

# Dynamics of population rate codes in ensembles of neocortical neurons

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**Abstract**

Information processing in neocortex can be very fast (Thorpe et al., 1996), indicating that neuronal ensembles faithfully transmit rapidly changing signals to each other. Apart from signal-to-noise issues, population codes are fundamentally constrained by the neuronal dynamics. In particular, the biophysical properties of individual neurons and collective phenomena may substantially limit the speed at which a graded signal can be represented by the activity of an ensemble. These implications of the neuronal dynamics are rarely studied experimentally. Here, we combine theoretical analysis and whole-cell patch-clamp recordings to show that encoding signals in the *variance* of uncorrelated synaptic inputs to a neocortical ensemble enables faithful transmission of graded signals with high temporal resolution. In contrast, encoding signals in the mean current is subjected to low pass filtering.

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**Keywords:** signaling by variance, neuronal decorrelation, irregularity of neuronal discharge

## INTRODUCTION

The firing rate of many neurons in cortex is known to depend on various aspects of stimuli in a smooth way. While this finding suggests that the graded rate of an individual neuron is used to distinguish between different stimuli, it can be estimated only after a sufficiently large number of spikes have occurred. For this reason reliable rate signals are necessarily slow, if obtained from single neurons, and hence, cannot account for the rapid information processing observed in the cortex (Thorpe et al., 1996). In contrast, at the level of populations, this problem of signal-to-noise can be overcome, such that the *population rate* of an ensemble of neurons (i.e. the average number of spikes in the population per time interval) can be estimated on a time scale that is even smaller than the interspike intervals of the individual neurons. If the neuronal responses are statistically independent from each other, the achievable time resolution will in fact depend only on the total number of neurons in the ensemble: the larger the ensemble, the higher the temporal precision with which the population rate can be estimated. While it is possible to overcome the limitations of temporal precision due to noise by using an increasing number of neurons, there is another constraint on the speed of signal transmission caused by the neuronal dynamics: intrinsic properties of individual neurons like the membrane time constant, and population effects like synchronization can severely limit the ability of neuronal ensembles to realize rapid rate codes (Knight, 1972). Therefore, we here investigate to which extent rapid transmis-

sion of graded rate signals between populations of cortical neurons rely on the encoding strategy due to the neuronal population dynamics.

In contrast to the usual characterization of a signal by the *temporal* mean and the variance components we here consider the instantaneous distribution of input currents into the neurons of a functional ensemble at each moment in time. In this case the synaptic inputs can also be divided into two components: one component is given by the input averaged over the ensemble; the other component is given by the deviations of individual inputs from the average: the population variance. Both components can in general fluctuate with time, contributing to the observable fluctuations in the synaptic currents of single neurons. The output of the ensemble can be described by an instantaneous '*population rate*', estimated by the number of spikes emitted by the entire ensemble in small time intervals divided by the number of neurons. The population rate depends on the amplitudes of both components of the input. This reasoning allows one to conclude that signals delivered to a neuronal ensemble could in principle be carried by (encoded in) either the common, correlated, part of the synaptic inputs to the neurons, or the variance of the inputs across the population, or in both.

In this study we examine the respective implications of these coding strategies for rapid and reliable transmission of information between neuronal ensembles. We show that correlated input currents cannot be used to transmit rapidly changing signals, while encoding the signal in the variance enables faithful

signal transmission in the population rate on a millisecond time scale irrespectively of the membrane time constants of the neurons.

## METHODS

### *General methods*

*INTEGRATE-AND-FIRE MODEL.* The leaky integrate-and-fire neuron is characterized by its membrane potential, whose dynamics is described by the circuit equation (Lapicque, 1907; Tuckwell, 1988):

$$\tau \frac{dV}{dt} = -(V - V_{rest}) + R_{in} I_{syn}, \quad (1)$$

where  $\tau$  denotes the membrane time constant,  $V_{rest}$  is the membrane resting potential,  $I_{syn}$  is the synaptic current, and  $R_{in}$  is the input resistance of a neuron. This equation is supplemented by the condition that each time the membrane potential hits the threshold potential,  $V_{th}$ , a spike is emitted and the membrane potential is instantaneously reset to a certain sub-threshold level,  $V_{reset}$ .

In order to achieve an analytical understanding of the population dynamics, we consider an infinite population of identical integrate-and-fire neurons indexed by  $i$ , receiving input currents of the form

$$I_i(t) = \mu(t) + \sigma(t)\eta_i(t), \quad (2)$$

where  $\mu(t)$  denotes the mean input across the population at time  $t$ ,  $\eta_i(t)$  is Gaussian white noise with unit spectral density, and  $\sigma(t)$  is a scaling factor measuring how strongly the individual input currents deviate from the mean. Rigorously speaking, Gaussian white noise does not exist in nature, but it is understood as a reasonable idealization that together with Eq. (1) becomes a well defined mathematical term in the sense of Langevin equations.

