the equations given in Baker et al. (1997) and used fast-Fourier transforms of 512 point data sections covering the 1.024 s prior to the “End Hold” marker of the precision grip task. Our previous work has identified this period as that of maximal oscillatory activity in the precision grip task (Baker et al. 1997, 2001). The bin-width in the coherence spectra was thus 0.97 Hz.

In most recording sessions, more than one LFP was available for analysis, permitting an improvement in the signal-to-noise ratio. To take advantage of this, we first calculated the cross-correlation between each LFP pair during the task hold period selected as described above. Most pairs showed a strong positive correlation, peaking at zero lag; those that did not were excluded from subsequent analysis. We assume that electrodes were mostly in lamina V, since they recorded antidromically identified PTNs. For cell–LFP coherence calculation, we excluded the LFP from the same electrode that recorded the neuron, to avoid artifactual coherence with low-frequency components of the spike waveform. The remaining LFPs were then averaged together, and the composite waveform was used in coherence calculations. Calculations using only a single LFP, without this averaging procedure, produced similar cell–LFP phase and coherence estimates, although the coherences were slightly lower and the phase estimates less consistent due to the reduced signal-to-noise ratio.

For each analysis, a threshold level of coherence in one frequency bin was calculated for significance at $P < 0.05$ (see Baker et al. 1997 and Kilner et al. 1999 for details). Over a particular, predefined frequency range, coherence was then defined as significant if it crossed this threshold level in at least a criterion number of bins, chosen to provide an overall significance level of $P < 0.05$ (binomial test).

Where a range of frequencies showed significant coherence in the cell–LFP calculations, the phase for these points was plotted versus frequency. Care was taken to “unwrap” phase values that lay close to $\pm 180^\circ$ (Rosenberg et al. 1989). A regression line was fitted. If it was not significant ($P > 0.05$, $t$-test on regression coefficient), it was assumed that the relationship was best characterized by a constant phase, which was estimated by averaging over the band of interest. If, by contrast, the line had a significant, nonzero slope, the relationship was characterized as a constant phase plus a delay (Rosenberg et al. 1989). The phase was determined as the value of the regression line at the midpoint of the frequency range; the delay was determined directly from the line’s slope, in accordance with linear systems theory.

The combination of coherence across multiple recorded pairs is a nontrivial statistical problem (see Baker 2000; Halliday and Rosenberg 2000). We accordingly used two different approaches; their broad agreement provides confidence that the result is not simply an artifact of a particular statistical technique. The “nonparametric” method simply counted the percentage of bins at a particular frequency that exceeded the $P < 0.05$ significance line in the individual coherence spectra. The “parametric” method first converted an individual coherence spectrum $C(f)$ into a normally distributed variable $Z(f)$ by the following transform (Rosenberg et al. 1989)

$$Z(f) = \sqrt{2Z} \arctan(\sqrt{C(f)})$$

The variable $Z$ will have an approximately unit SD. If there is no coupling between the two signals used to calculate the coherence, the mean of $Z$ will not however be zero, but will be biased. The size of this bias depends on the number of data sections used to calculate the coherence (Benignus 1969). We found that formulae given in the literature failed to approximate this bias well for our data and instead chose to estimate it as the mean of $Z$ over the 100- to 250-Hz frequency range, a range chosen as having no significant coherence. This bias estimate was subtracted from $Z$ at all frequencies. The $Z$-scores so produced were then combined across individual measurements by

$$Z'(f) = \frac{1}{\sqrt{N}} \sum_{i=1}^{N} Z(f)$$

The composite $Z$-score so produced should have zero mean and unit variance if there was no coherence on average at that frequency.

In analysis of population data on the phase of coherence, the circular mean $\theta$ and mean resultant length $\hat{R}$ were calculated, defined according to Fischer (1993) as

$$\hat{R} \text{e}^{-i\theta} = \frac{1}{N} \sum_{n=1}^{N} e^{i\phi_n}$$

where $\phi_n$ are the individual phases measured. $\hat{R}$ varies from 0 to 1 and indicates the degree of cancellation that would occur in a population average of the activity due to phase dispersion, with $\hat{R} = 0$ indicating complete cancellation and $\hat{R} = 1$ indicating no cancellation (all phases in the population identical).

**Computational model**

To investigate the underlying cellular processes that could lead to the observed coherence between LFP and PTN firing, a computer...
model was used. This is illustrated in Fig. 5A and more fully explained in the description of that figure. The integrate-and-fire model neuron was simulated according to the differential equation

\[
C \frac{dV}{dt} = g_L(V - E_L) + g_E(E_E - V)
\]

where

\(C\) is the membrane capacitance, \(V\) is the membrane potential, \(g_L\) is the leak conductance, \(E_L\) is the membrane resting potential, \(g_E\) is the excitatory synaptic conductance, and \(E_E\) is the equilibrium potential of the excitatory synaptic channels.

\(C\) was taken as 1 \(\mu\)F/cm\(^2\), and \(g_L\) was 0.1 mS/cm\(^2\), chosen to give a membrane time constant of 10 ms. The resting potential \(E_L\) was taken as –70 mV and \(E_E\) as 0 mV. Each synaptically evoked current input to the cell generated a transient rise in \(g_E\) shaped as an alpha function, with time to peak of 1 ms and peak conductance 0.74 mS/cm\(^2\); these values gave excitatory postsynaptic potentials (EPSPs) of 100 \(\mu\)V in amplitude. Equation 4 was solved numerically using exponential integration (MacGregor 1993) with a time step of 0.5 ms in the MATLAB environment. Whenever the membrane potential exceeded a threshold (MacGregor 1993) with a time step of 0.5 ms in the MATLAB environment. Whenever the membrane potential exceeded a threshold of –60 mV, a spike was deemed to have been generated, and the potential was reset to \(E_L\).

