# INFORMATION MEASURES IN A SMALL NETWORK OF SPIKING NEURONS

Maria Teresa Giraudo, Laura Sacerdote and Roberta Sirovich

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Abstract. The information transmission among the elements of a small network of spiking neurons is studied using the normalized differential entropy and the Kullback Leibler distance information measures. The attention is devoted to the information content of the spiking activity of a reference neuron subject to excitatory and inhibitory stimuli coming from other elements of the considered network. The use of information measures allows to quantify the effects of the input contributions. The role of inhibition in the spiking activity of the reference neuron is enlighted and the effect of considering different distributions for the time events of the stimuli is discussed.

**1 Introduction** The classical characterization of the input-output properties of neurons, as well as of the neuronal models, makes use of the so called frequency transfer functions. Several definitions exist for these functions but they all share the feature of being plots of the output frequency of firing against the strength of the input signal. The differences are confined to the measure employed to quantify the input strenght. Use of the transfer function tacitely assumes that the information is coded by the frequency of action potentials ([2]). However, frequency is only one possible descriptor of the properties of a spike train and it is evident that different spike records may have the same frequency but at the same time they may be very dissimilar. Therefore, other tools to quantify the differences among spike records have been investigated. Considering the well established relevance of noise in neuronal coding (cf. for example [3, 15, 21, 19]), alternative measures that could be able to catch the random properties of the firing activity have been introduced.

In this direction many efforts have been made to apply Shannon theory of communication and information transmission ([20]) to the study of the properties of the nervous system. Among the wide literature on this topic, we cite here for example [11], where the Fisher information is used to study the input-output effect for some neuronal models. In [12] and [13] this measure is applied to the stochastic leaky integrate-and-fire (LIF) model, identifying the input signal as a constant additive term in the drift of the Ornstein-Uhlenbeck process used to describe the underthreshold membrane potential dynamics. In [9] a normalized version of the differential entropy is introduced to study the randomness for the same neuronal model.

Information theory is applied here to the study of the information transmission among the units of a small network of spiking neurons. In particular we are interested in the quantification of the response of a reference neuron to excitatory and inhibitory inputs coming from other units of the network. The problem has been already considered in [22] and [23] but the study is performed there with different tools of analysis, as histograms, autocorrelation functions and crosscorrelation functions.

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Goal of this paper is to recover features of the input-output transmission, already discussed in [23], using suitable information measures to quantify the observed phenomena. To this aim we employ the normalized differential entropy and the Kullback-Leibler distance (cf. [4]) to characterize the firing behavior of the neuronal network.

The paper is organized as follows. In Section 2 the network and its mathematical model are introduced. In Section 3 the information measures that will be applied and their interpretation in the neuronal context are presented. In Section 4 we compare the features exhibited by the reference neuron output under a set of different conditions for the spiking distribution of the excitatory and inhibitory neurons and for the noise intensity. As discussed in Section 5, the results globally confirm those obtained in [23], but the use of information theory tools allows to make some quantitative observations that could not be obtained otherwise.

**2 The model** We consider here a particular kind of stochastic neuronal model. The state of the neuron, described by its membrane potential, is assumed to evolve in time as a realization of the stochastic process $V = \{V_t; t \geq 0\ , V_0 = v_{rest}\}$, where $v_{rest}$ is the membrane resting potential. When the membrane potential reaches a fixed level $S$ for the first time the cell is assumed to generate an action potential and $V$ is reset to the resting value $v_{rest}$. The firing times of the model neuron are given by the sequence of the first passage times through the threshold $S$ of the process $V$. Their sequence gives then rise to a renewal point process. In this way the distribution of the random variable first passage time

$$T = \inf\{t \geq 0 | V_t \geq S, v_{rest} < S\} \tag{2.1}$$

is the mathematical counterpart of the inter-spike interval (ISI) distribution.

In the model we consider here, the process $V$ is solution to the following stochastic integral equation:

$$V_t = v_{rest} + \int_0^t \left(-\frac{1}{\theta}V_s + \mu\right) ds + \sigma W_t + a^+ N_t^+ + a^- N_t^-, \tag{2.2}$$

where $\theta, \sigma \in \mathbb{R}^+$, $\mu \in \mathbb{R}$, $a^+ > 0$, $a^- < 0$, $a^+, a^- \in \mathbb{R}$, $N^+ = \{N_t^+; t \geq 0, N_0^+ = 0\}$ and $N^- = \{N_t^-; t \geq 0, N_0^- = 0\}$ are two independent counting processes and $W = \{W_t; t \geq 0\}$ is a standard Brownian motion. It is a jump-diffusion process whose trajectories are continuous in time except for point discontinuities at the times occurring as epochs in the processes $N^+$ and $N^-$. For $a^+ = a^- = 0$, eq. (2.2) is the stochastic integral equation for the so called Ornstein-Uhlenbeck process, known in the stochastic neuronal modeling literature as the LIF model (for a review see for example [5, 18, 25]). The generalization of such a model for $a^+ \neq 0$ and $a^- \neq 0$ has been derived in [16] from Stein's model and successively studied and reinterpreted in [22] and [23].