Correspondingly, the time evolution of the population density function  $P(V, t)$  then obeys the Fokker-Planck equation:

$$\frac{\partial P(V, t)}{\partial t} = -\frac{\partial J(V, t)}{\partial V}, \quad (3)$$

where the probability flux  $J(V, t)$  is given by

$$J(V, t) = \frac{R_{in}\mu(t) - V(t)}{\tau} P(V, t) - \frac{R_{in}^2\sigma(t)^2}{2\tau^2} \frac{\partial P(V, t)}{\partial V}. \quad (4)$$

The flux consists of two components: the first term is the drift component, which is governed by the mean  $\mu(t)$ , and the second term is the diffusion component representing the effect of random fluctuations. The firing threshold and the reset potential impose boundary conditions on the flux, which imply  $P(V_{th}, t) \equiv 0$  for all  $D = \frac{\sigma(t)^2}{2} > 0$  (Tsodyks and Sejnowski, 1995; Risken, 1984). Therefore, at threshold, the only contribution to the flux is given by the diffusion component.

### *Data collection*

*INTRACELLULAR RECORDINGS.* Parasagittal slices 300  $\mu\text{m}$  thick were obtained from Wistar rats (14-16 days old). Slices were incubated for 30 minutes at 32-34°C before being transferred to the recording chamber. The recordings were made at 32-34°C. Neurons were selected for recording according to the morphology of the soma and proximal dendrites, as visualized by IR-DIC optics using a Zeiss Axioscope and Hamamatsu CCD camera. The bathing solution consisted of (in *mM*): NaCl 125, NaHCO<sub>3</sub> 25, glucose 25, KCl 2.5, CaCl<sub>2</sub> 2, NaH<sub>2</sub>PO<sub>4</sub> 1.25, MgCl<sub>2</sub> 1. Whole-cell recordings were made using patch pipettes (5-10M $\Omega$ ), containing (in *mM*) K-gluconate 110, KCl 10, HEPES 10, phosphocreatine(Na) 10, MgATP 4, NaGTP 0.3 and biocytin 2%. Recorded voltage and current were amplified by axopatch 200/B amplifiers (Axon instruments). Acquisition was done using IGOR-Pro software (WaveMetrics, inc.). Injected current was calculated for each individual injection using random values drawn from a Gaussian distribution. The values for  $\tilde{\mu}$  and  $\tilde{\sigma}^2$  were determined according to the discharge rate of the stimulated neuron. Current recordings used for stimulation were obtained in voltage-clamp mode and inverted before injection. Activity in the slice was induced by a gradual application of 4AP, a blocker of transient potassium current.

## RESULTS

It is instructive to first analyze a simplified model, where the issue of population coding can be addressed in a mathematically rigorous way. We consider an (infinitely) large population of identical integrate-and-fire neurons indexed by  $i$  (see Methods), each receiving fluctuating synaptic inputs of the following form:

$$I_i(t) = \tilde{\mu}(t) + \tilde{I}_i(t). \quad (5)$$

Here  $\tilde{\mu}(t) := \frac{1}{N} \sum_{k=1}^N I_k(t)$  stands for the instantaneous amplitude of the averaged, correlated part of the input (common to all the neurons), while  $\tilde{I}_i(t) := I_i(t) - \tilde{\mu}(t)$  are the deviations of individual inputs from the average (unique for every neuron). A global measure for the time-dependent input diversity is given by the instantaneous *population variance* of the input  $\tilde{\sigma}^2(t) = \frac{1}{N} \sum_{k=1}^N \tilde{I}_k^2(t)$ . In general, both of the variables,  $\tilde{\mu}(t)$  and  $\tilde{\sigma}^2(t)$ , change in time, and thereby can serve as signals carrying information from presynaptic populations. The main goal of the analysis is to estimate how the activity of the population reflects the signals carried by either variable. In particular, we are interested in the conditions under which the instantaneous population rate will faithfully follow the analog value of the signal *at any time* ('perfect signaling').

The analysis can be pursued to the ultimate solution, if  $\tilde{I}_i(t)$  are mutually independent and temporally uncorrelated random processes (i.e. Gaussian white noise; see methods). In that limiting case  $\tilde{\sigma}^2$  diverges, because of the vanishing correlation time. It is understood, however, that if the correlation time  $\tau_c$  of a real current is sufficiently small (i.e. clearly smaller than the membrane time constant) essentially only the product  $\sigma^2 = \tilde{\sigma}^2 \cdot \tau_c$  is relevant. For the sake of convenience, we will therefore call  $\sigma^2$  somewhat incorrectly a “*population variance*”, too, in the following.

Since the momentary state of an integrate-and-fire neuron is defined by only one variable - its membrane potential  $V(t)$ , the state of the whole population is completely characterized by a probability density function  $P(V;t)$ , i.e. the fraction of neurons with membrane potential close to  $V$ . The time evolution of the density function is governed by both the average and the fluctuating components of the inputs via the so-called Fokker-Planck equation (Risken, 1984; see Methods).

One can visualize this formulation by considering a collection of point-like particles moving independently along a one-dimensional axis under the combined influence of the deterministic force and a random, diffusing force which tends to equilibrate the particles along the axis. In this analogy,  $P(V)$  is just the density of particles on the axis of  $V$ .

