

Auto-structure of presynaptic activity defines postsynaptic firing statistics and can modulate STDP-based structure formation and learning

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Abstract. In this paper we study the impact of presynaptic activity that is deviating from Poissonian firing onto the postsynaptic firing of an conductance based integrate and fire neuron. We first show that the compound activity of a large group of neurons, e.g. presynaptic cells, cannot be described by a Poisson process in general. Then we demonstrate that the auto-structure of the presynaptic drive has strong impact onto the auto-structure of the postsynaptic spike-train. And finally, we discuss the potential impact of non-Poissonian presynaptic activity on the structure formation in recurrent networks based on Spike Timing Dependent Plasticity (STDP).

Key words: STDP, self-organization, non-Poissonian spike activity, recurrent networks, *Gamma*-process, *Log-normal*-process

1 Introduction

Neuronal self-organization and structure formation in recurrent networks have been proposed to be crucial elements in shaping the way the brain processes information [1–4, 7]. However, most theoretical and simulation based approaches that investigate neuronal self-organization tacitly use the assumption that spiking activity can be modeled by Poisson point processes. Since it is now undoubted that real neuronal activity is often strongly deviating from Poisson processes these theoretical and simulation based approaches might lack a major component of real neuronal firing.

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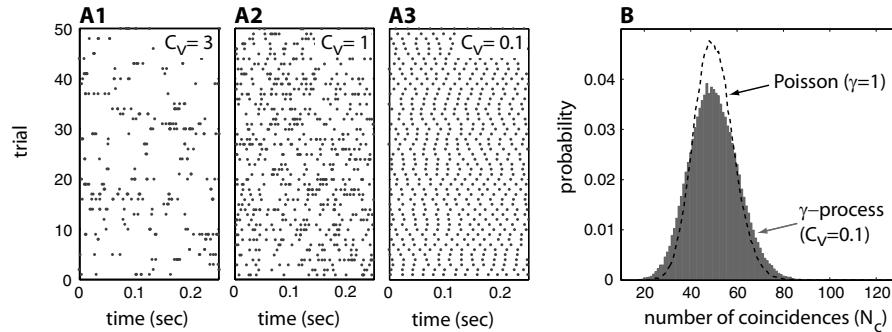


Fig. 1. (A1-3) Raster plot for 50 trials of *Gamma*-processes with 3 different coefficient of variations of the inter-spike interval distribution (A1: $C_V=0.1$; A2: $C_V=1$, A3: $C_V=3$). (B) Distribution of coincidence counts shared by pairs of mutually independent *Gamma*-processes of the same kind as shown in A. Dashed curves in (B) shows the coincidence count distribution for the cases of Poisson processes ($\gamma = 1$) and a *Gamma*-process with $C_V=0.1$. Coincidences were evaluated per trial based on a binned version (bin-length: $\Delta t = 4$ ms) of the original renewal process. Each distribution in B represents sample from $T = 100000$ trials of each 5 s length. The spike rate was chosen to be $R = 50$ ap/s.

2 Presynaptic activity deviating from Poisson

A typical cortical neuron receives input from many, up to more than 10^4 , other neurons. Taking the presynaptic neurons as independent it was believed that the compound process of the activity of many input spike-trains can be described as a Poisson process [5, 6]. In this case the auto-structure of individual pre-synaptic neurons could be ignored and a Poissonian statistics would have been a good approximation.

However, it has been shown analytically that this belief is wrong [8]. Lindner demonstrated that only the inter-spike interval (ISI) distribution and the ISI correlation at a finite time-lag approaches the ones of a Poisson process in the limit of large number of presynaptic spike-trains. Nevertheless, the power-spectrum of the compound process is identical to the power spectrum of the original spike-trains. This clearly demonstrates that presynaptic activity that is individually non-Poissonian will lead to a also non-Poissonian compound activity. For example, individual either regular or oscillatory presynaptic activity will lead to the same type of regular or oscillatory spiking in the compound process at the same time scale. This occurs even if many thousands spike-trains are added up. Hence, the impact of any presynaptic activity that is for example either regular or oscillatory is falsely ignored by present studies that model the compound activity as Poisson processes.