The model reproduces the output spike train of a small network of neurons composed of a reference neuron $A$ that receives strong inputs from an excitatory unit $E$ and from an inhibitory unit $I$ and that we suppose to lie embedded into a larger network that contributes with weaker inputs. Such inputs are summed up to generate the global net input that corresponds to the the term $\mu$ in eq. (2.2). In the same way the overall variability of the inputs coming from the surrounding environment is represented by the noise level term $\sigma$.

Figure 1 illustrates the interpretation of the model. Neuron $A$ is the cell whose membrane potential is described by means of the process $V$ in eq. (2.2) and that receives inputs by the whole surrounding network (many weak inputs contributing to a background fluctuation of the cell membrane potential) and by strong excitatory ($E$) and inhibitory ($I$) neurons.

The excitatory and inhibitory inputs from units $E$ and $I$ occur at time epochs driven by the stochastic processes $N^+$ and $N^-$. Their effect is to cause the occurrence of upward and downward jumps of amplitudes $a^+$ and $a^-$ respectively in the membrane potential $V$ of cell $A$.

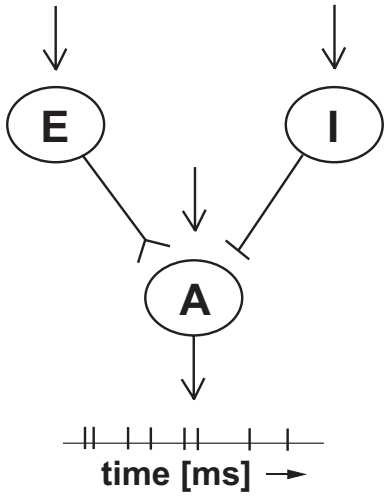


Figure 1: Illustration of the model described by eq. (2.2).

The ISI distribution of cells $E$ and $I$ has not been specified in eq. (2.2). We consider here two different ISI distributions for the excitatory and inhibitory units $E$ and $I$: the Exponential distribution and the Inverse Gaussian (IG) distribution. In the first case $N^+$ and $N^-$ are two independent Poisson processes of parameters $\lambda^+$ and $\lambda^-$ respectively while in the second case they are independent counting processes with IG inter-event distribution. Let us recall that a random variable is said to be IG$(a, b)$ distributed if its probability density function (p.d.f.) $p$ is given by

$$p(t; a, b) = \sqrt{\frac{b}{2\pi}}\, t^{-3/2} \exp\left[-\frac{b(t-a)^2}{2a^2 t}\right], \quad t > 0. \tag{2.3}$$

Hence, for $a$ and $b$ chosen such that $a = |S|/\mu$ and $b = S^2/\sigma^2$, the preceding equation gives the p.d.f. of the random variable first passage time to the threshold $S > 0$ of a Wiener process originated in $x_0 = 0$ with drift $\mu$ and diffusion coefficient $\sigma$. From a modeling point of view this corresponds to the ISI distribution of a neuronal cell modeled as a perfect integrator (cf. [25]). In what follows we denote with $\mu_+$, $S_+$, $\sigma_+^2$ and $\mu_-$, $S_-$, $\sigma_-^2$ the drift term, the threshold and the diffusion coefficient characterizing the IG distribution of excitatory and inhibitory inputs respectively.

To summarize, the small network of spiking neurons described in eq. (2.2) is composed by a reference neuron $A$ whose membrane depolarization is modeled with a LIF model and that receives further strong inputs from two excitatory and inhibitory units $E$ and $I$. The firing behavior of these cells is modeled either by means of an IG inter-event distribution or with an Exponential inter-event distribution.

## 3 Information Measures

**3.1 Definitions** Information theory provides a quantitative mean to measure dependencies between the components of real or simulated neuronal networks. Examples of its use can be found in the neuroscience context in [7, 8, 9, 11, 12, 13]. We are concerned here with two kinds of information measures: the normalized differential entropy and the Kullback-Leibler distance, that we will use to quantitatively characterize the code determined by the laws governing the network introduced in Section 2.

The entropy $H$ of a discrete random variable was introduced by Shannon in [20] as a measure of uncertainty. Since information can be considered as a decrease in uncertainty, the entropy is a quantity that measures the information. The extension of the notion of entropy to a continuous random variable $X$ having the probability density function $f(x)$ is called the differential entropy $h(X)$ of the variable $X$ or also the differential entropy $h(f)$ of the distribution $f(x)$ and is defined (cf. [4]) as

$$h(f) = -\int_I f(x) \ln f(x) dx \tag{3.4}$$

where $I$ denotes the range of the random variable $X$. The differential entropy $h(f)$ unlikely the entropy $H$ of a discrete random variable can become negative and is not constant under coordinate transform. Hence it cannot be employed as an absolute measure of the information content carried by a distribution. However the higher is $h(f)$ the higher can be considered the degree of randomness of the given distribution. In other words, distributions with a higher level of information are characterized by lower differential entropy.