The number of EPSPs impinging on the cell within one time step was determined from a Gaussian random variable with mean and variance \(k\). The decision to scale the EPSP count to have equal mean and variance was motivated by the fact that a Poisson counting process has this property; the Gaussian distribution is a good approximation to the Poisson distribution for moderate to high means. Using this approximation, the number of EPSPs per time step was noninteger and occasionally (although rarely) negative; no attempt was made to correct for this. The factor \(k\) was chosen to give the desired output firing rate by interpolating a curve of rate versus \(k\) determined in initial simulations; it varied from 3.65 for 5 Hz firing rate to 6.47 for 100 Hz.

RESULTS

A total of 176 PTNs recorded in three monkeys was analyzed in this paper; most data come from monkey 33 (9, 146, and 21 neurons from monkeys 29, 33, and 35, respectively); in monkey 33, the data were captured over 23 recording sessions in M1. The neuronal activity was recorded for between 38 and 563 successful trials. All neurons were active during the hold period of the precision grip task; a detailed description of their activity during the task is given in Baker et al. (2001).

Figure 1 shows raw data recorded during performance of two trials of the task. LFPs, recorded simultaneously from three electrodes, are shown in Fig. 1A, together with an average of all simultaneously recorded LFPs (Fig. 1B). Spike events from a simultaneously recorded PTN are shown in Fig. 1C. This PTN showed two phases of activity during the task: a burst in the movement phase and a more sustained discharge during the hold period (see Baker et al. 2001). Index finger and thumb movement traces are illustrated in Fig. 1D. Oscillations in the LFP were clearly present during the hold phase; the period used for analysis is indicated by gray shading.

Coherence between PTNs and LFP

Coherence calculated between the discharge of a single PTN and the LFP frequently rose above the theoretical significance level at multiple frequencies. An example of such a calculation for two cells is shown in Fig. 2, A and B. The dashed horizontal line in each plot shows the theoretical \(P < 0.05\) significance level. In Fig. 2A, there is a broad peak of coherence that rises above significance for most of the 20- to 40-Hz band. In Fig. 2B, two separate peaks can be seen, one around 20 Hz, the other around 40 Hz. In both cases, the coherence values, while significant, were extremely small, with peak coherences <

![Figure 1](http://jn.physiology.org/)

**Figure 1.** Raw data recorded from one session in monkey 33 during performance of 2 successive trials of the precision grip task. A: local field potential (LFP) recorded from 3 electrodes. The diagrams to the left show the relative location of the electrodes on the 4 \(\times\) 4 grid of our recording system (interelectrode spacing 305 \(\mu\)m). B: average of 9 simultaneously recorded LFPs, as used in subsequent analysis. C: single-unit events discriminated from an antidromically identified pyramidal tract neuron (PTN). D: thumb and index finger position traces. The shaded area indicates the period used for analysis; it represents 1.024 s before the "End Hold" behavioral marker.
In engineering applications of coherence analysis, such tiny coherences would be considered of little functional significance; this point will be considered in more detail using computer simulation below.

Figure 2, C and D shows the phase of the coherence illustrated in Fig. 2, A and B; phase is only plotted where coherence was statistically significant (P < 0.05). In both cases, the phase of the 20- to 40-Hz coherence lay between −π and 0 radians. In Fig. 2C, the phase seemed to show a linear relationship with frequency, indicative of a constant delay in the coupling between this PTN and the LFP.

A similar analysis was carried out for all 176 PTNs recorded; the results pooled across this population are illustrated in Fig. 3. Figure 3A shows the percentage of cells showing significant coherence (P < 0.05) at a given frequency. Dotted line shows the significance level (P < 0.05) using a binomial test. B: coherences combined as Z-scores, as described under METHODS. Dotted line shows the P < 0.05 significance level. Shaded regions in A and B indicate the 3 frequency bands analyzed subsequently. C–E: distribution of the phase of coherence (left) and peak coherence (right) for each frequency band, for PTNs showing significant coherence in a given band.
\( \dot{R} \) would be 1. These values therefore show that there was a particular phase dispersion for the 17- to 31-Hz band.

A small number of PTNs showed significant linear regressions of phase versus frequency within an individual frequency band \((n = 5, 11 \text{ and } 3 \text{ cells for the low-, medium-, and high-frequency band, respectively})\). The calculated delays were negative for 1/5, 1/11, and 3/3 cells in each band, indicating that the PTN was delayed relative to the LFP. The mean absolute delay, calculated across all 19 significant regression lines, was 30.1 ± 17.0 ms (mean ± SD).

On the right side of Fig. 3C–E the peak coherence for cells with significant coherence in each band is illustrated. The coherence values are small, in agreement with the data of Fig. 2, with mean peak coherences of 0.036, 0.042, and 0.026 for each frequency band, respectively. In the great majority of cell–LFP pairs, coherence was below 0.05. Given the small values of coherence, it is likely that some of the PTNs for which no significant coherence were seen were in fact synchronized with the LFP, but at such a low level that insufficient data were available to reach significance.