The advantage of this approach is that one can derive an exact expression for the instantaneous firing rate of a population of neurons, which is given by the flux of particles to the firing threshold  $V_{th}$ :

$$R(t) = -\frac{R_{in}^2 \sigma(t)^2}{2\tau^2} \frac{\partial P(V; t)}{\partial V} \Big|_{V=V_{th}} \quad (6)$$

The instantaneous population rate  $R(t)$  is defined here as the number of spikes emitted by the whole population in an (infinitely) small time bin around time  $t$ , normalized by the duration of the bin. Importantly, if the uncorrelated noise has a positive amplitude, only the diffusion component of the flux (see Methods) is contributing to the firing rate, since in this case the density of particles at the threshold is zero. The above formula, together with the Fokker-Planck equation, represents a complete characterization of the population response in its dependence on the parameters of the input. In a stationary situation when  $\mu$  and  $\sigma^2$  are constants, the density function adjusts its shape to the values of these parameters. The output rate, as stated above, is therefore a function of both parameters (Roy and Smith, 1969; Tuckwell, 1988).

In the general situation, when the signals depend on time, the density function  $P(V; t)$  evolves according to the Fokker-Planck partial differential equation (see METHODS), thereby exhibiting a low-pass filtered response to changes in the signals  $\mu(t)$  and  $\sigma^2(t)$  with a filter whose parameter depends on the membrane time constant of the neurons in a population (Brunel and Hakim, 1999). This means that the output rate  $R(t)$  given by Eq. (6) depends not

only on the current values of the input parameters, but also on their previous values, i.e. the instantaneous population rate does *not* faithfully reflect the instantaneous signals. However, if the modulations of the input signals around some baseline values are much faster than the membrane time constant, the shape of  $P(V; t)$  will be almost stationary, due to the above mentioned filtering property of the Fokker-Planck Equation.

To verify that the density function is indeed stable to very fast modulations of the input signals, we performed numerical simulations of the activity of a large population of integrate-and-fire neurons receiving noisy input currents with an instantaneous mean value oscillating in time with increasing frequency. The time evolution of the density function was then computed from the results of the simulations and its stability was assessed. In Fig. 1A, B we show a sample of probability density functions across the population computed over subsequent 1 ms bins for two different values of the frequency. Obviously, the variations in the density function are smaller for higher frequency. To quantify this result, we plot on Fig. 1C the standard deviation of the density function averaged over all positive values of the voltage, as a function of the frequency of the signal. For very high frequency the standard deviation reduces to a low residual value that is explained by the finite size of the neuronal population. Qualitatively similar results were obtained for oscillating instantaneous variance in the input current (results not shown).

This result, together with Eq. (6), implies that the output population rate  $R(t)$  will be proportional to the quickly changing instantaneous values of the population variance of the uncorrelated component of the inputs,  $\sigma^2(t)$ , and ignores the modulation in the mean. In other words, the population can faithfully transmit rapid signals, if they are encoded in the amplitude of the uncorrelated 'noise'.

Intuitively, this effect can be understood most easily in a population of neurons simultaneously receiving excitatory and inhibitory inputs. An excess of excitation at a given time will drive the neurons towards threshold. Some neurons will be caused to fire and immediately afterwards will be synchronously refractory. In order to achieve a rapid response, the excitatory pulse would have to be huge, which in turn would temporarily saturate the population activity. Therefore the analog signal cannot be transmitted this way since the population activity will not be able to remain constantly at the intermediate values dictated by the signal. If, in contrast, the inhibition is increased simultaneously together with the excitation this implies an increased population variance of the inputs. In this case only a fraction of the neurons will receive big excitatory pulses and thereby respond rapidly, while a large proportion will be less affected or driven away from threshold. In this way the balance of excitation and inhibition randomly selects changing subsets of neurons that are driven across threshold. This mechanism avoids population saturation and the number of firing neurons at each moment in time (population rate) faithfully

reflects the graded signal encoded in the population variance of the input.

Because this theoretical prediction could be of fundamental importance for the issue of the neural code in neocortex, we undertook a series of experiments to test its validity (Methods). Experimental test is clearly warranted due to the many simplifications in the mathematical model, most notably the neglect of the kinetics of ionic channels.

We prepared an ensemble of virtually white noise current traces (see caption Fig.2) characterized by particular  $\mu(t)$  and  $\sigma^2(t)$  and injected them into neocortical neurons while monitoring their spiking response. Ideally we would then proceed to estimate the instantaneous firing rate of a large neuronal population with every neuron receiving a different current trace from the prepared ensemble. Since intracellular recordings from so many neurons would be extremely time consuming, we instead repeatedly injected into single neurons, but every time choosing a *different* current trace from the same ensemble. The 'population' activity can then be estimated by computing the average number of spikes emitted by the neuron in subsequent time bins of 1 ms, i.e. a so called peri-stimulus-time-histogram (PSTH). Substituting the PSTH for the population firing rate can be justified only if every neuron will exhibit the same time course of the PSTH response (up to a possible scaling factor), since in this case subsequent responses obtained in single neurons would accurately represent different neurons in a population.