Since the particular value of $h$ depends on the mean value of $X$, $\mathbb{E}(X)$, in order to make the differential entropy independent on linear scalings the random variable $X$ can be transformed into the dimensionless variable $\xi = X/\mathbb{E}(X)$ (cf. [9]). The variable $\xi$ has mean value $\mathbb{E}(\xi) = 1$ and its entropy is the "normalized entropy" $\eta(X)$ of the unscaled random variable $X$ (cf. [9]). It is related to the differential entropy of $X$ as

$$\eta(X) = h(X) - \ln \mathbb{E}(X). \tag{3.5}$$

The value of $\eta$ can reach infinite negative values and is maximized ($\eta = 1$) in the case of maximum randomness, that is realized by an Exponential distribution for $X$. Hence its value can be read as a measure of farness of the considered random variable from the Exponential one. We will use the normalized entropy in Section 4 to measure the information contribution to the neural network of Section 2 of changes in the distribution of the inputs from excitatory/inhibitory neurons or to investigate the information gain related to changes in the noise intensity regulating the reference neuron.

To compare two different random variables $X$ and $Y$ with probability density functions $f(x)$ and $g(x)$ respectively defined over the same range $I$ we employ the Kullback-Leibler distance $K(f, g)$ of the distribution $f$ with respect to the distribution $g$, also called Kullback entropy (cf. [10], [4]), defined as

$$K(f, g) = \int_I f(x) \ln \frac{f(x)}{g(x)} dx. \tag{3.6}$$

The Kullback-Leibler distance is not a measure since it is not symmetric and it does not satisfy the triangle inequality. However $K(f, g)$ is always not negative, it is invariant with respect to a transformation of variable and $K(f, g) = 0$ if and only if the two distributions are identical, $f(x) = g(x)\ \forall x \in I$. Hence it can be used to compare the distributions $f$ and $g$ or more specifically to quantify the information content of $f$ with respect to $g$ taken as a reference. In this last case the reference distribution can be considered as the state of leak of information for the purpose intended (cf. [24]). In the case of spontaneously

active neurons it is then possible to compare the ISI distributions with and without added stimulations. Any increase in the Kullback-Leibler distance will measure the importance of the stimulation with respect to its absence.

**3.2 Methods** To obtain the differential entropy and the Kullback-Leibler distance from data sets it is necessary to employ suitable reliable estimation techniques.

The problem of estimating differential entropy has been the object of great attention in literature during the last years due to its crucial role in many applications (cf. for example [17] and references quoted therein). We employ here a modification of the entropy estimator proposed by Vasicek in [26] based on the spacings between the realizations of the given random variable. Let us consider a random variable $X$ with probability density function $f(x)$. Let $x_1, x_2, ..., x_n$ be a sample extracted from the distribution $f$. Expressing the differential entropy $h(f)$ defined in (3.4) as

$$h(f) = \int_0^1 \ln\left\{\frac{d}{dp}F^{-1}(p)\right\} dp, \tag{3.7}$$

where $F$ is the distribution function of $X$, an estimate of (3.7) can be obtained by replacing $F$ with the empirical distribution function $F_n$ and then using a difference operator instead of the differential operator. Denoting as $x_{(1)} \leq x_{(2)} \leq ... \leq x_{(n)}$ the order statistics of the sample extracted from the distribution of $X$, the derivative of $F^{-1}(p)$ can be estimated by $\left(x_{(i+m)} - x_{(i-m)}\right) n/(2m)$ for $\frac{i-1}{n} < p \leq \frac{i}{n}$, $i = m+1, m+2, ..., n-m$ where $m$ is a positive integer smaller than $\frac{n}{2}$ and one-sided differences of the type $x_{(i+m)} - x_{(1)}$ or $x_{(n)} - x_{(i-m)}$ are used in place of $x_{(i+m)} - x_{(i-m)}$ when when $p \leq \frac{m}{n}$ or $p > \frac{n-m}{n}$ respectively. The Vasicek estimator of $h(f)$ is then:

$$H_{mn} = \frac{1}{n}\sum_{i=1}^{n} \ln\left\{\frac{n}{2m}\left(x_{(i+m)} - x_{(i-m)}\right)\right\} \tag{3.8}$$

with $x_{(j)} = x_{(1)}$ for $j < 1$, $x_{(j)} = x_{(n)}$ for $j > n$. An optimal choice of the parameter $m$ for a given $n$ should depend on the (unknown) distribution $F$. The heuristic formula

$$m = \left\lfloor \sqrt{(n)} + 0.5 \right\rfloor, \tag{3.9}$$

where the symbol $\lfloor x \rfloor$ denotes the floor of $x$, is often used in applications (cf. for example [6]). The estimator (3.8) is consistent, i.e. $H_{mn} \to h(f)$ as $n \to \infty$, $m \to \infty$ and $\frac{m}{n} \to 0$ (cf. [26]), but it is not unbiased. The unbiased version of the Vasicek estimator for differential entropy is (cf. [27])

(3.10)

$$V_{mn} = H_{mn} - \ln(n) + \ln(2m) - \left(1 - \frac{2m}{n}\right)\Psi(2m) + \Psi(n+1) - \frac{2}{n}\sum_{i=1}^{m}\Psi(i+m-1),$$

where $\Psi(x)$ is the digamma function defined as $\Psi(x) = \frac{d}{dx}\Gamma(x)$ with $\Gamma$ the gamma function (cf. [1]). The estimator (3.10) is consistent (cf. [26]) and in [27] it has been shown that it reveals the smallest root mean square error when compared with other existing estimators of the differential entropy.