It was of interest to determine whether the coherence between PTNs and the LFP depended on the firing rate of the PTNs (see RESULTS of the computer model that follow). This was investigated in two ways. First, the peak coherences measured in Fig. 3 in each frequency band were plotted versus the neuron firing rate during the hold period of the task. There was no significant linear regression of peak coherence on firing rate for any of the three bands \((P > 0.05)\). Second, PTNs that both had significantly greater coherence judged over the entire range of 10–44 Hz than expected by chance and had been recorded for more than 150 trials of the task were selected. Eighty-three PTNs passed this selection criterion. The trials available were divided into two sets, according to whether the cell fired fewer than, or more than, the median number of spikes during the task hold period. Coherence with the LFP was recalculated using only the high-rate or only the low-rate trials, and the coherence was measured at the frequency bin that had shown maximal coherence when all the data had been used. The mean coherence for the low-rate trials was significantly lower than for the high-rate trials (coherence of 0.043 versus 0.057; paired \( t \)-test, 2-tailed \( P = 0.0012 \)).

**Coherence between the discharge of PTNs**

Cross-correlation histograms calculated between the spike trains of motor cortical neurons often show central peaks (Baker et al. 2001; Smith and Fetz 1989); it is therefore expected that PTN–PTN coherence should also rise above significance, since it is the frequency domain analog of cross-correlation. Figure 2E shows coherence calculated between the spike trains of the two PTNs that were illustrated in the earlier part of that figure. There is strong coherence at low frequencies, indicating a common slow task-related modulation of firing rate during the hold period. Such slow modulations are usually discounted when assessing peaks in the cross-correlation histogram (Baker et al. 2001). Coherence at higher frequencies is unexpectedly low and only rises above significance (dashed line; \( P < 0.05 \)) for a few bins. Figure 2F shows the coherence phase for these two PTNs. It is close to zero phase for all frequencies at which significant coherence was seen, consistent with previous observations that cross-correlation peaks are generally close to zero lag.

Figure 4 presents data on PTN–PTN coherence combined across all 460 cell pairs that were available; the display conventions are as in Fig. 3. The coherence, pooled using the two different techniques described under METHODS is shown in Fig. 4, A and B. Unlike in Fig. 3, there is no clear demarcation between the different frequency bands, but instead an apparent steady decline in the coherence with frequency. For bins falling within the highest frequency band marked, coherence barely rises above significance.

Measures of the PTN-PTN coherence in each of the three frequency bands used in Fig. 3 are shown in Fig. 4C–E. These represent PTN pairs with significant coherence \((n = 67, 68, \text{ and } 38 \text{ PTN pairs in the low-, middle-, and high-frequency band, respectively})\). On the left of each panel is shown a phase histogram. When computing PTN–PTN coherence, the choice...
of the neuron order is arbitrary. The phase of coherence between PTN1–PTN2 will be the same as that of PTN2–PTN1, but with the opposite sign. Accordingly, to ensure that the phase histogram truly represents the data, the phase calculated for a given pair has been plotted twice, once with a positive and once with a negative sign. In all three frequency bands, the phases are close to zero (mean resultant length \( R \) equals 0.52, 0.48, and 0.69 for the three bands, respectively). The plots to the right of Fig. 4C–E show the peak coherence for each frequency band. As for the PTN–LFP data of Fig. 3, the peak coherences are small (mean peak coherence 0.049, 0.026, and 0.022 for each band, respectively).

Simulation of PTN–LFP coherence

A surprising finding of the present work is the generally low level of coherence seen between LFP and PTN spikes. Oscillatory activity in the LFP is very clear (Fig. 1) and presumably represents a considerable level of oscillatory synchrony in the local network. As a part of this network, it would be expected that a substantial proportion of the input to a PTN would be synchronized with the LFP. To investigate further why coherence values of individual PTNs were so low, computer simulations of an integrate-and-fire model neuron were carried out.

Figure 5A illustrates the model. Two independent sources of Gaussian white noise were generated; one represented the LFP (white noise 1), the second represented other sources of synaptic input uncorrelated with the observed LFP (white noise 2). Experimentally, neither set of neuronal inputs will have the flat power spectra assumed by using white noise; however, this is a simplifying assumption that does not bias the subsequent calculations to favor any particular frequency band. These signals were processed using a filter that had attenuation as a function of frequency \( X(f) \) for the LFP and \( 1 - X(f) \) for the noise. The filtered signals were summed, to produce a result that was Gaussian white noise (flat spectrum up to the Nyquist frequency) with unit variance. The coherence as a function of frequency of this signal with the LFP was, by definition, \( X(f) \). The composite signal was rescaled to have a mean and variance of \( k \) (see METHODS); this was used to determine the size of the EPSP beginning in the model neuron at each time step.

Figure 5B presents the coherence calculated between the output spike train from the integrate-and-fire model neuron and the simulated LFP, in the condition where \( X(f) \) was equal to 0.2 at all frequencies. The coherence between the LFP and the input to the neuron was therefore fixed at 0.2. The different lines in this graph show the results when the mean cell firing rate was set to 5, 10, 20, 50, and 100 Hz. This was achieved using different values of the input scale factor \( k \), chosen to generate the desired mean firing rate following initial calibration simulations. It can be seen that, at all frequencies, the coherence is lower than the value of 0.2, which might have been expected. Two factors affect the coherence. The first is the average cell firing rate. Coherence at a given frequency is higher for cells with larger firing rates. Second, although the coherence between LFP and synaptic inputs did not vary with frequency, there was a strong decline in the LFP–spike coherence at higher frequency.