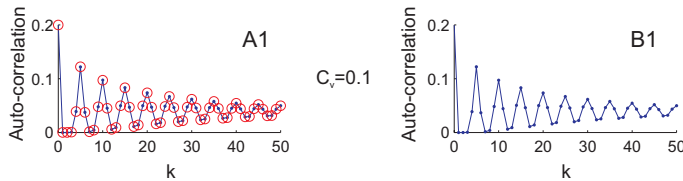


Fig. 2. Auto-correlation of the coincidences of *Gamma*-point processes (**A**), and a *log-normal*-point processes (**B**) with a C_V of 0.1 of the inter-spike interval distribution. A bin-length of $\Delta t = 4$ ms was used. The Auto-correlation is computed for time-lags τ with $\tau = k \cdot \Delta t$). The spike rate were chosen to $R = 50$ ap/s. Solid line indicate results derived from simulations, while circles locate an analytical evaluation.

3 Impact of Non-Poissonian firing on synchronous drive

Neurons are very sensitive to the synchronous firing of sub-populations, especially if they are in a state of balanced excitation and inhibition. Under this regime the membrane potential of a neuron is typically fluctuating around its spiking threshold and a small number of coincident input can be particularly effective in driving the cell above threshold. It is therefore important for to determine the parameters affecting the synchrony of presynaptic spike-trains. Only recently the influence of the auto-structure of renewal processes on the likelihood of synchronous firing between mutually independent processes was studied in Ref.[9]. The authors described the influence of the auto-structure of different mutually independent renewal processes (Fig. 1, panels A1-A3) on the shape of the coincidence count distribution (Fig. 1, panel B) and on the auto-correlation of coincidences (Fig. 2) for individual and independent trials. To this end the authors described the shape of the distribution by the Fano factor FF_C of the coincidence distribution. Please note that the Fano factor FF_C is equal to a scaled variance in case of a renewal processes, since the expected frequency of coincidences is independent of the type of the process [9].

Neuronal activity is continuous stream in time. Therefore the concept of individual and independent trials might look inappropriate. However, if the auto-correlation time of the spiking activity is short compared to the length of trials, one can understand trials as subsequent and independent pieces taken form the same continuous stream of neuronal activity. In this case, the Fano factor expresses the variability of counts of coincident events for different periods of the neuronal activity. In other words, the Fano factor FF_c describes the degree of clustering of coincidences in time. Please note that since the expected value of coincidences is independent of the type of the renewal process, different auto-structures lead only to a redistribution of the same number coincidences and therefore to different clustering in time.