As far as the estimate of the Kullback-Leibler distance (3.6) is concerned, we proceed by splitting it in the following way:

$$\begin{aligned} K(f,g) &= \int_I f(x)\ln f(x)dx - \int_I f(x)\ln g(x)dx \\ &= -h(f) - \int_I f(x)\ln g(x)dx. \end{aligned} \tag{3.11}$$

While the first term at the r.h.s. of the preceding formula can be estimated by means of (3.10), the evaluation of the remaining integral requires to estimate the appearing densities from a finite number of ISIs. This means that instead of the (unknown) theoretical distributions we have to rely on finite sample ISI histograms. We then employ the Laplacian sample size correction estimator for probabilities (cf. for example [28]) that can be described as follows. Given a total of $B$ bins for the histogram of the simulated ISI distribution with frequency distribution values $n_1, n_2, ..., n_B$ and $N$ ISI counts one can estimate the related probabilities as

$$q_i = \frac{n_i + \epsilon}{N + \epsilon B},\ i = 1, ..., B, \tag{3.12}$$

where $\epsilon$ is a constant usually taken smaller than 1. It is shown in [14] that this estimator for the ISI distribution is asymptotically (i.e. for $N \to \infty$) unbiased and consistent. Employing (3.12) for both distributions $f$ and $g$ involved in $K(f, g)$ also avoids a divergence of the Kullback-Leibler distance, i.e. an infinite information gain, when the estimated value of $g$ vanishes in correspondence of a bin where the estimated value of $f$ is still away from zero.

Our study is then performed via computer simulation of the ISI distributions for the reference neuron both when the input neurons $E$ and $I$ in the network are silent and when they are in activity. For the spiking times of the input neurons we employ an algorithm similar to the one described in [22]. To obtain the spiking times for the processing neuron we use a jump-adapted simulation procedure analoguous to the one deviced in [22]. For every case $N = 10000$ simulation runs for the processing neuron are executed.

For the value of $m$ required in the estimation of the differential entropy by means of (3.10) we always employ the relationship given in (3.9).

**4 Results** We present here some examples to illustrate the role of excitatory and inhibitory contributions and the effect of the noise intensity $\sigma^2$ in the signal transmission among the units composing the small network illustrated in Section 2.

As far as the parameter choice for cell $A$ is concerned, the resting potential is set to $v_{rest} = 0$ mV, being (2.2) an equation for the membrane depolarization. The threshold is fixed to $S = 10$ mV and the time constant to $\theta = 10$ ms, in accordance with biologically reasonable values. We choose the net input parameter as $\mu = 0.7$ or $\mu = 0.98$ mVms$^{-1}$ depending on the specific example. The first value ($\mu = 0.7$) corresponds to the so called under-threshold regime, i.e. the neuron would be unable to produce a spike in the absence of the noise contribution represented by the term $\sigma W_t$ in equation (2.2), while the second value ($\mu = 0.98$) is such that $\mu\theta \sim S$, being the dynamics of the cell governed by the deterministic term in eq. (2.2). Whenever kept fixed, the level of noise is set to $\sigma^2 = 0.05$ mV$^2$ms$^{-1}$.

To represent the intensity of the strong inputs carried onto the reference unit $A$ by the two unities $E$ and $I$, large values of $a^+ = -a^- = 5$ mV are chosen with respect to the threshold value for the membrane potential of cell $A$. Moreover we consider the condition with balanced excitatory and inhibitory strong inputs, such that $a^+ = -a^-$. When the jump processes are characterized by IG distributed ISIs the parameters $S_+ = S_- = 10$ mV are kept constant while the frequency and the variability parameters are varied.

**4.1 Normalized Differential Entropy** To describe the response of the model reference neuron to the stimuli coming from the excitatory and inhibitory units $E$ and $I$ in the network in terms of normalized differential entropy we distinguish among the two following settings for the jump processes:

**(a)** IG distributed ISIs for the input neurons.

**(b)** Exponentially distributed ISIs for the input neurons.

*4.1.1 Dependency on the parameters characterizing the excitatory and inhibitory inputs* **Case (a).** In Fig. 2, left panel we represent the behavior of the normalized differential entropy in case $(a)$ for $\mu = 0.98$ mVms$^{-1}$ when the values of the frequency parameters for the jump processes are kept fixed to $\mu_+ = \mu_- = 0.5$ mVms$^{-1}$ while modulating the variability parameters. The value of the normalized entropy in the absence of stimulation from cells $E$ and $I$ ($a^+ = -a^- = 0$ mV) is also represented in the plot. The normalized entropy is increasing for increasing values of $\sigma_+^2 = \sigma_-^2$ and it is greater than its level in the absence of stimuli when moderate and high values of $\sigma_+^2 = \sigma_-^2$ are considered. This hints to a relevant role of these parameters in determining the change in the amount of information carried by the reference neuron as a consequence of the inputs received by the other two unities in the network. Indeed for small values of $\sigma_+^2 = \sigma_-^2$ the ISI distribution of the excitatory and inhibitory inputs is characterized by a small variance and hence quite regular stimuli from units $E$ and $I$ reduce the randomness in the firing times of cell $A$.