Normally, coherence between two signals can be interpreted as the fraction of the variance at a given frequency in one signal that can be explained by the other. The results of Fig. 5B show that coherence between a spike train and LFP considerably underestimates the proportion of the inputs to that cell that are synchronized with the LFP. Furthermore, the extent of the underestimation is greater for slowly firing cells and for high-frequency bands.
Figure 5C presents the phase of LFP-spike coherence for these simulations. In all cases, the phase is close to zero. When the firing rate is 50 or 100 Hz, the phase initially increases linearly with frequency, implying a constant delay between LFP and spiking. The slopes of the lines imply that, for low frequencies, the cell spikes lead the LFP by a constant 2.6 or 2 ms for the 100- or 50-Hz firing rates, respectively. There is a sharp fall in phase to become negative around the frequency equal to the firing rate of the cell.

The simulation results of Fig. 5 could explain why such low PTN–LFP coherence values were found. To determine how great these effects might be in experimental data, further simulations were carried out. An experimentally recorded PTN that had a coherence spectrum with the LFP clearly rising above significance was chosen. Its firing rate during the period of activity analyzed was determined, and the computer model of Fig. 5A was run, choosing the parameter \( k \) so that the model would replicate this mean firing rate. The coherence spectrum between simulated LFP and spikes was compared with the experimentally measured spectrum, and the model parameters \( X(f) \) were adjusted, in an iterative fashion, until the spectra became similar. The number of fitted parameters \( X(f) \) equaled the number of coherence observations, so that it was unsurprising that the experimental spectra could be well matched in this way. However, the values of the \( X(f) \) could then be examined to provide insight into the underlying mechanisms generating the observed coherence.

Figure 6 shows the results of such a series of simulations for four different PTNs. The top row of the figure (Fig. 6A–D) plots the experimentally determined spike–LFP coherence spectra (thin line) and those generated by the simulations with the final values of \( X(f) \) (thick line). The bottom row (Fig. 6E–H) presents the values of \( X(f) \). These can be interpreted as an estimate of the fraction of the cells’ input at a particular frequency that is synchronized with the LFP. This could be determined directly from the coherence, if the cell acted as a linear system. However, the nonlinear nature of the neuron makes it necessary to go through this modeling stage to access these values. The \( X(f) \) spectra have considerably higher values than the coherence spectra, showing that a considerable portion of the input to the PTNs is likely to be synchronized to LFP oscillations. The form of \( X(f) \) is also different from the coherence spectra. For example, in Fig. 6C, the coherence close to 20 Hz is approximately twice that close to 40 Hz. The plot of \( X(f) \) in Fig. 6G makes clear, however, that this is likely to result simply from the decreased sensitivity of coherence at high frequencies: the estimated values of \( X(f) \) for the high- and low-frequency peaks are very close.

**Coherence with corticospinal population output**

The simulations presented above show that coherence does not provide an accurate estimate of the proportion of the input to a PTN that is synchronized with LFP oscillations. However, by definition, the coherence is the correct estimate of the proportion of the cell’s output synchronized with the LFP. The low values of LFP–PTN coherence thus indicate that a single PTN carries a rather poor representation of the synchronized oscillations that occur in the motor cortex.

A single motoneurone does not receive input from just one PTN, but from many. It is therefore of interest to determine how cortical oscillations might be carried by a population of PTNs, each of which is only weakly synchronized with the LFP. Figure 7A presents results of simulations that addressed this question. A population of PTNs was simulated. Each cell received inputs as described in the legend to Fig. 5A. However, while the input representing the LFP (Fig. 5A, White Noise 1) was common to all cells, the other input (Fig. 5A, White Noise 2) was independent in each neuron. Each cell had \( X(f) \) equal to 0.05 for all frequencies and fired at a rate of 20 Hz. This latter value is close to the mean firing rate of PTNs during the hold period of the precision grip task (Baker et al. 2001). A population activity signal was generated by counting the total number of spikes produced by the entire cell population in each time bin. This is similar to an experimental recording of multunit activity.

Figure 7A plots the coherence between the population spike activity and the LFP as a function of frequency. Each line shows the result for a different number of cells in the population, from 1 to 100 neurons. As expected from the simulations already presented, a single cell shows very little coherence. However, with increasing population size, the cortical oscillations are more and more faithfully conveyed by the cell spiking activity. With the largest population tested of 100 neurons, the population activity has a very high coherence with the LFP of 0.65 at 25 Hz.

The mechanism of this effect is straightforward. The “noise” introduced by a single cell, both by inputs uncorrelated to the
are conveyed to motoneurones can be determined by calculating the coherence between LFP and the EMG of a contralateral contracting muscle. Figure 8 illustrates such a calculation for the adductor pollicis muscle, which shows good tonic activity during the hold phase of the precision grip task. EMG recordings were not available for monkey 35; however, in the other two monkeys, coherence rose above significance in both the middle- and high-frequency bands used in Figs. 3 and 4. There was no significant coherence in the low-frequency band. In monkey 33, coherence was seen at very low frequencies (<5 Hz); this probably reflects the frequency domain counterpart of movement evoked potentials, correlated with the EMG modulation produced during movement.

**DISCUSSION**

The present study has applied coherence analysis to a large population of identified PTNs and shown that these cells are synchronized with LFP oscillations, although coherence values are weak. Using computer simulations, we have investigated the functional implications of these weak coherence values. Our results show that a substantial fraction of the inputs to PTNs must be synchronized with the network oscillations. Furthermore, population firing among corticospinal neurons could reliably encode the motor cortical oscillations and thereby transmit them to spinal motoneurones. This has important consequences for the possible role of oscillations in encoding parameters for precise motor control by cortical outputs (Kilner et al. 2000).