In our first series of experiments we compared the response to two simple forms of input signals, for which either  $\mu(t)$  or  $\sigma^2(t)$  increases abruptly at a particular time (Fig. 2A, B). Eq. (6) predicts that there should be a gradual response to the jump in  $\mu$ , and an instantaneous initial response to a change in  $\sigma^2$ , since the latter enters as a multiplying factor in the expression for the response. The amplitudes of the signals were calibrated such that the steady-state level of a neuron's firing was identical in both cases, because this implies that the average firing rates are almost identical in both cases, too. However, the time profile of the PSTH in response to abrupt increase in  $\mu$  and  $\sigma^2$  was very different: while in the first case, there was a gradual change in the PSTH, in the second case we observed an instantaneous initial response, with subsequent decay to the new stationary level. These results are in agreement with the theoretical prediction (Eq. (6)).

Similar results were obtained in all 18 of a wide variety of neocortical neurons, which included pyramidal neurons and different types of interneurons. We emphasize that in both cases, the output rate continued to change after the transition, when the input signal (the values of either  $\mu$  or  $\sigma^2$ ) stayed constant at a different level. This means that the instantaneous population rate did not follow the step in the signal amplitude when this was modulating the mean current to the population. On the other hand the rate response faithfully followed the step in the population variance  $\sigma^2$ , but was then also slowly changed thereby decreasing to the steady-state discharge level. Note that in

both cases the responses did NOT faithfully reflect the overall shape of the single step and showed temporal modulations, which are also a prediction of the theory presented above.

We then proceeded to test our main theoretical prediction by studying the population response to signals that undergo rapid *ongoing* changes in time. To this end, we injected the same type of current forms as in the first experiment, while now both,  $\mu(t)$  and  $\sigma^2(t)$  are rapidly fluctuating all the time. In other words, the two signals were present simultaneously in the input. In order to separate the effectiveness of these two signals, we constructed  $\mu(t)$  and  $\sigma^2(t)$  by randomly assigning new values to each of them independently at every millisecond drawn from uniform distributions (see Fig. 3A,B). The ranges for both kinds of input signals were calibrated for each neuron individually, such that the corresponding output ranges of the firing rates were of the same size (i.e. the calibration makes sure that the stationary firing rate for constant  $\mu, \sigma^2$  is identical in both cases,  $(\mu = \mu_{min}, \sigma^2 = \sigma_{max}^2)$  and  $(\mu = \mu_{max}, \sigma^2 = \sigma_{min}^2)$ ).

The spiking PSTH response to these currents was obtained (Fig. 3C). Strikingly, the instantaneous population rate reliably followed the signal carried by the variance of the input currents, and there was no observable correlation with the signal contained in the mean (Fig. 3F). To quantitatively test the prediction contained in Eq. (6) about the dependency of the response on the components of the input, we plotted the instantaneous values of the PSTH response vs the instantaneous variance of the currents (Fig 3E). In agreement

with the prediction, the points in this graph scatter around the straight line passing through the origin. The same graph, but with the signal contained in the average current  $\mu$  does not result in any significant dependency (Fig. 3D), again following the theoretical prediction. Very similar results were obtained in all 4 pyramidal neurons which were tested. We therefore conclude that substituting the PSTH responses obtained in single neurons for instantaneous population rate of large neuronal populations is justified, and that the instantaneous population rate reflects the signal carried by the rapidly changing instantaneous variance of the input.

The above experiments confirm that signaling with variance applies to the neurons in cortex and does not rely on the particular choice of the model neuron used in the theoretical analysis. However, because in these experiments the injected currents were artificial and in particular, did not correctly reflect the temporal correlations of real input currents, we in addition obtained realistic synaptic currents from whole-cell voltage-clamp recordings in cortical slices with different levels of excitation (Methods). With these currents we performed a control experiment, by injecting them into a neuron and recording the discharge responses for both, step changes in mean current and step changes of the variance.

As in the first experiments, the discharge response increased gradually when we injected recorded currents containing abrupt changes in the mean current (Fig. 4A), whereas the change in discharge was much faster when the variance

of the current increased abruptly (Fig. 4B). These results demonstrate that the difference between *signaling by variance* and signaling by mean persists with real synaptic noise currents (c.f. Brunel et al., 2001). In particular, signaling by variance does not require white noise currents, but is possible with the synaptic currents generated in neocortex.

Finally, in this study we did not consider the effects of changing neuronal conductances, which would result from the barrage of excitatory and inhibitory synaptic inputs. Experimentally, this issue could be addressed by employing the dynamic clamp technique, which remains a challenge for future studies. In a series of simulations (results not shown) with populations of integrate-and-fire neurons receiving inputs in the form of noisy conductance changes, we found qualitatively the same effects as presented in this paper.

## DISCUSSION

Cortical neurons in vivo fire irregularly (Softky and Koch, 1993) and in an apparently irreproducible manner (Schiller et al., 1976; Vogels et al., 1989). As a possible explanation, this phenomenon has been suggested to originate from a balance of excitatory and inhibitory synaptic inputs (Gerstein and Mandelbrot, 1964; Shadlen and Newsome, 1995), in which case the mean becomes small and signaling by variance is particularly relevant. The resulting variability, however, if considered noise, clearly impairs the precision of rate estimates.