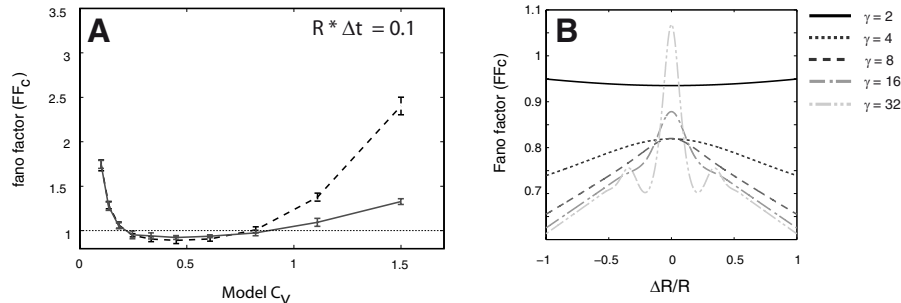


Fig. 3. (A) Fano factor of the coincidence count distribution as a function of the model C_V , for a *Gamma*-process and a *log-normal*-process. Coincidences were evaluated based on a binned version (bin-length: $\Delta t = 1$ ms) of the original renewal process. Both process types (*Gamma*- and *log-normal*) were parameterized by the product of the bin-length Δt in units of seconds and the firing rate R in units of [ap/s] (see legend). Each estimation of the C_V or Fano factor was based on $T = 2000$ trials of each 5 s length. The spike rate were chosen to $R = 50$ ap/s. (B) Analytically determined Fano Factor FF_C of the coincidence count distribution for two neurons with different rate $R_1 = R + \Delta R$ and $R_2 = R - \Delta R$ as function of $\Delta R/R$. *Gamma*-processes with shape parameter $\gamma = 2, 4, 8, 16, 32$ are explored. $R\Delta t = 0.2$.

4 Clustering of synchronous activity in time

We have studied then the Fano factor FF_C as a measure of clustering of synchronous activity for renewal processes with *Gamma*- and *log-normal*-distributed interspike intervals. The Fano factor of the coincidence count distribution for pairs of processes with identical spike-rates is represented in Fig. 3A for several coefficients of variations (C_V). High values of FF_C indicate strong clustering. To describe the changes of FF_C we will use the Poisson process as a reference model since the Poisson process was commonly used in modeling studies. We have observed that only intermediate regularity in the processes ($0.2 < C_V < 0.8$) lead to smaller values of FF_C and consequently to less clustering than in the case of a Poisson process. For very low and very high C_V s (corresponding to very regular and bursty spiking, respectively) the Fano factor can exceed the FF_C of a Poisson process by a factor larger than 2. However, if the two independent processes have different rates (Fig. 3B), the FF_C and therefore the clustering of synchrony for *Gamma*-processes is in general lower than in case of a Poisson process that exhibits a $FF_C = 1.4$ for the given parameters.

In summary, our first major finding is that deviations from individual Poissonian firing can induce clustering of coincidences between mutually independent point-processes in time. The amount of clustering depends on the detailed properties of the inter-spike interval distribution, and can be described by the Fano Factor FF_C of the coincidence count distribution. Our second major finding is that the clustering depends very critically on whether the rate across different point-processes is the same or not. We found that small differences of the the

spike-rates of two processes in the order of a few percent can cause changes in the clustering measured by changes of FF_c of up to 50% in some cases (Fig. 3B). The third major finding is that both, bursty and regular firing, can induce clustering of coincidences. However, the underlying mechanism and cause of the clustering for bursty and regular processes is different. In the case of bursty processes clustering is due to a repeated firing of individual neurons in a period as short as the bin-width Δt that defines the time-scale of a coincidence. In contrast, in case of regular firing clustering is caused by a kind of periodic repetition of the same pattern on a time-scale defined by the expected inter-spike interval.

5 IF neurons and presynaptic *Gamma*-processes

A prior step in demonstrating that deviations from Poissonian firing can modulate the structure formation in recurrent networks based on neuronal plasticity like STDP, is to show first that different variations of non-Poissonian presynaptic activity impact the postsynaptic spiking activity of a neuron.

To this end we simulated a conductance based Integrate and Fire neuron that receives input from one excitatory and one inhibitory population, with $N_E = 400$ and $N_I = 100$ cells, respectively. Exponentially decaying synaptic currents with time constants $\tau_{ampa} = 2$ ms and $\tau_{gaba} = 5.6$ ms were used respectively for the AMPA and GABA mediated receptors. The membrane time constant was set to $\tau_m = 20$ ms. The synaptic conductance strength g_{gaba} and g_{ampa} were chosen to be identical across all synapses of the same type. We model the postsynaptic neuron to be in a state close to balanced excitation and inhibition. To this end we defined g_{ampa} and g_{gaba} based on the balance between excitation and inhibition β as well as the total conductance in units of the leakage conductance g_{leak} . Motivated by studies that measured this relation in vivo we have chosen the total conductance to be four times higher than the leakage.