**Size of coherence**

During the hold phase of the precision grip task, oscillatory activity in the motor cortex can readily be observed in both monkey LFP (Fig. 1) (Baker et al. 1997) and in human EEG or

![Image](https://via.placeholder.com/150)
MEG (Conway et al. 1995; Kilner et al. 1999, 2000; Salenius et al. 1997). Since these field potential measures must represent the summed synchronous synaptic potentials in a large number of cells, and since PTNs are an integral part of the motor cortical network (Jackson et al. 2002), we would expect a substantial part of the inputs to PTNs to be synchronized with the global oscillatory activity recorded by the LFP. Although previous reports have shown neuron–LFP synchrony (Baker et al. 1997; Murthy and Fetz 1996b; Donoghue et al. 1998; Pinches et al. 1997), this is the first study to quantify the extent of synchronization using coherence analysis. Contrary to expectations, the LFP–PTN coherence values are very low, with peak coherences all below 0.1 and most below 0.05. This would conventionally be taken as evidence that only a small fraction of the PTN inputs are synchronized with the LFP.

However, using the computer model of Fig. 5A, we have shown that such a conclusion is invalid. This is because interpretation of coherence as a fraction of one signal synchronized with another requires that the system under study is linear, whereas even the simplest model of a neuron is nonlinear because of the presence of the spike threshold. The effect of this nonlinearity appears to low-pass filter the coherence between a spike train and an input signal (Fig. 5B), so that high-frequency components of the input are transmitted less effectively than the lower frequency components. In addition, the attenuation of all components is greater for slowly firing cells. This is to be expected, since if a cell fires only a small number of spikes within a given time, it cannot encode fluctuations in its inputs as effectively as a cell that emits many spikes. Stein et al. (1972) and Halliday (2000) also used simulations to study the low-pass filtering of coherence by spiking, although the impact of different cell firing rates was not investigated. Protopapas and Bower (2001) showed in intracellular recordings from pyriform cortex that a neuron was less able to represent high-frequency components of a stimulus in its firing than low frequencies; high frequencies were better represented for the cells with higher firing rates. Matthews (1997) used simulations of motoneurones responding to sinusoidal inputs to investigate the same phenomenon, although, since he did not calculate coherence, his results cannot be directly compared with the present work.

When our computer model was used to simulate data with similar coherence spectra to the experimental recordings and the same cell firing rate, we were able to show that the fraction of the cell input synchronized with the LFP needed to be at least an order of magnitude greater than the coherence to reproduce the experimental spectra (Fig. 6). Little weight should be placed on the exact numbers generated by these simulations. The model used was a great oversimplification of the complex firing behavior of a cortical pyramidal neuron. It was a single compartment model (i.e., it had no dendrites), with no active conductances and a fixed spike threshold. All of these features, if included in the model, would add to the richness of its nonlinear behavior and would most likely change the exact numerical findings of Fig. 6. However, the model captures the main nonlinearity of the neuron response, namely the spike threshold. It should perhaps be seen as one step less naive than the implicit assumption of coherence analysis that the spiking neuron is a linear system. The critical finding that a substantial portion of the cell input is synchronized to the LFP would probably not be altered by using a more sophisticated model.

Thus the model shows that coherence analysis, applied without further processing, provides a poor assessment of the proportion of a PTN’s input that is synchronized with the LFP. However, by definition, it is the correct measure if we are interested in the cell’s output. The low LFP–PTN coherences then can only be interpreted as showing that a single PTN’s spikes carry the cortical oscillations with a signal-to-noise ratio normally smaller than 1:20. The transmission of oscillations from synaptic inputs to output spiking of the motoneurones should be subject to similar processes as examined here for the PTNs. If anything, the attenuation of the signal will be even greater for the motoneurones. Figure 5B makes clear that low firing rates lead to weaker input–output coherence, and motoneurones have lower firing rates than PTNs (around 10–30 Hz, Milner-Brown et al. 1973). The LFP signal should therefore be represented in the EMG with a signal-to-noise ratio smaller than around 1:20$^{2} = 1:400$. In fact, coherence measured between cortical field potentials and EMG is usually around 0.05–0.1 (see Fig. 8) (Baker et al. 1997; Conway et al. 1995; Salenius et al. 1997).

The solution to this problem appears to lie in an improvement to the signal-to-noise ratio with which the cortical oscillations can be conveyed to the spinal cord by averaging over a number of PTNs. Figure 7A shows that, while the LFP–PTN coherence may be small, it can rise to high levels if computed between the LFP and the firing of a population of 50–100 cells. There is only sparse and indirect evidence of how many PTNs might project to a single motoneurone. Compound EPSPs evoked in a motoneurone by pyramidal tract stimulation vary from 0.5 to 7.5 mV in amplitude (Fritz et al. 1985); the mean is 2.0 mV in forearm motoneurones and 3.5 mV in those innervating the hand. The only two published unitary cortico-motoneuronal (CM) EPSPs have heights of 25 and 120 $\mu$V (Asanuma et al. 1979), and similarly small values probably account for most of the postspike facilitation observed in hand muscles from CM cells (see Porter and Lemon 1993). This implies that perhaps 30–50, but possibly many more, PTNs form the cortical “colony” for a single motoneurone, so that averaging across a reasonably sized population probably does occur. Similarly, at the motoneurone level, between 100 and 600 motoneurones innervate a given limb muscle (Burke 1981), so that the EMG also represents a population average. At both the level of LFP to PTN and PTN to motoneurone transmission, therefore, substantial population averaging probably underlies the observed strength of transmission of oscillatory activity.