This raises the question whether there is a functional reason that may justify to take this loss. To this puzzle, our study contributes a novel, independent argument demonstrating that in case of neocortical pyramidal neurons, signaling by variance, in contrast to the mean, allows for rapid population rate codes. This result does not rely on synaptic connectivity within an ensemble, but reflects the basic biophysical properties of populations of neocortical neurons. It could be generated either by the dynamics of a network in which the neurons are embedded, or by the activity of presynaptic ensembles. Indeed, network models have been developed, whose activity can rapidly follow the input (Battaglia and Treves, 1998; vanVreeswijk and Sompolinsky, 1996; Tsodyks and Sejnowski, 1995; Abbott and vanVreeswijk, 1993). In conclusion, our work suggests that signaling by variance may be important as being a simple mechanism for desynchronizing neuronal populations, which is necessary for the realization of rapid population rate codes. Future experiments are required to clarify, whether this is indeed the case.

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## FIGURE LEGENDS

### Figure 1:

Simulations of the probability density function evolution in response to fast changes in input mean. A population of 20000 integrate and fire neurons with  $\tau = 10$  ms,  $V_{th} = 1$ ,  $V_{rest} = V_{reset} = 0$  are simulated (see Methods), with constant input variance  $\sigma = 0.3$  and  $\mu(t)$  oscillating between the values of 0.4 and 0.9, applied for 200 ms. (A) Overlaid probability density functions computed for subsequent 1 ms time bins, where  $\mu(t)$  oscillates with a frequency of 50 Hz. (B) Same as A, but now  $\mu(t)$  oscillates with a frequency of 1000 Hz. (C) The standard deviation of the density function averaged over positive values of voltage, plotted as a function of the frequency.

### Figure 2:

Response of neocortical neurons to abrupt changes in input parameters  $\mu$  and  $\sigma^2$ . In each case 4000 different virtually white noise current traces (sampling interval in all of the experiments is  $\tau_{sample} = 0.25$  ms) were injected into a pyramidal neuron sequentially. (A) Lower trace, an example of a single current trace injected. The arrow indicates the moment at which the amplitude of the mean current was increased. Middle trace, histogram of the "population" response with a time bin of 1 ms. At the transition point, the mean current was increased from 120 pA to 200 pA. Upper trace - raster plot of spike trains for 10 randomly chosen trials. Solid red lines show the stationary levels of the

response before and after the transition. (B) Same as in A, but the set of current time-courses featured a change in the population variance  $\sigma^2 = \tilde{\sigma}^2 \tau_{sample}$  from 22.5 (pA)<sup>2</sup>sec to 90 (pA)<sup>2</sup>sec. Histogram binning is 1 ms.

**Figure 3:**

Fast signaling by current variance. The response of a pyramidal neuron to virtually white noise current injection ( $\tau_{sample} = 0.25$  ms) in which two temporally uncorrelated signals are encoded. (A) Instantaneous values of mean current. (B) Instantaneous values of population variance. (C) Instantaneous response (PSTH) in time bins of 1 ms. For every time bin, the PSTH was computed from the spiking responses of a neuron to repeated injections of different current traces as explained in the text. (D+E) Instantaneous response plotted vs the instantaneous value of  $\mu$  and  $\sigma^2$ , respectively, after compensating for a 1 ms delay. Every point on the graph represents a pair  $(\mu(t), \text{PSTH}(t))$  or  $(\sigma^2(t), \text{PSTH}(t))$  for consecutive time bins. (D) Correlation coefficient  $R = 0.15$ . (E) Red line is a linear regression. The sample correlation coefficient is  $\rho = 0.79$  and the sample correlation ratio is 0.65, which is very close to  $\rho^2$  and hence confirms the linear relationship (Stuart and Ord, 1994). Overall, 5600 current traces were injected (i.e. the observed correlation reflects the precision that would be achieved in a population at a time scale of 1 ms in a population of about 5000 neurons. (F) The cross-correlation values between the "population" response and the signals carried by the mean and the variance of the

input currents.

**Figure 4:**

Response of neocortical neurons to currents obtained from voltage-clamp experiments. (A) Lower trace, an example of a segment of a current obtained in a voltage-clamp recording from a slice with low activity level (see Methods). At the transition time, a constant value was added to the current. Upper trace, histogram of 4000 responses to different current segments. (B) Same as in A, but with a step change in the variance of the current. This was achieved by switching to current traces, recorded at higher activity level.