Panels A in Figure 4 shows the postsynaptic spike triggered average of the presynaptic spike activity, which describes the average presynaptic activity of the excitatory population (upper trace) and the inhibitory average activity (downer trace) in a window center around a postsynaptic spike. For the two point processes studied (Poissonian in A1 and *Gamma*-distributed in A2), on average an increase in the excitatory and/or a decrease in the inhibitory activity are necessary to drive the postsynaptic neuron to spike. Please note that both are by definition random fluctuation since the presynaptic activity has a constant expected spike count and is mutually independent across neurons. Remarkably, the the presynaptic spike triggered average for regular *Gamma*-processes (Fig. 4, A2) shows a damped periodic oscillation with the expected interspike interval of the presynaptic activity. This demonstrates that random fluctuations leading to increases of the excitatory or decreases of the inhibitory drive have the same auto-correlation structure as each of the original presynaptic point-processes.

This notable feature relies on the thresholding properties of the postsynaptic neuron and can be intuitively understood as follows. Since the neuron is close to a state of balanced excitation/inhibition, synchronous activity from a

rather small subpopulation of neurons is sufficient to make the neuron firing. For simplicity we assume for now that all presynaptic processes of such driving subpopulation are identical. We know from the work of Linder [8] that the power-spectrum of the compound process is the same as for each individual neuron, and consequently we know that a subpopulation that is synchronous by chance will have a cross-correlation with the same periodicity as the auto-correlation as each of the individual neurons. Hence, a chance synchronization of a subpopulation will lead to a package of postsynaptic spikes that follow on average the same auto-structure as each individual presynaptic process. This is illustrated in the auto-correlation function of the postsynaptic point-process shown in panel B2 of Figure 4. Interestingly, the ISI distribution shows multiple peaks. This is because the average postsynaptic spike rate is much lower than the compound presynaptic activity. Hence, the postsynaptic cell does not fire at the timing corresponding at each peak of the auto-correlation of the presynaptic drive. Such effect can be understood as a modulation of the firing probability of a stochastic point-process. Therefore, the postsynaptic neurons might do cycle skipping what induces presynaptic and postsynaptic spike-rates to be different.

6 Non-Poissonian input and structure formation in recurrent networks

Once we have evaluated the role of non-Poissonian presynaptic input in the firing properties of a postsynaptic neuron we are now in position to discuss the potential impact of non-Poissonian presynaptic activity on structure formation and learning via STDP. This form of plasticity has been applied to sequence learning and has been discussed to be involved in spontaneous and activity driven pattern formation [10, 1, 4]. STDP strengthens potentially causal relations between presynaptic drive and postsynaptic activity by increasing the synaptic strength of all synapses that have been activated immediately before a postsynaptic spike is generated. Since the clustering of coincidences in the presynaptic activity has been shown to impact postsynaptic events, it is also expected to modulate STDP-based pattern formation. Specifically, bursty presynaptic activity leads to clusters of presynaptic coincident events on a very short timescale and just a single postsynaptic potential can be sufficient to modulate the strength of neuronal plasticity.

In case of regular presynaptic activity the situation is different. In case of regular presynaptic activity the situation is different, such that regular presynaptic activity leads to periods postsynaptic activity with the same regularity as the presynaptic drive. In this scenario the temporal relation of presynaptic and postsynaptic activity needs to be maintained for a duration of the order of a few expected interspike intervals of the presynaptic activity to exert a significant influence. Exactly this maintenance of the temporal order between presynaptic drive and the postsynaptic activity has been observed in our simulation of an IF neuron subjected to structured presynaptic input. Hence, the clustering of