A number of authors have recently drawn attention to the detrimental effect that correlated noise can have on the improvement in signal-to-noise ratio achieved by population averaging (Lee et al. 1998; Shadlen and Newsome 1998). If the component of the input to the PTNs that was not correlated to the LFP was correlated between the PTNs, this would prevent the PTN population discharge from reliably encoding the cortical oscillatory signal. The Appendix gives a derivation of formulae that can be used to predict the LFP–population coherence that would be expected if the noise is uncorrelated, or perfectly correlated, between cells comprising the population. Figure 7B shows that, for a small population of six PTNs that were simultaneously recorded, the actual LFP–population coherence was only slightly lower than that expected if the noise were wholly uncorrelated between cells. It therefore seems
likely that population averaging will be effective in this system and that the descending corticospinal command will be substantially modulated by the oscillatory activity monitored by the LFP.

**Phase of coherence**

The phase of coherence between LFP and PTNs was centered around $-\pi/2$ (Fig. 3C–E). This indicates that the PTN firing is related to the integral of the LFP. The relationship of field potentials to intracellularly recorded activity is complex and depends critically on the location of the recording electrode relative to the active synapses. However, if the recording electrode is close to these synapses, the extracellular field potential will be approximately proportional to the transmembrane current and hence proportional to the derivative of the intracellular potential (Hubbard et al. 1969). Our results are therefore consistent with the PTNs being locked at zero phase lag with the synchronous postsynaptic potentials in the local population.

In contrast to our results, a number of previous studies have presented spike-triggered averages of LFP and have shown that the neuron spike is close to the negative peak of the LFP (Baker et al., 1997; Donoghue et al. 1998; and Murthy and Fetz 1996b in motor cortex; Gray and Singer 1989 in visual cortex). In addition, Murthy and Fetz (1996b) provided quantitative data on a population of such spike-triggered averages: the delay between spike and negative LFP peak corresponded to phases predominantly between $-\pi/2$ and zero, with a mean of $-0.48$ radians. All of these previous studies used time domain methods of analysis compared with the frequency domain coherence measure used here. This may underlie the slight differences between previous reports of near zero phase spike–LFP synchrony and our current finding of a phase difference of around $-\pi/2$.

The simulation of Fig. 7A assumed that all cells of the population were locked to the population activity with identical phase. Where there is variation in the distribution of phases across the population, there will be some cancellation in the population average and a consequent reduction in the amplitude of the oscillatory activity that is conveyed. Figure 3 showed that there was some phase variation between individual PTNs. This was greatest in the middle-frequency range examined and would have caused a reduction in the population oscillation amplitude to 29% of the size expected with no cancellation. Although this is appreciable, a sizable oscillatory signal will still reach the motoneuones.

For a small number of PTNs, there was a significant linear relationship between coherence phase and frequency within a frequency band, indicating that the cell firing was delayed or advanced relative to the LFP by a constant time. The small number of frequency bins available for each band meant that only relatively long delays were likely to reach statistical significance, and it would be unwise to place too much emphasis on the size of the delays that were calculated from the slopes of the regressions, which had a mean of 30.1 ms. However, it is known that the majority of intracortical fibers are slowly conducting, such that the mean velocity may be only 0.2 ms$^{-1}$ (see Pauluis et al. 1999), and further delays could be imposed postsynaptically by temporal summation and integration. The existence of rather long time delays between LFP and the firing of some PTNs is thus not unrealistic. In addition, phase advances can be introduced by the spiking process itself (see Fig. 5). Similar results have been described from modeling work by Matthews (1997) and Stein et al. (1972), where this is discussed in more detail. In our simulations, such phase advances were small.

**Implications for corticomuscular coherence**

A number of different workers have demonstrated coherence between measures of cortical population activity and contralateral EMG, in both man (Conway et al. 1995; Kilner et al. 1999; Salenius et al. 1997) and monkey (Baker et al. 1997; Murthy and Fetz 1992). Similar results were obtained in the two animals used for the present work for which EMG recordings were available (Fig. 8). As discussed above, our results imply that motor cortical oscillations are carried by the population discharge of PTNs reasonably faithfully. The coherence between the firing of PTNs and motoneurones will be influenced by the same mechanisms as discussed for LFP–PTN discharge. Even if the synchronous oscillations form the major part of the input to a motoneurone at a given frequency, the coherence between a single motor unit and the synchronous input is expected to be low. This is confirmed by the observation that coherence between hand muscle motor units during performance of the precision grip task is also rather low (Kilner et al. 2002). However, EMG is a population measure that sums over many motoneurones; by the same arguments as presented above, the coherence of PTN oscillations with EMG may thus be increased by population averaging. Hence we expect that synchronous cortical oscillations will be well represented in the EMG of a contracting muscle, producing the corticosral coherence that is observed.

Corticomuscular coherence is most often reported in the “beta” band of around 15–30 Hz (Baker et al. 1997; Conway et al. 1995; Kilner et al. 2000; Salenius et al. 1997). During strong contractions, coherence can also be seen at higher frequencies around 40 Hz, although it is not only confined to high-force contractions (Brown et al. 1998). The simulations of the present study provide a possible explanation for the latter finding. At higher force levels, motoneurones will fire at higher rate. The coherence of each single motor unit with the cortical oscillations at around 40 Hz will therefore be greater (similar to the effect shown in Fig. 5B), leading to a greater coherence between the EMG and the cortical recording. While it may be possible to see around 40 Hz coherence at low force levels, our simulation results predict that it is more likely to rise above significance for strong contractions.