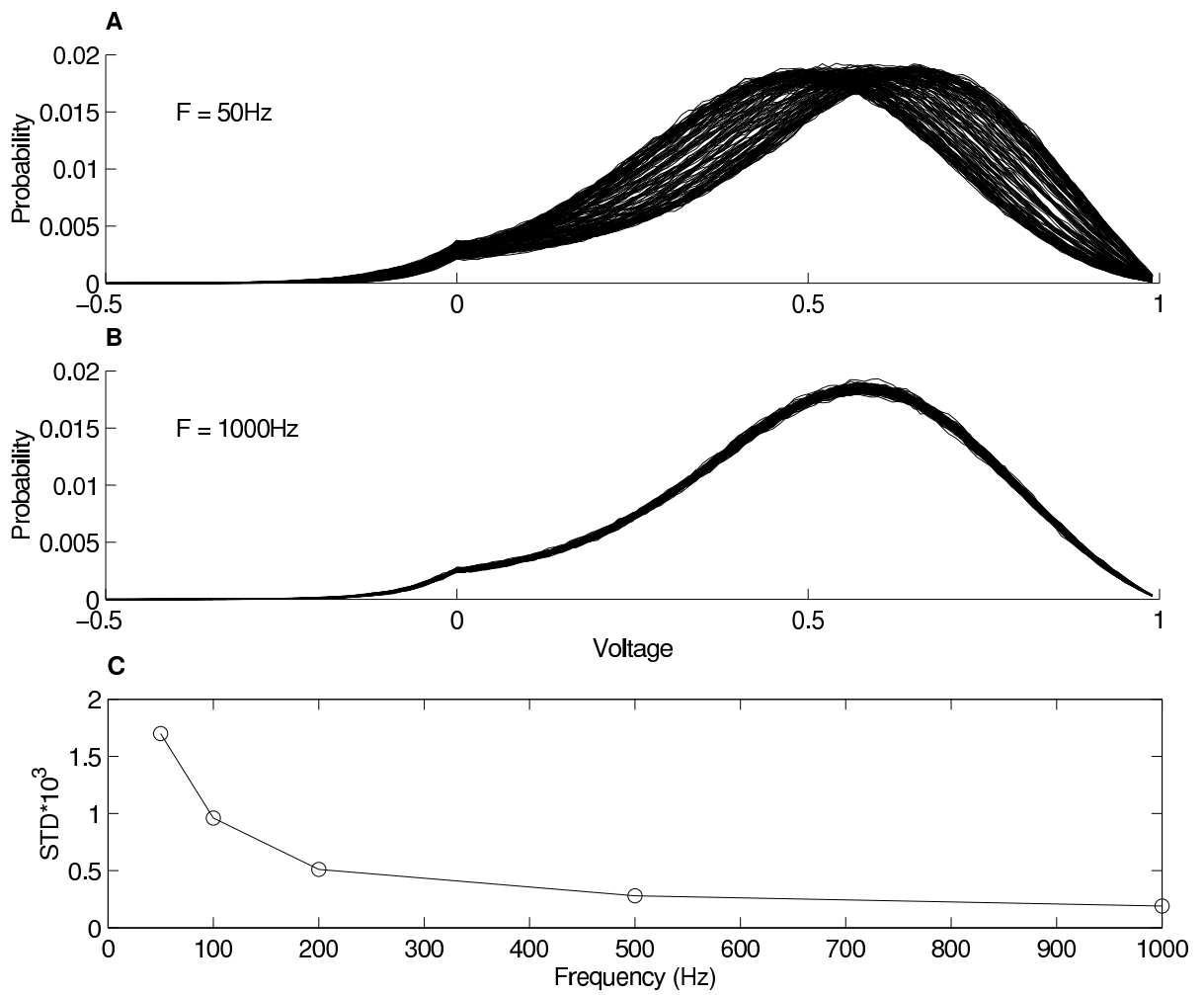


Figure 1: Silberberg et al

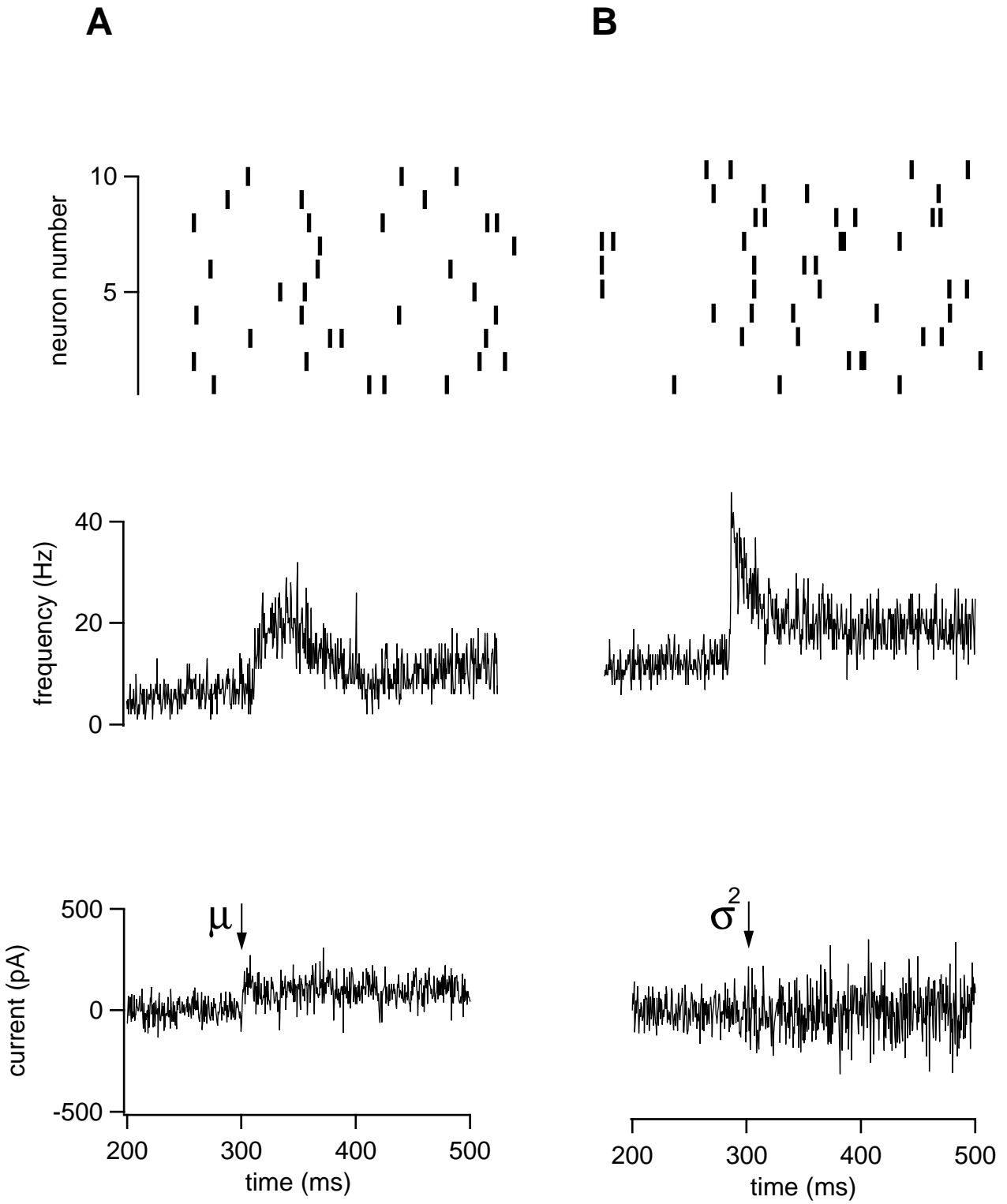