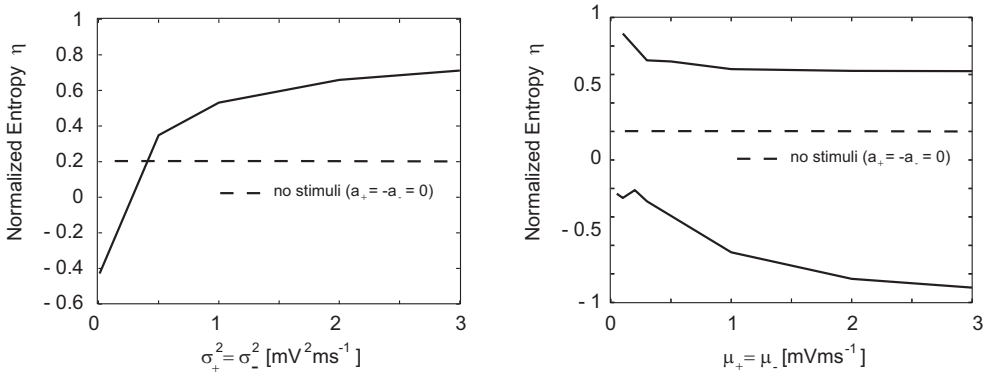


Figure 2: Normalized differential entropy of the simulated spike train for cell $A$ in the presence of excitatory and inhibitory inputs from units $E$ and $I$ (continuous lines), compared with the case with no stimuli (dashed lines). Inputs from $E$ and $I$ are supposed IG inter-distributed. Both panels: $S = 10$ mV, $\theta = 10$ ms$^{-1}$, $\mu = 0.98$ mVms$^{-1}$, $\sigma^2 = 0.05$ mV$^2$ms$^{-1}$, $a_+ = -a_- = 5$ mV. Left panel: $\mu_+ = \mu_- = 0.5$ mVms$^{-1}$. Right panel: $\sigma_+^2 = \sigma_-^2 = 1$ (upper continuous line) and 0.01 mV$^2$ms$^{-1}$ (lower continuous line).

In Fig. 2, right panel we represent the behavior of the normalized differential entropy when the values of $\mu_+ = \mu_-$ are varied while fixing $\sigma_+^2 = \sigma_-^2$ to 0.01 (lower continuous line) and 1 mV$^2$ms$^{-1}$ (upper continuous line). Here again the reference value of the entropy in the case of no strong stimuli for cell $A$ is reported. In correspondence with low levels of variability for the input processes the differential entropy is lower than the reference value. A loss of randomness in the spiking behavior of cell $A$ can be recognized due to the added regularity of the incoming inputs from cells $E$ and $I$. When the level of variability expressed by $\sigma_+^2 = \sigma_-^2$ is deeply increased the effect is reversed. The differential entropy becomes higher than in the reference case for all values of $\mu_+ = \mu_-$. In this case the ISI

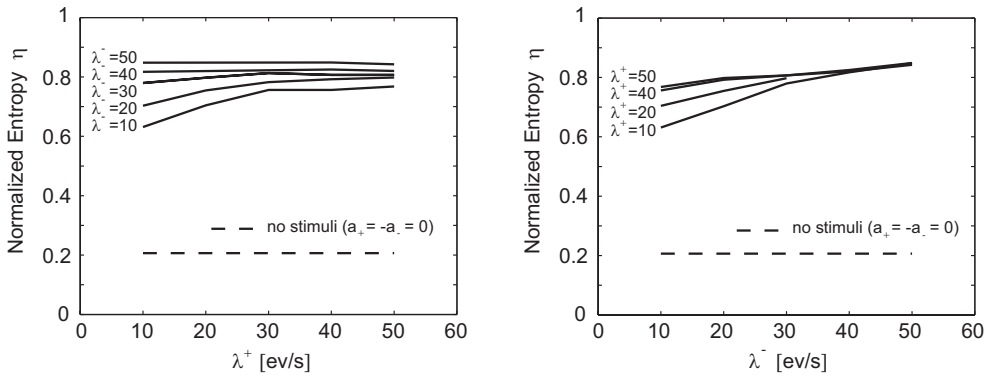


Figure 3: Normalized differential entropy of the simulated spike train for cell $A$ in the presence of excitatory and inhibitory inputs from units $E$ and $I$ (continuous lines), compared with the case with no stimuli (dashed lines). Inputs from $E$ and $I$ are supposed to be exponentially distributed in time. Both panels: $S = 10$ mV, $\theta = 10$ ms$^{-1}$, $\mu = 0.98$ mVms$^{-1}$, $\sigma^2 = 0.05$ mV$^2$ms$^{-1}$, $a_+ = -a_- = 5$ mV.

distributions for the input neurons are much more spread thus enhancing the randomness in the spiking behavior of the receiveng cell $A$. The differential entropy always decreases for increasing $\mu_+ = \mu_-$, but the reduction is larger for smaller values of $\sigma_+^2 = \sigma_-^2$.

**Case (b).** In Fig. 3, left panel we plot the values of the normalized differential entropy of the ISI distribution for cell $A$ versus the input process intensity $\lambda^+$ (expressed in events per second, ev/s) in correspondence with different values of the intensity $\lambda^-$. The right panel in the same figure shows the reverse condition where the intensity $\lambda^-$ is on the $x-$axis and a curve is plotted for each value of $\lambda^+$. Let us notice that here the parameters $\lambda^+$ and $\lambda^-$ determine both the frequency and the variability of firing for units $E$ and $I$. The effect of adding jump stimuli that are exponentially distributed in time to the dynamics of cell $A$ membrane potential induces an increase in the degree of randomness with respect to the case of no stimuli independently on the input process intensities. This result is in agreement with the fact that the exponential distribution maximizes the differential entropy. However the behavior is not symmetric between the excitatory and inhibitory input contributions. Indeed in correspondence with higher values of $\lambda^-$ the normalized entropy increases with moderate to intermediate excitatory intensity values more than what happens in the reversed case.