One aspect of our results does not fit with the published data on corticosral coherence and is a puzzling anomaly. Coherence between cortical recordings and EMG is rarely seen around 10 Hz (see Fig. 8); if present, it is usually weak compared with that around 20 Hz (Conway et al. 1995; Kilner et al. 2000; Salenius et al. 1997). Exceptionally, some recent reports have demonstrated such low-frequency coherence between EMGs and electrocorticograms recorded from subdural grids implanted in patients undergoing surgery for intractable epilepsy (Ohara et al. 2000; Raethjen et al. 2002) and also between MEG and EMG (Gross et al. 2002; Marsden et al. 2001). It is possible that the findings with epileptic patients have been influenced by the underlying pathology of the sub-
jects or its pharmacological treatment. However, the discrepancy in the reports from normal subjects remains puzzling.

Contrary to these findings with EMG, we have shown here that there is clear coherence between PTNs and the LFP around 10 Hz, which is on average similar in strength to the coherence in the beta band (Fig. 3, A and B). The low-pass-filtering effect of neuronal spike trains on coherence described in detail in the simulations above should favor the low-frequency coherence and ensure that it is larger for a given proportion of synchronized input than the coherence at higher frequencies. This is true both for the transmission of the oscillations from LFP to PTN population discharge and from this descending command to the EMG. Given our present observations, it is thus remarkable that strong coherence at around 10 Hz is not routinely observed between motor cortical field potentials and EMG.

One speculative possibility is that a specific mechanism exists that prevents the motoneurone pool from synchronizing with the around 10 Hz oscillations present in the cortical descending command. This could be important in preventing excess physiological tremor, which occurs at frequencies around this value (Raethjen et al. 2000) and may be produced by instabilities in the spinal circuitry (Allum et al. 1978; Elble and Koller 1990). It is possible that tremor could become intolerable if such a system were driven strongly at its resonant frequency by corticospinal inputs and that this is prevented by some form of feedback or damping circuit within the spinal cord that is specific for this frequency range. Matthews (1997) investigated the response of the stretch reflex to sinusoidal inputs and concluded that a centrally mediated phase advance that acted together with the reflex conduction lags to minimize reflex oscillations around 10 Hz was present. Matthews (1997) demonstrated by simulations that part of this phase advance was probably produced by the intrinsic firing properties of the motoneurones, in a similar fashion to the phase advance that our present model is capable of introducing (Fig. 5C). However, there was also an additional component due to other central mechanisms that remained uncertain. Such a system could act to prevent the motoneurones from responding to descending inputs at 10 Hz.

In this context, it is interesting that, when Raethjen et al. (2002) observed around 10-Hz corticospinal coherence in epileptic patients, it was at a very high level, being larger than 0.6. Such a value compares with studies that have reported 15- to 30-Hz corticospinal coherence in normal subjects, where coherence is usually around 0.1. This would agree with the idea that, in the epileptic patients of Raethjen et al. (2002), some pathological state has disrupted the proposed desynchronizing mechanism, leading to resonance and abnormally strong coherence.

If such a desynchronizing system acting at 10 Hz exists, it might explain the different findings on 10-Hz corticospinal coherence in normal subjects. The extent to which the system is activated could depend on the precise task performed. Thus, in the studies of Marsden et al. (2001) and Gross et al. (2002), some feature of the task, such as contraction strength or muscle groups used, might have led to little activation of the desynchronizing mechanism, and hence allow 10-Hz corticospinal coherence to be seen. By contrast, in other studies the particular constellation of task parameters could have led to the desynchronizing mechanism being more active, producing little or no 10-Hz coherence. This speculative hypothesis clearly requires further investigation.

Synchronization between PTNs

Our previous work using time domain analysis has shown that PTNs are synchronized during the hold phase of the precision grip task (Baker et al. 2001) and that around half of this synchronization is due to frequencies in the 18- to 37-Hz range. Figure 4 indicates that PTN–PTN synchrony during steady holding can also be seen using coherence analysis. There is clearly a component of the synchrony in the middle-frequency band here investigated (17–31 Hz), although there is also considerable coherence at lower frequencies. In addition, the coherence phase was strongly clustered around zero, which agrees with the finding of near-zero lag cross-correlation peaks in time domain analysis. These results therefore broadly agree with our previous work.

The size of the PTN–PTN synchrony deserves some additional comment. Figure 3 indicates that the peak coherence of a single PTN with the LFP was normally <0.05. Linear systems theory shows that, if two PTNs are coherent with the LFP, this should cause them to be synchronized with each other with a coherence equal simply to the product of the two PTN–LFP coherences (Bendat and Piersol 1993). This would predict that PTN–PTN coherence should be almost always lower than 0.052 = 0.0025. Such low values of coherence would require 1,200 trials of data to be recorded before they would rise above the P < 0.05 significance level and would thus never have been detected in our present study. By contrast, however, we often did see significant PTN–PTN coherence, which had a magnitude not very different from the PTN–LFP coherence (compare Fig. 3C–E with Fig. 4C–E).

The solution to this problem lies in the nonlinear behavior of the PTNs shown in the simulations of Figs. 5–7. As noted above, this means that the LFP–PTN coherence values do not represent the fraction of the input to the PTN synchronized with the LFP at a given frequency. The PTN–PTN coherences cannot thus be simply predicted from the product of the LFP–PTN coherences. Further quantitative analysis is likely to be unsound, since it would rely on the PTN model being close to the behavior of a real PTN. However, our data do seem consistent with the idea that a major part of the synchrony between PTN pairs in the beta band is likely to be caused by them both locking to the common oscillatory signals present in the LFP.

APPENDIX

Given a number of simultaneously recorded single neuron spike trains, it is straightforward to calculate the coherence of the summed population activity with the LFP. It is also possible to determine the coherence of each single cell with the LFP. We show here that the single-cell coherence measurements can be used to place upper and lower limits on the population to LFP coherence. How close the actual value is to these limits will depend on the nature of the cell inputs that are uncorrelated to the LFP.