Figure 2: Silberberg et al

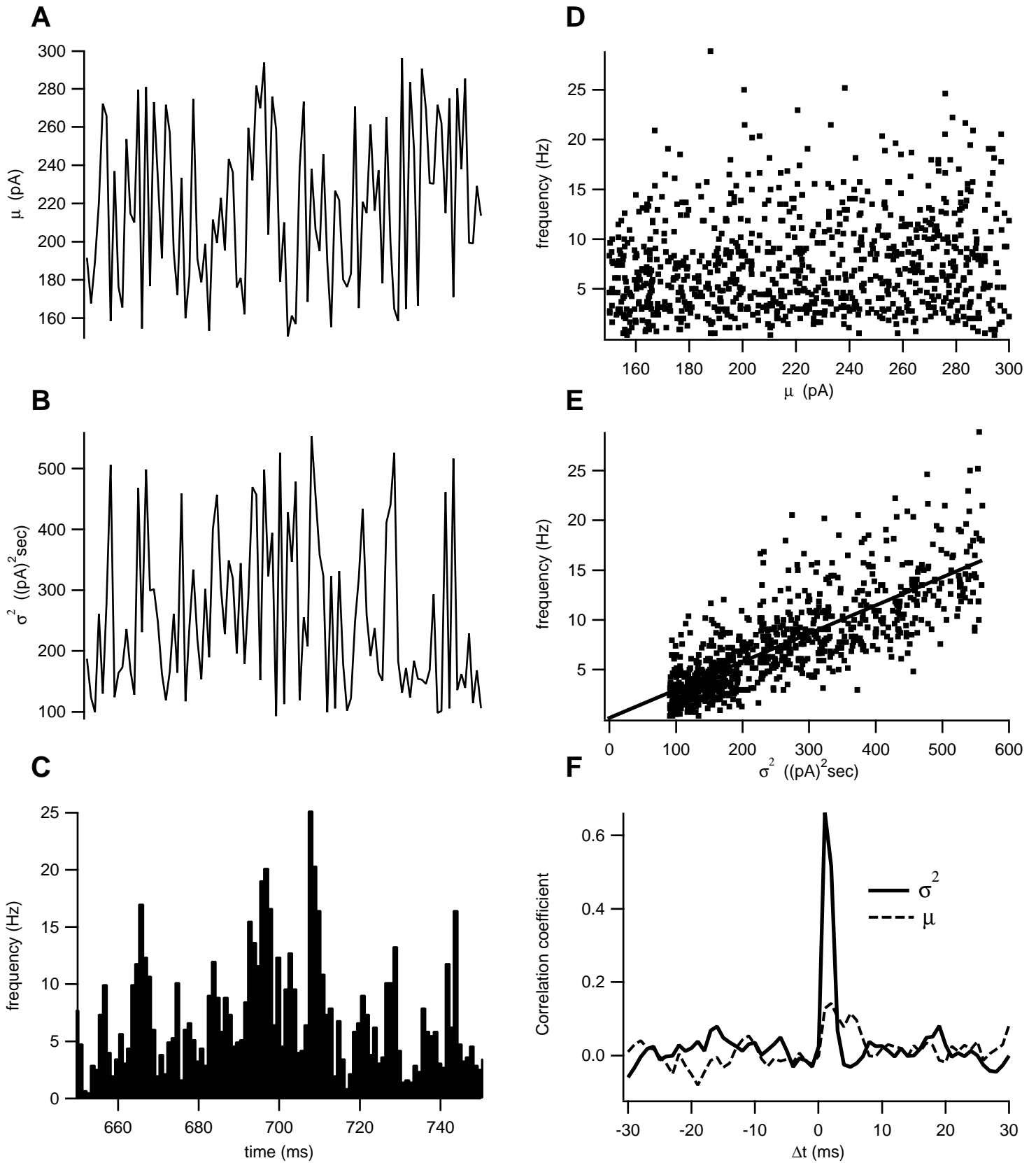


