Modulation and coordination of V1 stimulus processing by saccade-driven network activity

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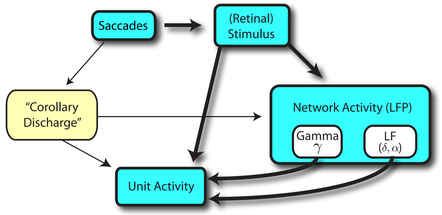
Background

Saccades play a fundamental role in visual processing, beyond the obvious role in directing the gaze. During free viewing, saccades occur on average 3 times a second in primates, and they evoke transient responses in V1 neurons [1-7]. Saccades also modulate V1 local field potentials (LFPs) [3-6], which are known to correspond to fluctuations in cortical excitability on multiple time scales [8-10], and which are themselves modulated by the visual input [13-16]. Thus, these studies suggest a a complex set of interactions between the retinal input, V1 network activity, and single unit activity that is likely important for understanding V1 processing of natural vision.

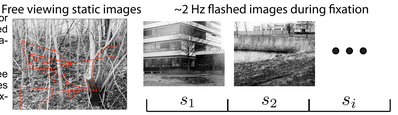
Here, we seek to better understand how saccades shape cortical processing in terms of their effects on V1 'network' activity, coupled with models describing how units are modulated by the stimulus and network activity



Methods

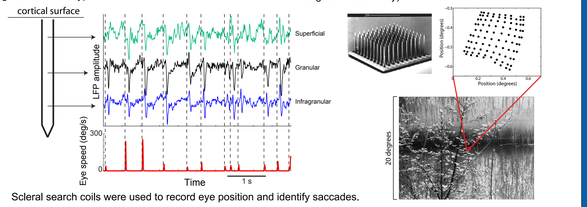
Two monkeys viewed a monitor displaying histogram-equalized natural images (van Hateran database).

Two conditions were used: free viewing and flashed natural images at ~2 Hz while the monkeys fixated.



Two types of multi-electrode arrays were used to record unit activity and LFPs. In one monkey, a 24-channel laminar prob was used to record LFPs from parafoveal V1 (~5 degrees eccentricity).

A 96-channel Utah array was used for recordings in the second monkey. The array was implanted in foveal V1 (~0.5 degrees eccentricity)



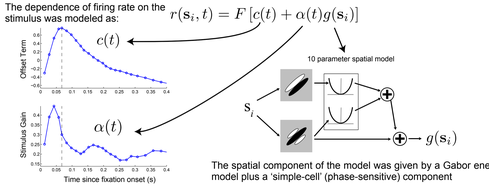
Scleral search coils were used to record eye position and identify saccades.

Time-frequency analysis of the LFPs was done using the continuous wavelet transform with a complex Morlet mother wavelet (with f indexing wavelet scale). Instantaneous phase and amplitude at each scale/frequency were then given by the phase and modulus of $W_f(t)$

$$W_f(t) = y(t) * \psi^{\dagger}\left(\frac{t'-t}{f}\right)$$

The dependence of firing rate on the stimulus was modeled as:

$$r(\mathbf{s}_i, t) = F\left[c(t) + \alpha(t) g(\mathbf{s}_i)\right]$$



The spatial component of the model was given by a Gabor energy model plus a 'simple-cell' (phase-sensitive) component

Two temporal kernels were used to capture the average firing rate and stimulus-response gain as a function of time relative to fixation onset.

The dependence of firing rate on the LFP was modeled as a sum of contributions at each scale/frequency. The contribution of each frequency was modeled as a sinusoidal function of LFP phase with and without multiplication by the LFP amplitude [17]. A second summation across LFP channels was also used to model the effects of multiple LFPs

$$r(t) = F[\textstyle\sum_f \{A(t) a_f \cos(\varphi_f(t) - \phi_f)\} + \cdots]$$

$$a_f \cos(\varphi_f(t) - \phi_f) = b_f \cos(\varphi_f(t)) + c_f \sin(\varphi_f(t))$$

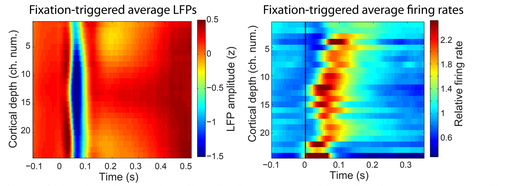
Stimulus modulation of the LFP was quantified using the mutual information between the stimulus and the (frequency resolved) LFP at each time relative to stimulus onset. Information was computed parametrically, assuming normal conditional and marginal distributions.