*4.1.2 Effect of the noise level* In Fig. 4 we plot the normalized differential entropy of the ISI distribution of cell $A$ in the presence of stimuli from units $E$ and $I$ versus the noise level $\sigma^2$ for unit A.

**Case (a).** When the stimuli are IG distributed in time (Fig. 4, left panel), in correspondence with low levels of variability for the input processes (lower $\sigma_+^2$ and $\sigma_-^2$) the normalized entropy is lower or comparable with the reference values taken in the absence of inputs. Higher levels of variability for the input processes make the degree of randomness of the ISI distribution of cell $A$ increase. The shape itself of the line representing the normalized differential entropy against the noise level is different for large or small values of $\sigma_+^2 = \sigma_-^2$. The presence of stimuli with high variability makes the normalized entropy be decreasing for small values of $\sigma^2$, while in the absence of stimuli and with stimuli with low variability the curve is monotonically increasing. Let us notice that those two different shapes have been observed in [9] for the normalized entropy of the first passage time distribution for an Ornstein-Uhlenbeck process through a constant boundary (here corre-

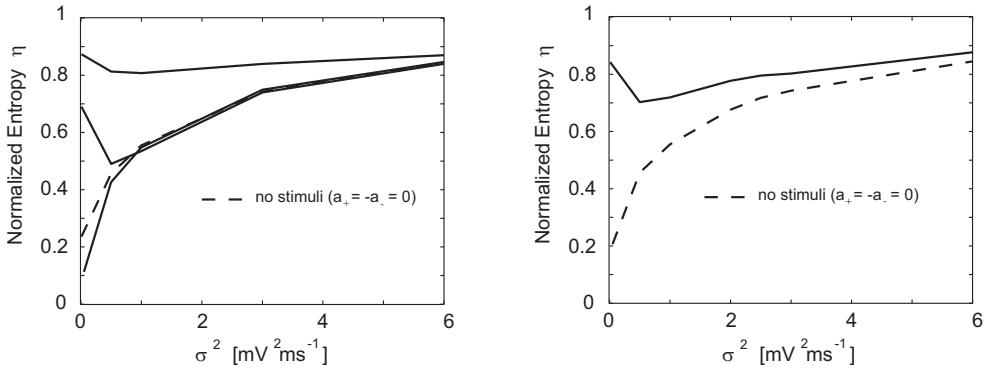


Figure 4: Normalized differential entropy of the simulated spike train for cell $A$ in the presence of excitatory and inhibitory inputs from units $E$ and $I$ (continuous lines), compared with the case with no stimuli (dashed lines). Inputs from $E$ and $I$ are supposed to be IG distributed in time (left panel) and exponentially distributed in time (right panel). Both panels: $\theta = 10$ ms$^{-1}$, $\mu = 0.98$ mVms$^{-1}$, $a_+ = -a_- = 5$ mV. Left panel: $\mu_+ = \mu_- = 0.2$ mVms$^{-1}$, $\sigma_+^2 = \sigma_-^2 = 10, 1$ and $0.2$ mV$^2$ms$^{-1}$ (continuous lines from top to bottom). Right panel: $\lambda^+ = \lambda^- = 20$ ev/s.

sponding to the ISI distribution of cell $A$ with no stimuli) in correspondence of lower and higher values of the drift parameter $\mu$ respectively. Here we observe that, depending on the variability of the strong inputs to the cell but keeping the parameter $\mu$ fixed, the two different behaviors are both exhibited.

**Case (b).** When the stimuli are exponentially distributed in time (Fig. 4, right panel), independently on the choice of the parameters $\lambda^+$ and $\lambda^-$, the normalized entropy increases and the shape of the curve changes being comparable with ISI distributions in the absence of stimuli and with smaller $\mu$.

To assess whether the different behavior shown by IG and esponentially time distributed stimuli is consistent with changes in $\mu$, in Fig. 5 we plot the differential normalized entropy versus the noise level $\sigma^2$ with $\mu = 0.7$ mVms$^{-1}$. For such a choice of the parameter $\mu$ the normalized entropy in the absence of stimuli (dashed line) shows the shape proper of lower values of $\mu$, such that $\mu\theta < S$.

**Case (a).** When the stimuli are IG distributed in time (Fig. 5, left panel) and have low variability, the normalized entropy decreases and the shape of the curve changes. Hence, IG time distributed inputs can modify the ISI distribution of cell $A$ in both directions: from a regime typical of $\mu$ such that $\mu\theta < S$ to a regime with $\mu$ such that $\mu\theta \sim S$ and vice versa.

**Case (b).** When the stimuli are exponentially distributed in time (Fig. 5, right panel), the entropy increases and no change in the shape of the curve can be observed. Hence under this setting in both cases with $\mu\theta \sim S$ and $\mu\theta < S$ the shape of the normalized differential entropy is typical of a regime with $\mu\theta < S$.