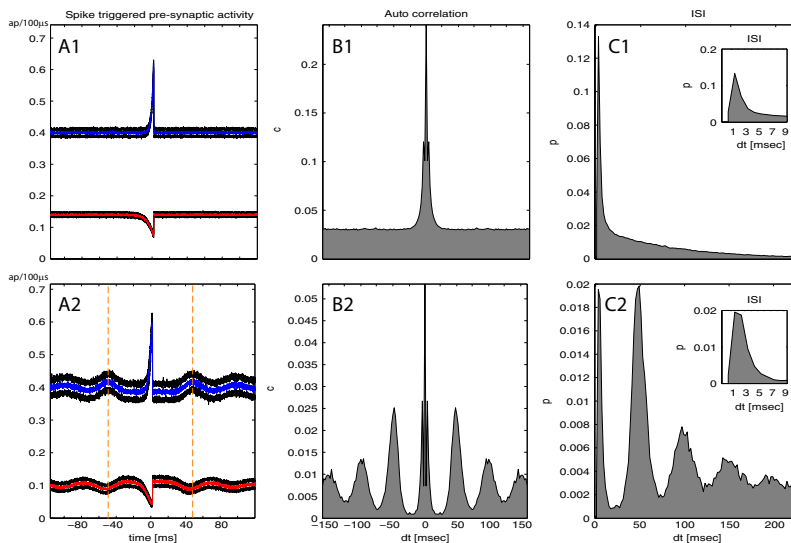


Fig. 4. (A1,2) Postsynaptic spike triggered average of the presynaptic activity in units of number of spikes per $100 \mu s$ of the whole presynaptic population of 400 excitatory (upper trace) and 100 inhibitory (downer trace) cells. (B1,2) Auto-correlation function of the postsynaptic spiking activity (bin-width $\Delta t=1$ ms). (C1,2) Inter-spike interval distribution of the postsynaptic spiking activity (bin-width $\Delta t=2$ ms). The indices 1 and 2 in the panels distinguishes between the individual Poissonian and *Gamma*-distributed presynaptic point process cases. The average postsynaptic spike rate was, 15.2 ap/s in the Poissonian case, and 3.36 ap/sec in the *Gamma* process. In both cases the presynaptic spike rate of the excitatory population was 28 ap/sec per synapse. The presynaptic spike rate of the inhibitory population was 20 ap/sec per synapse

coincident presynaptic activity, described by the Fano factor FF_C , is a critical parameter in studying the impact of different point-processes onto STDP.

7 Clustering and repetition of presynaptic synchronous spike pattern in dependence of the Fano Factor

Given our results concerning the temporal clustering of coincidences described by FF_C we can already distinguish between two scenarios. First, let us start by the high variance case where FF_C is large, and coincidences are more clustered than in case of Poissonian firing. This case occurs for very regular and bursty auto-structures (see Fig. 3A), and only in the case that the rates of the all spikes trains are identical or have a n:m relationship. In the second scenario FF_C is lower, and coincidences are less clustered than in case of Poissonian firing. This second scenario occurs predominately for non-rational rate relations of the processes and intermediate regularity.

Whether sets of individual neurons are either in the low FF_C , or high FF_C state, can have important implications on the neuronal dynamics and could be used by the neural system to modulate the clustering of synchronous neuronal activity that is occurring by chance. For example, the high FF_C regime seems to be well suited for spontaneous and activity driven structure formation due to neuronal plasticity, like STDP [10, 1, 4, 11]. Spontaneous pattern formation requires a spontaneous symmetry breaking in the activity driving the network such that a randomly occurring pattern is strengthened and embedded as a reliable reoccurring sequence. A regime where the frequency of coincident neuronal activity is highly clustered in time is especially suited for such a spontaneous pattern formation since a pattern that occurs once by chance have a higher likelihood to be repeated in a short temporal window than in case of Poissonian activity. This repetition of individual chance patterns could boost the effect of STDP for short periods in time since the same patterns occur more than once. However, there is a critical period length that STDP requires to reinforce a given pattern. If STDP requires many reoccurrences of the same pattern for reinforcement the period length will grow larger than, first a couple of interspike intervals, and second the auto-correlation time of the process. For periods that are long, the advantage of the high variability regime vanishes since periods with high numbers of coincidences will be followed by periods with low numbers of coincidences such that the variability of the coincide count distribution is reduced.