$$I(Y_t; S) = E_s\{D_{KL}\left(p(y_t|s) \parallel p(y_t)\right)\}$$

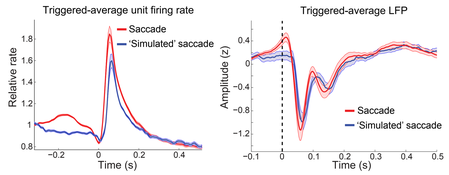
$$= \frac{(\mu_t - \mu)^2 + \sigma_t^2 - \sigma^2}{2\sigma^2} + \log\left(\frac{\sigma}{\sigma_t}\right)$$

Modulation of V1 activity by saccades

Saccades produce a strong modulation of LFPs and unit activity across cortical depth

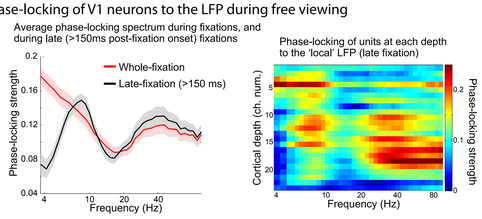


Saccades and 'simulated' saccades elicit similar average LFP and spiking responses in V1

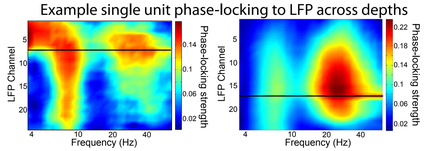


Modulation of V1 neurons by network activity (LFP)

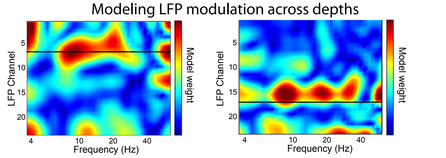
Phase-locking of V1 neurons to the LFP during free viewing



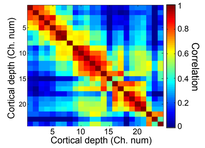
Example single unit phase-locking to LFP across depths



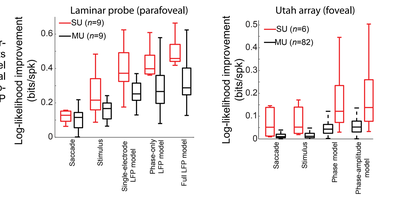
Modeling LFP modulation across depths



Correlations in the predictions of the LFP models between pairs of units can reveal how network activity induces firing rate correlations across cortical depths.

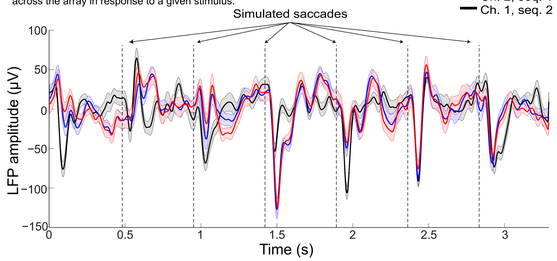


Cross-validated model performance shows large improvements when incorporating single-channel LFP modulation, and an additional large improvement when incorporating modulation by the full LFP depth-profile

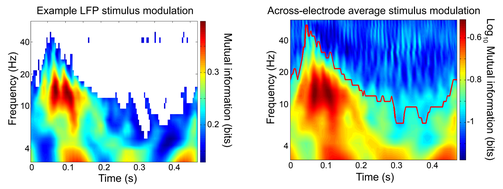


Stimulus modulation of LFPs

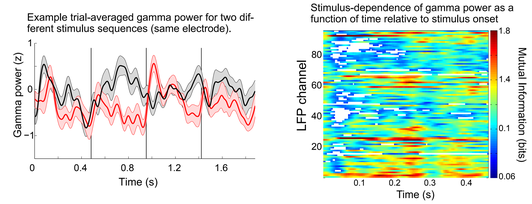
Repeated trials of 'simulated saccade' sequences (average n=25 trials) reveal stimulus-specific components of the LFP responses. Averages LFPs are very similar across the array in response to a given stimulus.



Stimululs modulation of the LFP is measured by decomposing LFP signals into different frequency bands and computing the mutual information with the stimulus at each time relative to stimulus onset. The strongest stimulus modulation is observed for the 10-20 Hz range over the first 150 ms post-stimulus, but significant stimulus modulation is observed for lower frequencies throughout the 470 ms stimulus interval.



While the gamma band LFPs are not phase-locked to the stimulus onset, the amplitude envelope of gamma shows significant stimulus modulation throughout the stimulus interval.



Conclusions

Here we use the LFP in different frequency bands as a proxy for underlying network state. The observed relationship between LFPs and neuronal firing rates suggests an important role of network modulation in structuring neural activity in V1.