Let us remark that once again the result seems due to the randomness of the input processes. Indeed, when no stimuli are present, the firing regime of cell $A$ with $\mu\theta < S$ (under-threshold) is characterized by high randomness, being the firing of cell $A$ due only to the noise term $\sigma W_t$ and not to the deterministic contribution in eq. (2.2). Hence, adding stimuli with low variability (IG distributed ISIs with small $\sigma_+^2 = \sigma_-^2$) increases the regularity of firing of cell $A$. Such a change is detected as a change in the shape of the normalized entropy versus $\sigma^2$ that becomes similar to curves proper of firing distributions with lower

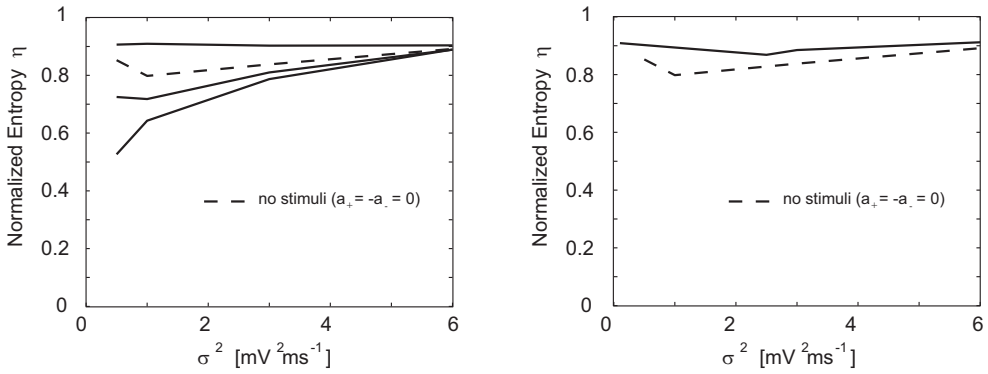


Figure 5: Normalized differential entropy of the simulated spike train for cell $A$ in the presence of excitatory and inhibitory inputs from units $E$ and $I$ (continuous lines), compared with the case with no stimuli (dashed lines). Inputs from $E$ and $I$ are supposed to be IG distributed in time (left panel) and exponentially distributed in time (right panel). Both panels: $S = 10$ mV, $\theta = 10$ ms$^{-1}$, $\mu = 0.7$ mVms$^{-1}$, $a_+ = -a_- = 5$ mV. Left panel: $\mu_+ = \mu_- = 0.2$ mVms$^{-1}$, $\sigma_+^2 = \sigma_-^2 = 10, 1$ and $0.2$ mV$^2$ms$^{-1}$ (continuous lines from top to bottom). Right panel: $\lambda^+ = \lambda^- = 20$ ev/s.

randomness (no stimuli but $\mu\theta \sim S$).

**4.2 Kullback-Leibler distance** In order to quantify the information carried by the excitatory and inhibitory inputs from units $E$ and $I$ and processed by unit $A$, we evaluate the Kullback-Leibler distance (3.6) of the probability density $f(t)$ for the ISI distribution of cell $A$ in the presence of stimuli from units $E$ and $I$ with respect to the probability density $g(t)$ for the ISI distribution of cell $A$ with no stimuli.

The analysis is first performed with excitatory and inhibitory inputs exponentially distributed in time for different values of the excitatory firing frequency $\lambda^+$ of unit $E$ and of the inhibitory firing frequency $\lambda^-$ of unit $I$. The results are plotted in Fig. 6.

We deduce that two regions exist in the space of the parameter values corresponding to different behaviors. For firing frequencies of units $E$ and $I$ that lie in a biologically plausible range (10-40 ev/s), an increment in the stimulation frequency (regardless of the excitatory or inhibitory feature of the inputs) produces an increment in the Kullback–Leibler distance and hence in the average information gain due to the stimulation. Such a result suggests that both excitatory and inhibitory inputs have an active role in the information coding and transmission. On the other hand for larger values of the stimuli frequencies, out of a biologically plausible range, an increment in the excitatory inputs frequency produces an increment of the information gain while an increment in the inhibitory inputs frequency corresponds to a reduction of the information gain. Hence in this range of the parameters excitatory and inhibitory inputs have different role in the information transmission.

Let us notice that the increase in the Kullback–Leibler distance as $\lambda^-$ increases (Fig. 6, right panel $\lambda^+ < 40$ ev/s) is stronger in correspondence of smaller values of the parameter $\lambda^+$. Hence the effect of intensified inhibitory inputs is stronger when $A$ is less excited. This feature did not arise in [23] where it has been shown that the effect of inhibitory inputs with increasing frequency is comparable for all the considered excitation intensities.

In order to test the robustness of these results to changes in the ISI distribution of the input neurons $E$ and $I$, in Fig. 7 we plot the Kullback-Leibler distance as in Fig. 6, but for IG time distributed excitatory inputs and exponentially time distributed inhibitory inputs.

