Multiple timescale encoding of slowly varying whisker stimulus envelope in cortical and thalamic neurons in vivo

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Supplemental Material

Fits to amplitude and phase lead: example neurons and goodness of fit

Each neuron’s rate responses to the stimulus envelope were fit for all recorded $T$s. Two example fits from both cortex (Supplemental Figures 1-2) and thalamus (Supplemental Figures 3-4) are shown. For some neurons the resulting phase lead varied as a function of $T$; these neurons did not behave individually as fractional differentiators. A summary of phase lead data for individual neurons is presented in Supplemental Figure 5.

Uniformity of population phase shift over different periods

For populations of cortical and thalamus neurons, we did not find a significant difference in phase leads for $T = 4$-32 s (main text). We also computed phase values relative to those at $T = 4$ s. That is, for each neuron and for periods longer than $T = 4$ s, we computed the difference between the phase advance for that period and its value for $T = 4$ s. We reasoned that if there were any systematic effect of $T$ on phase shifts, it would be apparent as a systematic departure from 0 in these subtracted values. We performed this analysis on the same data shown in Supplemental Figure 5 (see legend): all thalamic neurons and a subset of 13 cortical neurons. For this subset, as for the complete set in the main text, $T$ did not affect the absolute phase lead ($p = 0.93$, Watson-Williams circular ANOVA). The subtracted phase data are shown in Supplemental Figure 6. Consistent with the results in the main text, no systematic effect
of T on phase shifts could be observed (cortical data: \( p = 0.91 \), Watson-Williams; thalamic data: \( p = 0.76 \), Watson-Williams).

**Fitting \( \alpha \) for individual neurons**

In the main text, we found a single \( \alpha \) for each population of neurons using both phase leads and gains, by averaging phase leads or gains across all neurons at each \( T \). An alternative approach involves finding an \( \alpha \) for each neuron using either the best fit phase lead or gain and then averaging over neurons (for a periodic signal, a circular average is appropriate). This approach is not theoretically valid when the response has a frequency dependent phase shift with respect to the stimulus, and it can be difficult to ascertain this for individual neurons, given limited data and noise. Nonetheless, this procedure allows one to compare \( \alpha \) values determined by phase leads to those determined by gain on a neuron by neuron basis. For this analysis we used a subset of neurons which had approximately constant phase, i.e., had a minimal frequency dependent phase shift, and could therefore best be characterized as fractional differentiators. For cortex, we used neurons with at least 3 data points in the range 4-32 s and required that phase leads for those data points be within 15 degrees of their mean phase (8 neurons out of 18). For thalamus, recordings were less noisy and all neurons had phase leads within 15 degrees of their mean phase: we restricted the data set to neurons with phase leads within 10 degrees of their mean phase (5 of 8 neurons).

While this method necessarily used only a subset of data, with neurons being excluded for apparent fluctuations in phase across \( T \), results involving this approach were consistent with the population approach of the main text. For cortical neurons, the mean \( \alpha \) from phase and gain was 0.39 (95% CI 0.30-0.48 from bootstrap samples of the
individual neuron $\alpha$ values; bootstrapping method as in main text) and 0.38 (95% CI 0.14-0.57), respectively. For thalamic neurons, the mean $\alpha$ from phase and gain was 0.21 (95% CI 0.07-0.38) and 0.13 (95% CI 0.03-0.27), respectively.

**Synaptic facilitation can decrease phase leads in a neural network**

Using the same neural network as presented in Figure 5 of the main text, we show that an intermediate synapse with only facilitation can lead to phase lags (i.e. a negative phase lead) and effectively cancel out the effect of neuronal adaptation, which gives rise to phase leads (Supplemental Figure 8).
Supplemental Figure 1: Sinusoidal fits to the envelope responses of example cortical neurons. (a) Fits for an example neuron for $T = 2, 4, 8$, and 16 s. Blue lines
represent the average firing rate over all trials for the given $T$, while in green are the best sinusoidal fits. (b) Sum of squared error as a function of phase and amplitude.

Supplemental Figure 2: Sinusoidal fits to the envelope responses of example cortical neurons. (a) Fits for an example neuron for $T = 2, 4, 8$, and 16 s. Blue lines
represent the average firing rate over all trials for the given $T$, while in green are the best sinusoidal fits. (b) Sum of squared error as a function of phase and amplitude.

Supplemental Figure 3: Sinusoidal fits to the envelope responses of example thalamic neurons. Panels as for Supplemental Figure 1. Responses were recorded for $T = 1, 2, 4, 8, 16$, and 32 s.
Supplemental Figure 4: Sinusoidal fits to the envelope responses of example thalamic neurons. Panels as for Supplemental Figure 1. Responses were recorded for $T = 1, 2, 4, 8, 16$, and $32$ s.
Supplemental Figure 5: Summary data showing phase leads from best fits to envelope responses. Top, cortical data; bottom, thalamic data. Points at each $T$ are staggered for clearer display. Each line (different colors) corresponds to an individual neuron. Error bars represent 95% confidence intervals on the best fit phase lead from 300 bootstrap repeats of the fitting procedure on a randomly selected fraction (50%) of the data. Thus, the confidence intervals reflect the reliability of the fit for each neuron and $T$. Data are for the full set of thalamic neurons ($n = 8$) and for a subset of cortical neurons ($n = 13$). The full set of cortical neurons is not displayed because cortical recordings only included 4 out of the 6 possible periods, $T$; the present subset included all neurons with recordings for $T = 4$, 8, and 16 s.
Supplemental Figure 6: Differences in best-fit phase shifts across $T$ for individual neurons. Top, cortical data; bottom, thalamic data. Each line (different colors) corresponds to an individual neuron and shows the differential phase shift values across $T$, that is, the difference between the best fit phase value for each $T$ and the value for $T = 4$ s. While for individual neurons phase shifts may vary across $T$, at the population level this effect was not significant.
Supplemental Figure 7: Scatter plot of phase- and gain-based estimates of the order of differentiation, $\alpha$, determined on a neuron-by-neuron basis. Phase and gain estimates of $\alpha$ were found for a subset of cortical ($n = 8$) and thalamic ($n = 5$) neurons, as described above. Gain- and phase-based fits agreed well at the population level though not always at the individual neuron level, suggesting that, while slow envelope encoding by small populations is well described by the order of fractional differentiation, individual neurons can be characterized as fractional differentiators only to varying degrees.
Supplemental Figure 8: Neural network output showing the effect of synaptic facilitation. A neural network constructed as in the main text and shown in Figure 5. In all three cases, the initial and final neuronal layers contain adapting neurons that are approximately fractional differentiators; only synaptic parameters are altered. The synapse included depression and facilitation as in the main text (diamonds), no plasticity (circles), and only facilitation with $F$ increased by 0.1 and a time constant of 3 s (squares). With facilitation only, the synapses can counteract the adaptation of the initial and final neural layers, even leading to an overall phase lag in $T = 8$. 