

From the statistics of connectivity to the statistics of spike times in neuronal networks

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An essential step toward understanding neural circuits is linking their structure and their dynamics. In general, this relationship can be almost arbitrarily complex. Recent theoretical work has, however, begun to identify some broad principles underlying collective spiking activity in neural circuits. The first is that local features of network connectivity can be surprisingly effective in predicting global statistics of activity across a network. The second is that, for the important case of large networks with excitatory-inhibitory balance, correlated spiking persists or vanishes depending on the *spatial scales* of recurrent and feedforward connectivity. We close by showing how these ideas, together with plasticity rules, can help to close the loop between network structure and activity statistics.

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Introduction

Here, we focus on relating network connectivity to collective activity at the level of spike times, or *correlations* in

neurons' spike trains (see [Box 1](#)). Such correlations are known to have complex but potentially strong relations with coding in single neurons [1] and neural populations [2–5], and can modulate the drive to a downstream population [6]. Moreover, such correlated activity can modulate the evolution of synaptic strengths through spike timing dependent plasticity (STDP) ([7,8^{**},9], but see [10]).

Collective spiking arises from two mechanisms: connections among neurons within a population, and external inputs or modulations affecting the entire population [11–13]. Experiments suggest that both are important. Patterns of correlations in cortical micro-circuits have been related to connection probabilities and strengths [14]. At the same time, latent variable models of dynamics applied to cortical data have revealed a strong impact of global inputs to the population [15,16^{**},17,18].

At first, the path to understanding these mechanisms seems extremely complicated. Electron microscopy (EM) and allied reconstruction methods promise connectomes among thousands of nearby cells, tabulating an enormous amount of data [19–25]. This begs the question of what *statistics* of connectivity matter most — and least — in driving the important activity patterns of neural populations. The answer would give us a set of meaningful 'features' of a connectome that link to basic statistical features of the dynamics that such a network produces. Our aim here is to highlight recent theoretical advances toward this goal.

Mechanisms and definitions: sources and descriptions of (co)variability in spike trains

Neurons often appear to admit spikes stochastically. Such variability can be due to noise from, for example, synaptic release [28], and can be internally generated via a chaotic 'balanced' state [29,30,31^{**}]. As a consequence, the structure of spike trains is best described statistically. The most commonly used statistics are the instantaneous firing rate of each neuron, the autocorrelation function of the spike train (the probability of observing pairs of spikes in a given cell separated by a time lag s), and the cross-correlation function (likewise, for spikes generated by two different cells). As shown in [Box 1](#), even weak correlations yield coherent, population-wide fluctuations in spiking activity that can have a significant impact on

Box 1 Spike train statistics.

The spike train of neuron i is defined as a sum of delta functions, $y_i(t) = \sum_k \delta(t - t_i^k)$; t_i^k is the time of neuron i 's k th spike. Spike train moments can be obtained from samples of the spike trains of each neuron in a population. The first spike train moment is the instantaneous firing rate, $\langle y_i(t) \rangle$. The angular brackets $\langle \rangle$ denote an average over trials. The correlation of two spike trains is $\mathbf{m}_{ij}(t_i, t_j) = \langle y_i(t_i) y_j(t_j) \rangle$. If $i = j$ it is an autocorrelation, otherwise a cross-correlation. In general a n th order correlation, or moment, of n spike trains, is defined as a trial-average of products of those spike trains:

$$\mathbf{m}_{i_1, \dots, i_n}(t_1, \dots, t_n) = \left\langle \prod_{i=1}^n y_{i_i}(t_i) \right\rangle \tag{1}$$

In practice, time is discretized into increments of size Δt , and spike trains are binned. Equation 1 is recovered from its discrete counterpart in the limit $\Delta t \rightarrow 0$. If the spike trains are stationary (their statistics do not change over time) we can replace averages over trials with averages over time. The correlation in this case only depends on the *time lag* in between spikes:

$$\mathbf{m}_{i_1, \dots, i_n}(s_1, \dots, s_n) = \frac{1}{T} \int_0^T dt_1 y_{i_1}(t_1) \prod_{j=2}^n y_{i_j}(t_1 + s_j) \tag{2}$$