More relevant is the case where individual neurons have different spike rates. In that case changing the auto-structure of the individual processes can also regulate STDP processes. Our findings based on *Gamma*-processes with rates that are not related to each other as $n:m$ (with $n, m \in \mathbb{N}$) demonstrate that Poissonian firing yields the highest FF_C and therefore a higher clustering of coincidences than regular and bursty *Gamma*-processes. Hence, a Poissonian profile of spikes boost the effect of STDP compared to other statistics of spike-trains as long as the spike rates from individual neurons are different and does not express a $n:m$ relationship.

8 Conclusions

We have described how auto-structure of presynaptic activity results in general in a compound activity on non-Poissonian statistics. We have also concluded that the driving of a postsynaptic neuron by spike-trains deviating from a Poissonian profile influences the statistics of firing the postsynaptic cell in a well defined relationship with respect to the presynaptic characteristics. Finally, and assuming that a random pattern occurring between mutually independent processes can be seen as a seed for the process of spontaneous pattern formation, we have discussed how changes of the auto-structure, such as regularity of presynaptic spike-trains, can modulate and boost the efficiency of STDP for short epochs of the length of a few interspike intervals. Consequently, the regularity and burstiness of neuronal spike-train can potentially act as a gating or boost-

ing mechanism for the ability of spontaneous symmetry breaking and pattern formation.

Appendix

8.1 A: *Gamma*-process and *log-normal*-process

The ISI distribution for a *Gamma*-process with a given constant spike rate is described by:

$$p_\gamma(t) = t^{\gamma-1} \frac{(\gamma R)^\gamma e^{-\gamma R t}}{(\gamma - 1)!} \quad \text{for } t > 0 \quad . \quad (1)$$

This distribution is characterized by two variables, the spike rate, R , and the shape-parameter, γ . The *Poisson*-process is a special case of the *Gamma*-process for which $\gamma = 1$. Values of the shape-parameter γ smaller than 1 make short intervals more likely to occur than for a *Poisson*-process, and are used to model bursty firing, while large values of the shape parameter are used to model regular firing. We use the C_V of the ISI distribution to characterize the processes. The the shape-parameter, γ and the C_V value are related as follows:

$$\gamma = \frac{1}{C_V^2} \quad . \quad (2)$$

As a second kind of renewal process characterized by two variables we use the *log-normal*-process. Its interspike interval distribution ($p(t)_{\log-normal}$) is defined by:

$$p(t)_{\log-normal} = \frac{1}{k\sqrt{2\pi}} \frac{\exp\left(-\frac{(\ln(t)-a)^2}{2k^2}\right)}{t} \quad . \quad (3)$$

The spike rate, R , and coefficient of variation, C_V , can be expressed by a and k as follows:

$$a = -\ln R - \ln(C_V^2 + 1) \quad (4)$$

and

$$k = \sqrt{\ln(C_V^2 + 1)} \quad . \quad (5)$$

8.2 B: Coincidences

Given two parallel spike-train processes we define a coincidence based on binned versions of the original processes. The binned spike trains are obtained by segmenting the time axis into exclusive bins, each of length Δt , and counting the number of spikes per bin k . The number of coincidences in bin k shared by two spike trains for two simultaneous bins and of neuron 1 and neuron 2 is then defined by $N_c^k = n_k^1 * n_k^2$. This definition can be trivially extended to more than pairs.

Acknowledgements

The authors would like to thank Carl van Vreeswijk, and Larry Abbott for inspiring discussions and great support during the first stages of this project. This work was partially supported by the Hertie Foundation and the European Commission Project (FP6-NEST contract 043309).

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