Figure 3: Silberberg et al

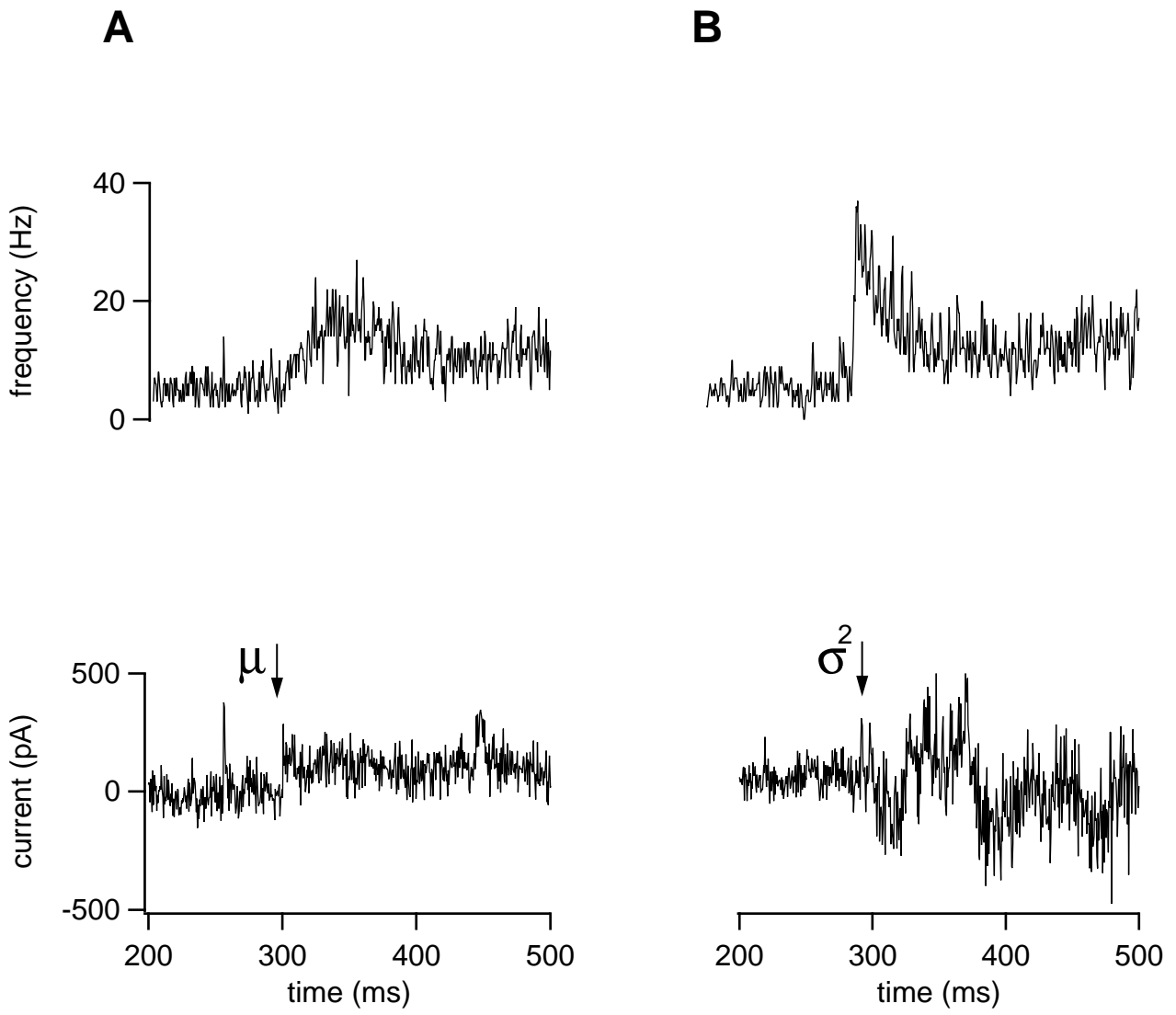


Figure 4: Silberberg et al