-- The relationship between LFP and unit activity during free viewing is most prominent in the alpha (~10 Hz) and gamma bands (30-60 Hz), and mostly at nearby depths, although LFPs at other depths also contribute

-- Low-frequency (<~20 Hz) LFPs in V1 are stimulus (image)-dependent, with stimulus modulation out to 300-400 ms

-- While the LFP in the gamma band is not phase-locked to saccade onset, the envelope of gamma-power shows consistent stimulus modulation throughout the 'fixation' period.

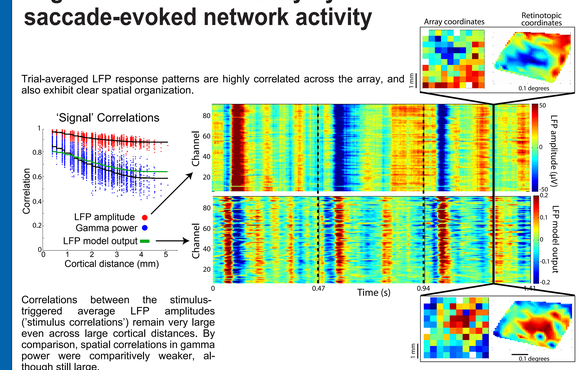
-- Both the stimulus-specific and 'spontaneous' components of LFP activity are highly correlated across the array, with clear spatial structure and correlation length scales of ~1 mm.

-- Using the models of unit modulation by LFPs, both the stimulus-specific and spontaneous components of the LFPs significantly improved predictions of unit activity. The spontaneous component of the LFPs had a much larger effect.

Thus, both stimulus-driven and spontaneous network activity may serve to temporally and spatially organize V1 activity during natural viewing.

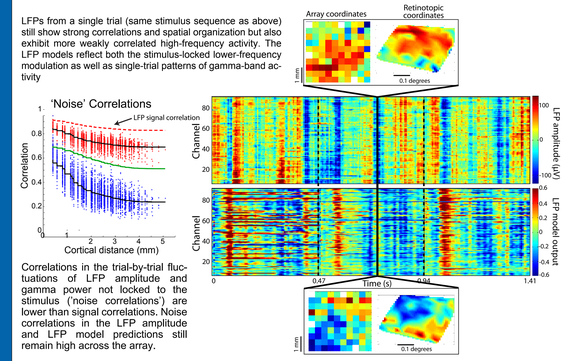
Organization of V1 activity by saccade-evoked network activity

Trial-averaged LFP response patterns are highly correlated across the array, and also exhibit clear spatial organization.



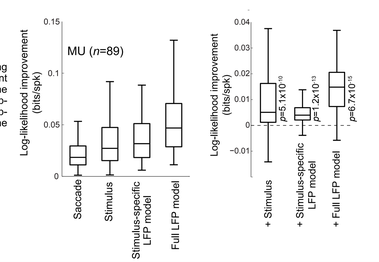
Correlations between the stimulus-triggered average LFP amplitudes ('stimulus correlations') remain very large even across large cortical distances. By comparison, spatial correlations in gamma power were comparitively weaker, although still large.

LFPs from a single trial (same stimulus sequence as above) still show strong correlations and spatial organization but also exhibit more weakly correlated high-frequency activity. The LFP models reflect both the stimulus-locked lower-frequency modulation as well as single-trial patterns of gamma-band activity



Correlations in the trial-by-trial fluctuations of LFP amplitude and gamma power not locked to the stimulus ('noise correlations') are lower than signal correlations. Noise correlations in the LFP amplitude and LFP model predictions still remain high across the array.

The LFP models (fit to a separate training data set) identify a small but significant modulation of unit activity by the stimulus-specific LFP patterns. A substantially larger modulation of units is observed by the trial-by-trial variability in the LFPs [17,18].



References:

[1] Gallant et al., *Neuroreport,* 1998; [2] Gawne & Martin, *J. Neurophys.,* 2002; [3] Rajkai et al., *Cereb. Cortex,* 2008.; [4] Kagan et al., *J. Vision,* 2008; [5] Bosman et al., *J. Neurosci.,* 2009.; [6] Ito et al., *Cereb. Cortex.,* 2011; [7] Ruiz & Paradiso, *J. Neurophys.,* 2012.; [8] Buzsaki & Draguhn, *Science,* 2004; [9] Lakatos et al., *J. Neurophys.,* 2005; [10] Hasenstaub et al., *J. Neurosci,* 2007; [11] Engel et al., *Nat. Rev. Neurosci.,* 2001; [12] Lakatos et al., *Science.,* 2008; [13] Kayser & Konig, *Eur. J. Neurosci.,* 2004; [14] Belitski et al., *J. Neurosci.,* 2008; [15] Montemurro et al., *Curr. Biology,* 2008; [16] Berens et al., *Front. Neurosci.,* 2008; [17] Haslinger et al., *PLoS ONE,* 2012; [18] Kelly et al., *J. Comp. Neurosci.,* 201.