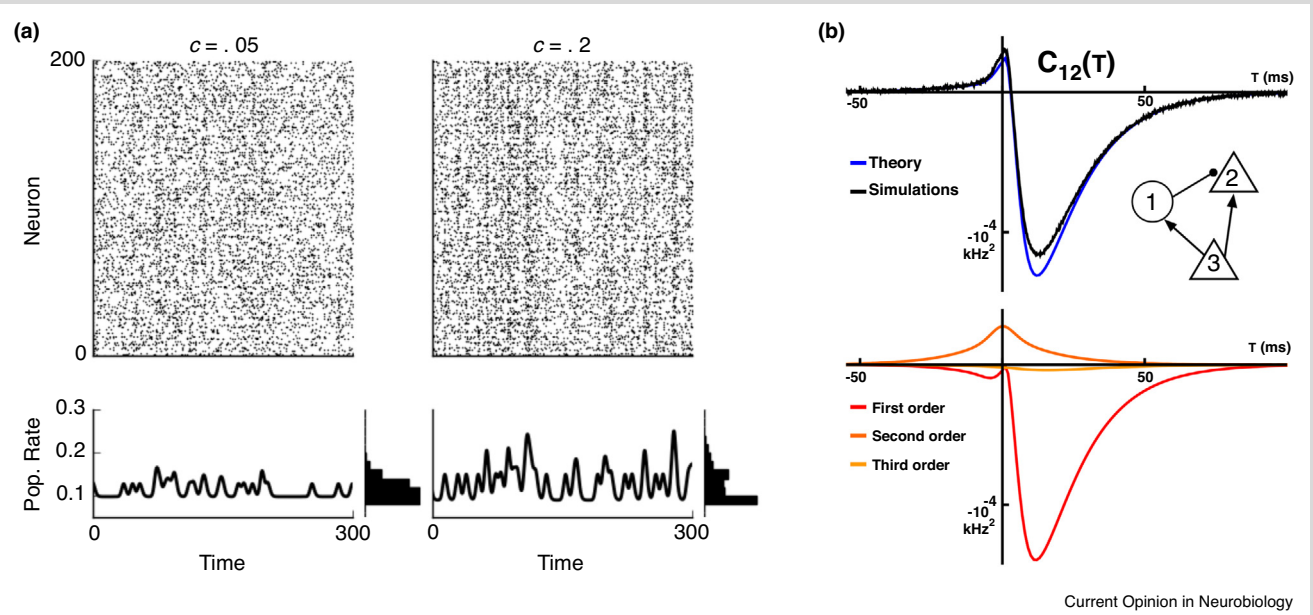
where $s_j = t_j - t_1$ for $j = 2, \dots, n$. The correlation function measures the frequency of spike pairs. Two uncorrelated Poisson processes with rates r_i and r_j have $\mathbf{m}_{ij}(s) = r_i r_j$, independent of the time lag s . The statistics of any linear functional of the spike trains (such as output spike counts, or synaptic outputs or inputs) can be derived from these spike train statistics [16,26].

The joint moments of the spike trains in a population also determine the variability and temporal correlations of the population-averaged activity, $y(t) = \frac{1}{N} \sum_{i=1}^N y_i(t)$. The average over the population can be interchanged with the average over trials and product over neurons in Equation 1 so that a m th order moment of the population activity, y , is given by:

$$m(t_1, \dots, t_m) = \frac{1}{N^m} \sum_{1 \leq i_1, \dots, i_m \leq N} \mathbf{m}_{i_1, \dots, i_m}(t_1, \dots, t_m)$$

Even weak correlations in \mathbf{m} can give rise to strong population fluctuations (**Panel A**).

Finally, moments mix interactions of different orders. To account for lower-order contributions, we can define *cumulants* of the spike trains. The first cumulant and the first moment both equal the instantaneous firing rate. The second cumulant is the covariance function of the spike train: $\mathbf{C}_{ij}(s) = \mathbf{m}_{ij}(s) - r_i r_j$. The third spike train cumulant similarly measures the frequency of triplets of spikes, above what could be expected by composing those triplets of individual spikes and pairwise covariances. Higher order cumulants have similar interpretations [27].



Pairwise correlations and population variability. (a) Variability of the population-averaged activity in 200 uncoupled integrate-and-fire neurons receiving white noise inputs with different strengths of spatial input correlation. (b) Cross-covariance of two neurons' spike trains in a feed-forward microcircuit with two excitatory (cell 2,3) and one inhibitory (cell 1) neurons. Top: simulation of (black) versus linear response theory (blue; Equation 6). Bottom: contribution of different length paths through the microcircuit (Equation 8). Adapted from [38].

cells downstream [6]. Similarly, higher-order correlations are related to the probability of observing triplets, quadruplets or more spikes in a group of neurons, separated by a given collection of time lags (Box 1).

Spike train covariability from recurrent connectivity and external input

In recent years, neuroscientists have advanced a very general framework for predicting how spike train

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