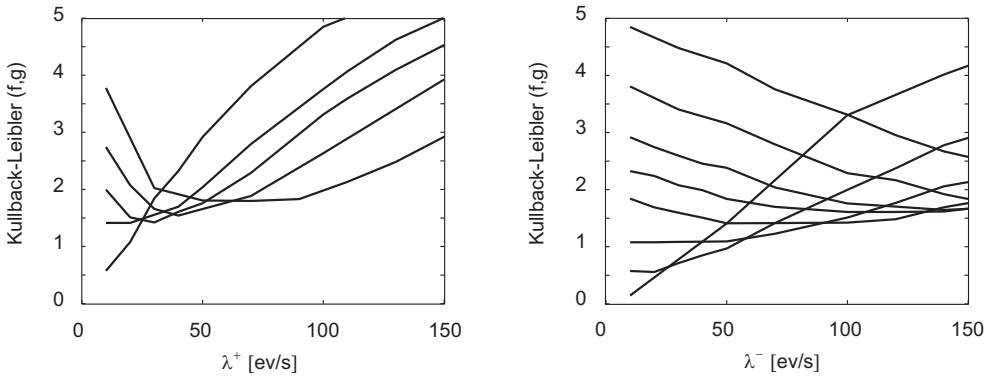


Figure 6: Kullback-Leibler distance of the ISI distribution of cell $A$ in the presence of excitatory and inhibitory inputs with respect to the ISI distribution of cell $A$ with no stimuli ($a_+ = -a_- = 0$ mV). Inputs from $E$ and $I$ are supposed to be exponentially distributed in time. Both panels: $S = 10$ mV, $\theta = 10$ ms$^{-1}$, $\mu = 0.98$ mVms$^{-1}$, $\sigma^2 = 0.05$ mV$^2$ms$^{-1}$, $a_+ = -a_- = 5$ mV. Left panel: $\lambda^- = 220, 140, 100, 70$ and 10 ev/s (from top to bottom on the left side of the panel). Right panel: $\lambda^+ = 100, 70, 50, 40, 30, 20, 10$ and 1 ev/s (from top to bottom on the left side of the panel).

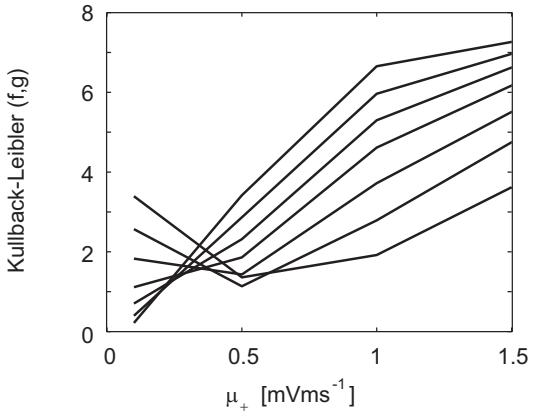


Figure 7: Kullback-Leibler distance of the ISI distribution of cell $A$ in the presence of excitatory and inhibitory inputs with respect to the ISI distribution of cell $A$ with no stimuli ($a_+ = -a_- = 0$ mV). Inputs from $E$ are supposed IG inter-distributed while inputs from $I$ are supposed to be exponentially distributed in time. $S = 10$ mV, $\theta = 10$ ms$^{-1}$, $\mu = 0.98$ mVms$^{-1}$, $\sigma^2 = 0.05$ mV$^2$ms$^{-1}$, $a_+ = -a_- = 5$ mV. From top to bottom on the left side of the panel: $\lambda^- = 170, 130, 100, 70, 50, 30$ and 10 ev/s.

The similarity between Fig. 7 and Fig. 6, left panel suggests that the role of excitatory and inhibitory inputs in the information transmission does not depend on the choice of the ISI distribution. Rather it may be due to the structure of the network itself and could be presumably generalized to any ISI distribution.

**5 Conclusions** The features of a small neuronal network proposed in [23] have been discussed with the aid of two information measures: the normalized differential entropy and the Kullback-Leibler distance. The use of such quantitative information measures allows to get the following results:

- the presence of strong excitatory and inhibitory contributions distributed in time with low variability, such as the IG distribution for small $\sigma_+^2$ and $\sigma_-^2$, determines an decrease in the randomness of the spiking activity of the receiving neuron. Note that this case corresponds to the strongly multimodal ISI distribution histograms showed in [23];
- the presence of strong excitatory and inhibitory contributions exponentially distributed in time (and hence with a distribution characterized by maximal entropy) always increases the randomness of the output response;
- the presence of IG distributed input events with low variability let switch from dependencies of the differential entropy on the noise intensity that are proper of under–threshold regimes to those of supra–threshold ones and vice versa;
- being the exponentially time distributed contributions characterized by strong randomness, they are able to produce a change in the dependency of the differential entropy on the noise intensity only from the supra-threshold regime to the under-threshold one;
- an increment in the frequency of both excitatory and inhitory processes, independently on the distribution of the inputs, corresponds to an increment in the Kullbak-Leibler distance, i.e. an increment in the average information gain due to the presence of excitatory and inhibitory stimuli. Hence both excitation and inhibition are found to carry information content. Note that this result is in agreement with the analysis performed in [23] through crosscorrelation histograms.

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*Maria Teresa Giraudo, Laura Sacerdote, Roberta Sirovich*
Department of Mathematics, University of Torino
Via Carlo Alberto 10, 10123 Torino, Italia
E-mail: {mariateresa.giraudo,laura.sacerdote,roberta.sirovich} @unito.it