Denote by C the common signals that are measured by the LFP. If s is the activity of a cell, then we assume

\[ s = \lambda C + \epsilon \]  

where all measures are in the frequency domain (the dependence on frequency is suppressed for simplicity of notation). \( \epsilon \) is noise uncor-
related to $C$ and $\lambda$ is a parameter that determines the coherence of the spike activity with the LFP. \textit{Equation A1} assumes a linear relationship between spiking and LFP. This is not correct, as shown in the main part of the paper, since the spike threshold introduces a substantial nonlinearity. However, coherence is a linear systems measure. For the purposes of placing limits on the population coherence estimates, therefore, it is sufficient to use the same linear model as implied by a coherence calculation.

The population activity of $N$ cells can be expressed as

$$S = \sum_{i=1}^{N} s_i$$  \hspace{1cm} (A2)

We wish to find the coherence between the population activity $S$ and $C$

$$\text{coh}(C, S) = \frac{\left| \langle SC \rangle \right|^2}{SS' \cdot CC'}$$  \hspace{1cm} (A3)

This can of course be directly evaluated from the data available. We can also form predictions of what this coherence would be depending on the nature of the noise $\epsilon$ present in each cell.

The power of $S$ is

$$SS' = \sum_{i=1}^{N} \sum_{j=1}^{N} s_i s_j$$  \hspace{1cm} (A4)

which is the sum of all the power and cross-spectra between the $N$ cells. Expanding the cross-spectrum between cells

$$ss'_j = (\lambda C + \epsilon)(\lambda C + \epsilon') = \lambda\lambda'CC' + \epsilon\epsilon'$$  \hspace{1cm} (A5)

$\lambda_i$ can be found from the cross-spectrum between a cell and the LFP

$$sC'C = \lambda C'C$$  \hspace{1cm} (A6)

hence

$$ss'_j = \frac{sC'C}{CC'} + \epsilon\epsilon'$$  \hspace{1cm} (A7)

Consider first the case in which the noise $\epsilon$ is uncorrelated between cells. Then the second term of \textit{Eq. A7} vanishes for $i \neq j$, and from \textit{Eq. A4} we have

$$SS' = \sum_{i=1}^{N} \sum_{j=1}^{N} \frac{ss'_j}{CC'} \hspace{1cm} \text{if } i \neq j$$  \hspace{1cm} (A8)

This estimate for the power of the population cell activity may be inserted into \textit{Eq. A3}. The coherence between the population activity and the LFP so calculated will then be an upper bound—the true value cannot be any higher than this. If there is correlation between the noise in individual cells $\epsilon_i$, this will tend to increase $SS'$ and so reduce the actual coherence measured.

If the noise $\epsilon_i$ is correlated between cells, then from \textit{Eqs. A4} and \textit{A7}, we have

$$SS' = \sum_{i=1}^{N} \sum_{j=1}^{N} \frac{ss'_j}{CC'} + \sum_{i=1}^{N} \sum_{j=1}^{N} \epsilon_i \epsilon'_j$$  \hspace{1cm} (A9)

We can form a lower bound for the population–LFP coherence if we make assumptions that will maximize $SS'$ (\textit{Eq. A3}). This will occur if the noise $\epsilon_i$ is perfectly correlated between all cell pairs, so

$$\text{coh}(\epsilon_i, \epsilon_j) = 1 = \frac{\epsilon_i \epsilon'_j}{\epsilon_i \epsilon'_j}$$  \hspace{1cm} (A10)

Thus

$$|\epsilon_i \epsilon'_j| = \sqrt{|\epsilon_i \epsilon'_j|} |\epsilon_i \epsilon'_j|$$  \hspace{1cm} (A11)

The noise powers $\epsilon_i \epsilon'_j$ may be estimated from \textit{Eq. A7}

$$\epsilon_i \epsilon'_j = \frac{s s'_j}{CC'}$$  \hspace{1cm} (A12)

Note that \textit{Eq. A11} gives only the modulus of the noise cross-spectrum, not its phase. We choose the phase by the following argument. \textit{Equation A9} shows that $SS'$ will be generated by summing both $\epsilon_i \epsilon'_j$ and $\epsilon_i \epsilon'_j$. These will have equal real parts, but imaginary parts with equal magnitude but opposite sign. The sum of these two terms will therefore be maximized if all of their modulus is contained in the real part, i.e., that their phase should be either 0 or $\pi$ radians, corresponding to positive or negative real part and zero imaginary part. Summation across $\epsilon_i \epsilon'_j$ for the different cell pairs will be maximized if they all have the same phase, i.e., are all positive or all negative, not a mixture of both. It is not possible for all pairs to have a phase of $\pi$ radians: if combinations 1 and 2 and 1 and 3 are synchronized with phase of $\pi$, then 2 and 3 must be synchronized with a phase of zero. Hence the greatest value for $SS'$ will be produced if all cross-spectra $\epsilon_i \epsilon'_j$ are assumed to have zero phase. Then

$$SS' = \sum_{i=1}^{N} \sum_{j=1}^{N} \frac{sC'C}{CC'} + \sum_{i=1}^{N} \sum_{j=1}^{N} \sqrt{s s'_j - (\epsilon_i \epsilon'_j)^2} \sqrt{s s'_j - (\epsilon_i \epsilon'_j)^2}$$  \hspace{1cm} (A13)

Inserting this into \textit{Eq. A3} will provide a lower bound for the population spike–LFP coherence, on the assumption that the noise $\epsilon$ is perfectly correlated between the cells.

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